WEAVE SPRINT . PAPER III

Child behavioural, emotional & other disorders

The disruptive behaviour and conduct disorders, from conduct disorder and its callous-unemotional specifier to oppositional defiant disorder and the impulse-control disorders; the childhood emotional disorders across separation anxiety, the anxiety, OCD and trauma family, childhood depression, disruptive mood dysregulation against the paediatric-bipolar controversy, early-onset psychosis and youth suicidality; then the attachment disorders and their differential from autism, the tic disorders and Tourette syndrome with tic pharmacotherapy, and the elimination and feeding disorders with enuresis pharmacotherapy, every dose scoped to its patient and indication, and the discriminations a candidate is examined on.

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Dr. Wilfred D'souza . Dr. Niharika Reddy

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PAPER III

Child behavioural, emotional & other disorders

Seven bands . one complete notes document

Conduct disorder

1 · The five groups and the shape of the chapter

A chapter reads more easily once you can see its skeleton. The disorders gathered under this heading are the ways children and adolescents come to behave, feel and function outside the expected range, and set out as a bare list they can look like a loose and only distantly related collection: conduct problems next to bedwetting, tics next to school refusal, a withdrawn deprived toddler next to a defiant teenager. That impression is misleading, and it is worth dissolving at the outset, because the candidate who carries a shape into the exam answers a mixed vignette far better than one who has memorised each entity as an island. The chapter's own title supplies the shape. It names three things in order: the behavioural disorders, the emotional disorders, and the other disorders, and those are not three arbitrary drawers but a genuine organizing logic that the rest of this material fills in.

The aim of this opening is to hand you that logic before any single disorder is taught. Three moves do the work. The first is an axis that links the two largest groups, distinguishing distress that a child turns outward from distress a child turns inward. The second is the recognition that not every childhood disorder belongs on that axis: three further families keep their own logic and are misread if forced onto it. The third is a principle that runs underneath the whole chapter, that these are disorders of degree and of context, judged against a developmental baseline that is itself moving. Hold those three and the individual diagnoses that follow slot into place rather than accumulating as a list.

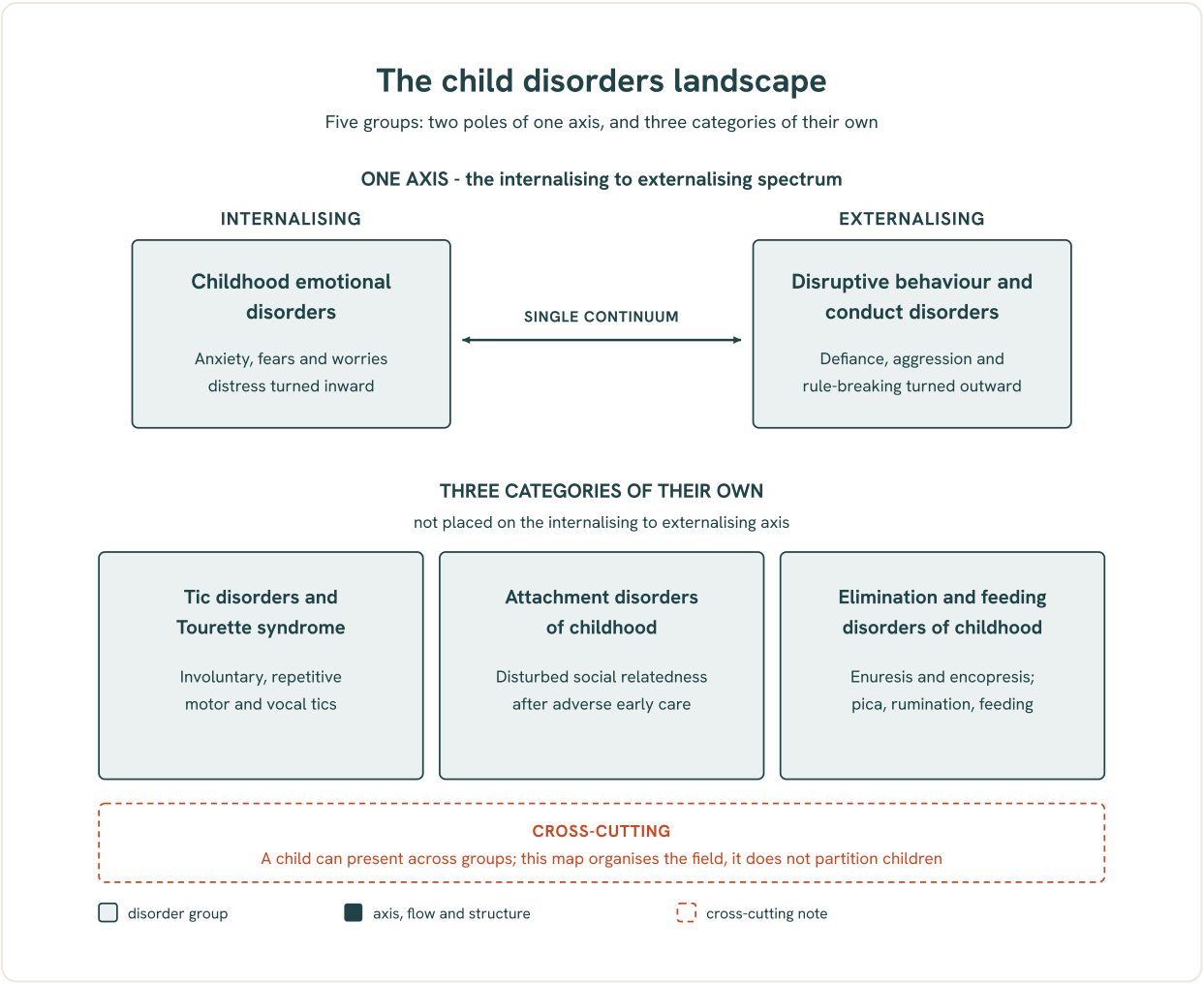

Fig 23.1. The child disorders landscape: emotional (internalising) and disruptive or conduct (externalising) disorders sit at the two poles of one axis, while tic, attachment, and elimination or feeding disorders form three categories of their own.

1.1 The organizing axis: distress turned outward and distress turned inward

The two largest groups are two directions of the same problem. The most useful single distinction in child psychiatry is between the disorders in which difficulty is expressed **externally**, as behaviour that impinges on other people and on the surrounding world, and those in which it is experienced **internally**, as suffering the child largely carries within. The externalising, or behavioural, group is the disruptive one: conduct disorder, oppositional defiant disorder and the impulse-control disorders, where the presenting problem is what the child does to others and the first complaint usually comes from an adult. The internalising, or emotional, group is the group of anxiety, obsessive-compulsive and trauma-related disorders and the childhood mood disorders, where the presenting problem is what the child feels and the first complaint, if it comes at all, often comes late and quietly. The value of the axis is not taxonomic tidiness but clinical direction: it tells you who is likely to be referred and who is likely to be missed, whose distress announces itself and whose has to be sought.

RULE **Externalising and internalising is one organizing dimension, not a complete classification.** It is a lens laid across the disorders to show a pattern, chiefly that outward-directed problems are referred and inward-directed ones are overlooked. Read it as a filing system into which every childhood disorder must drop and it stops helping, because several of the disorders in this chapter do not sit on it at all.

1.2 The three families that keep their own logic

The "other disorders" of the title are three groups with their own organizing principle. A frequent error is to treat the outward-inward axis as exhaustive and then to squeeze the remaining disorders onto it. They do not belong there, and each is better understood on its own terms. The **tic disorders**, including Tourette syndrome, are neurodevelopmental and motor in nature; their defining feature is an involuntary movement or vocalisation, not an emotional state turned in or a behaviour turned out. The **attachment disorders** are relational disorders that arise specifically from grossly deficient early care, so their organizing principle is the child's history of caregiving rather than any internalising or externalising temperament. The **elimination and feeding disorders** are disorders of bodily function and its regulation, from enuresis and encopresis to the feeding and eating problems of early childhood. Naming these three as their own categories, rather than as odd members of the behavioural or emotional groups, is itself a piece of examinable understanding, and it is why the chapter is built as five families and not two.

FAMILY	ORGANIZING LOGIC	REPRESENTATIVE MEMBERS (TAUGHT IN FULL IN THEIR OWN PLACE)
Behavioural (externalising)	Distress turned outward onto others and the world	Conduct disorder, oppositional defiant disorder, the impulse-control disorders
Emotional (internalising)	Distress carried within, often referred late	The childhood anxiety, obsessive-compulsive and trauma-related disorders, the childhood mood disorders
Tic disorders	Neurodevelopmental and motor; involuntary movement or vocalisation	Transient and chronic tic disorders, Tourette syndrome
Attachment disorders	Relational; rooted in grossly deficient early care	Reactive attachment disorder, disinhibited social engagement disorder
Elimination and feeding	Disorders of bodily function and its regulation	Enuresis, encopresis, and the feeding and eating disorders of childhood

The five families of the chapter set against the principle that organizes each. The axis of the first section explains only the top two rows; the lower three are named here as their own categories and are developed, each in full, later in this chapter.

1.3 Why the axis is a lens and not a wall: comorbidity across it

Outward and inward disorders live together far more often than the neat split suggests. The axis earns its keep by directing attention, but a candidate who treats it as a partition will be

caught by the commonest examination presentation of all, the child who sits on both sides at once. The disruptive, acting-out child very frequently carries an internalising disorder underneath the behaviour, and the anxious or depressed child not uncommonly presents through irritability and rule-breaking that look, at first glance, purely behavioural. That the two directions co-occur is not an untidy exception to the scheme; it is the clinical rule, and it is precisely why the outward-inward distinction has to be held as a way of looking rather than as a boundary between separable populations.

TRAP **Treating externalising and internalising as mutually exclusive.** The vignette that describes a defiant, aggressive boy invites the label of a pure behavioural disorder and tempts the candidate to stop there. The stronger answer notices that the behaviour may be the visible surface of an anxiety, trauma or mood disorder beneath it, and that the two so-called opposite groups are the disorders most likely to be found in the same child.

GOOD TO REMEMBER

When a child is referred for what one side of the axis makes obvious, look deliberately for the other. Screen the acting-out child for the anxiety, trauma and mood that hide behind conduct, and ask the withdrawn or anxious child about the irritability and defiance that a caregiver may not have thought worth mentioning. The referral tells you which door the family came through, not the whole of what is inside.

1.4 Disorder against a moving baseline: the developmental continuum

Almost every symptom in this chapter also exists as a normal part of growing up. Tantrums, fears, defiance, bedwetting, repetitive movements, clinginess at separation and the odd untruth are ordinary features of childhood, present in well children and expected at particular ages. This is the principle that sits under the whole chapter and separates its disorders from the developmental noise around them: a childhood disorder is rarely a behaviour that healthy children never show, and is far more often the same behaviour grown persistent, pervasive, developmentally out of step, and impairing. The **developmental continuum** is therefore not a soft caveat but the working definition of pathology here, because what is normal at one age is a red flag at another, and the baseline against which any symptom is read keeps shifting as the child grows. What counts as expected at a given age is set out in the Normal child development and milestones notes; the specific thresholds that separate each disorder from its normal counterpart, and the map that sets the whole set of them side by side, are drawn together toward the end of this chapter, where the comparisons can be made once and properly.

PITFALL

The characteristic error at this level is to diagnose from the behaviour alone, without asking where the child sits on the developmental continuum. A defiant three-year-old, a frightened five-year-old and an occasionally wet seven-year-old are, far more often than not, well children doing age-appropriate things. Reaching for a disorder because the behaviour is present, rather than because it is persistent, pervasive and impairing for that age, over-pathologises ordinary development and is the mirror image of the more familiar failure to recognise a genuine disorder.

1.5 Reading the chapter, and a word on classification in Indian practice

The map is worth carrying into every question this chapter can ask. With the five families and the two principles in hand, the chapter equips you to do a small number of things reliably, and it is worth naming them before the detail begins:

- place any child presentation into its family, and recognise when a presentation straddles two of them;
- read the outward-inward axis as a lens on referral and detection, not as a partition;
- separate a disorder from its normal developmental counterpart by persistence, pervasiveness and impairment rather than by the behaviour alone;
- and know which of these childhood presentations point outward, to the adult disorders and to the wider systems of assessment, services and protection that lie beyond this chapter.

Where the child's presentation reaches into the domains this chapter deliberately leaves to others, the pointers are fixed: attention-deficit/hyperactivity disorder and its treatment belong to the ADHD and specific learning/communication disorders notes, and the full assessment battery, the child mental health services and the machinery of child protection to the Child psychiatry: assessment, treatment, services and protection notes.

IN INDIA

Two realities colour how this chapter is used in Indian practice. The operative classification is ICD-11, so the labels that appear in most case files and referrals are its labels rather than the DSM equivalents, and the family-based, service-scarce setting means these children very often reach a paediatrician, a general physician or a school long before they reach a child psychiatrist. The organizing consequence is practical: the outward-inward axis predicts which of them arrive at all. Disruptive, externalising problems are brought in because they trouble the adults around the child, while the internalising disorders, which cause the child the greater private suffering, are the ones most likely to go unrecognised in a system with few dedicated child mental health services. The frame is not only a way to revise the chapter; it is a reminder of where to look hardest.

MNEMONIC

Out, In, and three apart

Two families share a single axis, the child who turns distress **Out**, the behavioural or externalising disorders, and the child who turns it **In**, the emotional or internalising disorders. **Three** further families keep their own logic **apart** from that axis: the tic disorders (neurodevelopmental and motor), the attachment disorders (relational, rooted in early care), and the elimination and feeding disorders (of bodily function). The whole chapter hangs on those five, and every one of them is read against the moving developmental baseline.

The spine of the chapter: two families on one axis, distress turned Out (behavioural, externalising) against distress turned In (emotional, internalising), and three families apart from it - tic, attachment, and elimination and feeding disorders - with every disorder defined against the developmental baseline rather than by the behaviour alone.

Out	In	Apart
THE BEHAVIOURAL, EXTERNALISING DISORDERS	THE EMOTIONAL, INTERNALISING DISORDERS	TIC, ATTACHMENT, ELIMINATION AND FEEDING

2 · Conduct disorder diagnostic criteria and subtypes (childhood-onset, adolescent-onset)

The diagnosis turns on a pattern, not on a single act. **Conduct disorder** is the syndrome of childhood and adolescence in which a young person repeatedly and persistently behaves in ways that violate the basic rights of others or the major age-appropriate rules of society. It is one of the commoner reasons a child reaches psychiatric attention: one-year population prevalence in high-income settings has been estimated at a median of about **4%**, with estimates ranging from 2% to more than 10% across studies, and it falls more heavily on boys and men, whose lifetime prevalence has been put at about **12.0% in men and 7.1% in women**. Marks accrue here because the diagnosis is deceptively easy to reach for and genuinely hard to make well. The behaviours are dramatic, the temptation to label a difficult adolescent is strong, and the criteria exist precisely to separate a disorder from ordinary misbehaviour, from isolated delinquency, and from the friction of a hard upbringing.

This topic owns the diagnostic architecture: the essential feature, the count and timeframe of Criterion A, the behavioural groupings the criteria fall into, the impairment and adult-exclusion criteria, the developmental onset subtypes that are the item's real teaching point, and the severity grading. The callous-unemotional presentation captured in the limited prosocial emotions specifier, the causes and risk factors, the full assessment and its instruments, the treatments, and the long-term trajectory into adult antisocial outcomes are each developed in their own right elsewhere in this chapter. Here they are named where the criteria demand it, not reopened.

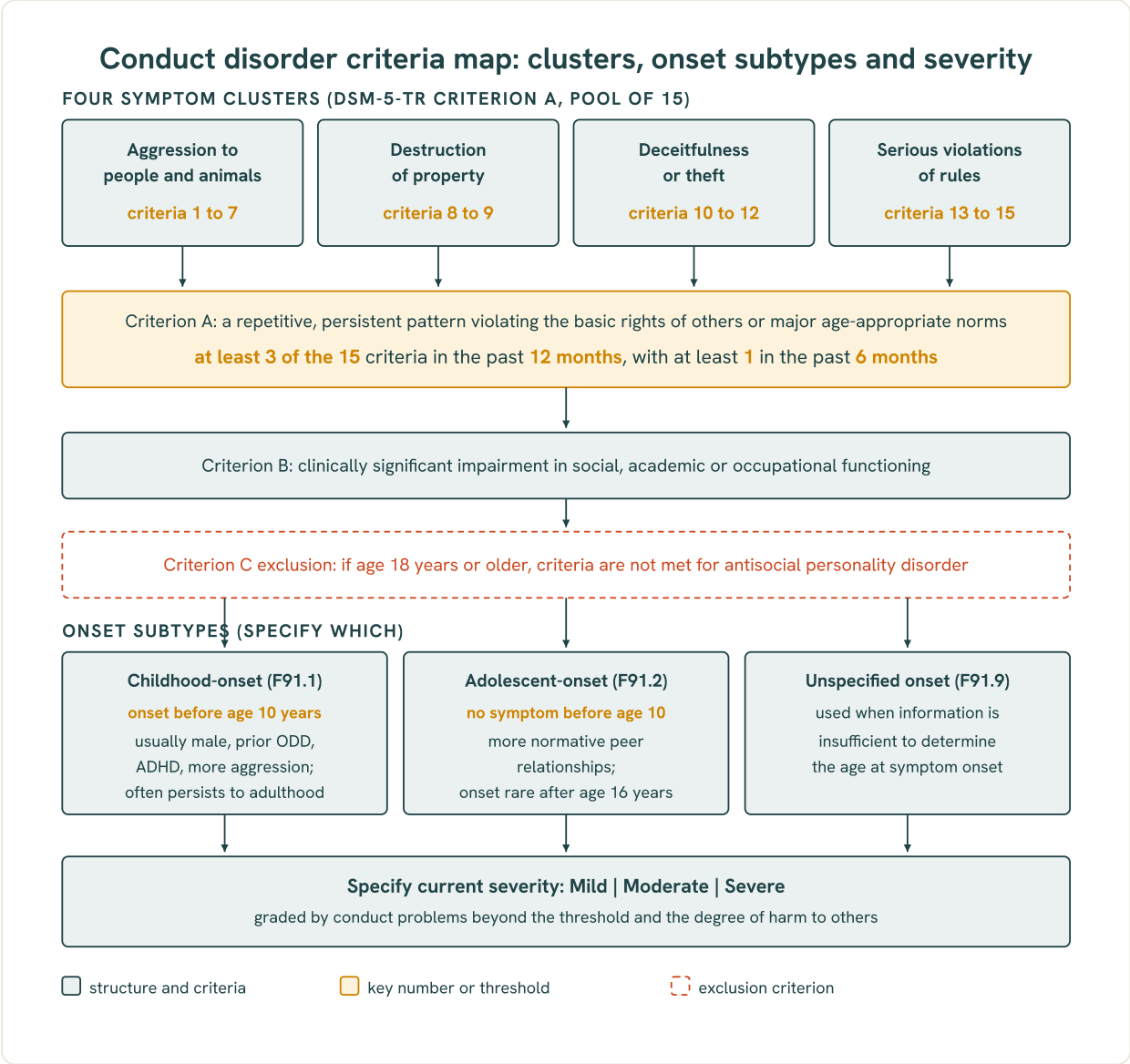

Fig 23.2. Conduct disorder criteria map: the four DSM-5-TR symptom clusters feeding the three-of-fifteen Criterion A threshold, the impairment and antisocial-personality-disorder gates, the childhood-onset and adolescent-onset subtypes, and the mild to moderate to severe severity grading.

2.1 The essential feature: a pattern, not an incident

The unit of diagnosis is a pattern of conduct over time. The essential feature is a repetitive and persistent pattern of behaviour in which the young person violates either the basic rights of others or the major age-appropriate norms and rules of society. Each part of that definition matters. Violating the rights of others captures aggression, cruelty, intimidation, and the taking of what belongs to someone else; violating major societal norms captures the serious rule-breaking, from chronic truancy to running away, that need not have a direct victim at all. What unifies the picture is not any single behaviour but its repetition and its persistence, and this is exactly why a solitary serious act, however shocking, does not by itself constitute the disorder. A first arrest, a single fight, or one episode of theft is an event to be understood, not a diagnosis to be made. The clinician's task is to establish whether a durable pattern sits behind the presenting incident, because it is the pattern, and not the incident, that predicts the course and justifies the label.

RULE **Conduct disorder is a pattern, never a single act.** The diagnosis rests on a repetitive and persistent course of rights-violating or norm-violating behaviour, so the assessor's first question is never what did the child do but what pattern does this belong to. An isolated offence read as a disorder is the error the criteria are built to prevent.

2.2 Criterion A: the count and the clock

The threshold and the timeframe are the examinable core. Criterion A operationalises the pattern into a rule that can be applied and defended. It requires that at least **three** of the **15** defined behaviours have been present within the past 12 months, with at least one of them present within the past 6 months. Each number earns its place. The count separates a genuine syndrome from a handful of troubling behaviours, and the recency window forces the clinician to show that the problem is current rather than historical, so that a child who has genuinely settled is not carried indefinitely under an old label. The number of symptoms present beyond the minimum is not wasted information either, because it feeds directly into the severity rating. Applied honestly, Criterion A is what stops the diagnosis drifting into a catch-all for any troublesome adolescent.

The core rule to carry into the exam: at least three of the 15 behaviours within 12 months, at least one within the past 6 months, forming a pattern and not a single act.

2.3 The four behavioural groupings

Fifteen behaviours, grouped into coherent families. The criteria are not a random list; they sort into four groupings that together describe the reach of the disorder from covert acts to overt ones. The first is **aggression to people and animals**, the behaviours that threaten or inflict physical harm, including bullying, fighting, cruelty, and the use of a weapon. The second is destruction of property, such as deliberate firesetting and other damage done on purpose. The third is deceitfulness or theft, the covert dimension of lying, conning others, and stealing with or without confrontation. The fourth is serious violations of rules, such as staying out at night against parental prohibition, persistent truancy, and running away from home. Reading a young person's symptoms as a profile across these families, rather than as a scatter of unrelated incidents, is what turns a list of complaints into a clinical picture.

MNEMONIC

Hurt, Wreck, Lie, Break

The groupings in order of how overtly they present: **Hurt** (aggression to people and animals), **Wreck** (destruction of property), **Lie** (deceitfulness or theft), and **Break** (serious violations of rules). Holding the families in mind stops the assessor cataloguing behaviours singly and missing the pattern that the diagnosis actually requires.

2.4 Impairment and the adult boundary: Criteria B and C

The behaviour must cost something, and the label must not outlive its usefulness. Criterion B keeps the diagnosis clinical rather than merely descriptive: the disturbance of conduct must

cause clinically significant impairment in social, academic, or occupational functioning. Behaviour that breaks rules but carries no cost to the young person's functioning does not qualify, which guards against pathologising nonconformity. Criterion C then draws the upper boundary. Conduct disorder can still be diagnosed in an adult, but only where the person, if aged 18 years or older, does not meet the criteria for **antisocial personality disorder**. Once that adult syndrome is established it takes precedence, and conduct disorder is reserved for those who have not crossed into it. The adult disorder itself, and the pathway a proportion of children take toward it, belong respectively to the Personality disorders notes and to a later discussion in this chapter; here the point is only the diagnostic hierarchy that Criterion C encodes.

TRAP **Conduct disorder does not automatically stop at adulthood.** Candidates assume the diagnosis is purely paediatric and expires the moment the patient turns adult, or conversely that every antisocial adult once carried it formally. Criterion C is subtler: in a person aged 18 or older the diagnosis still stands, provided the full criteria for antisocial personality disorder are not met. The exclusion is conditional, not automatic.

2.5 The developmental onset subtypes

When the trouble began matters more than what form it takes. DSM-5-TR requires the clinician to specify the developmental course, because the age of onset is the specifier with the greatest prognostic weight. In the **childhood-onset type**, at least one characteristic symptom of conduct disorder is present before age **10**; it is coded F91.1. In the **adolescent-onset type**, no characteristic symptom is present before age 10, the first appearing only in the teenage years; it is coded F91.2. Where the history is too incomplete to establish which course applies, for instance when the early childhood years cannot be reliably reconstructed, the diagnosis is left as unspecified onset, coded F91.9. The division looks administrative, but it is anything but: it sorts children who can look broadly similar at presentation into groups whose correlates and outlook differ sharply, which the next section takes up.

2.6 Why onset age matters: the clinical correlates

Early onset marks the more severe and more persistent illness. The reason the subtypes are worth specifying is that they carry different clinical company and different trajectories. The childhood-onset form is the more ominous. Such children are usually male, their peer relationships are frequently disturbed, and their conduct problems are often preceded in the earlier years by **oppositional defiant disorder**. They tend to show more aggression, and they frequently carry a concurrent attention-deficit/hyperactivity disorder, the assessment and treatment of which belong to the ADHD and specific learning/communication disorders notes. Crucially, the childhood-onset form is the one more likely to persist into adult life, which is why it, and not the adolescent-onset form, is the subtype that carries the recognised risk of an antisocial adult outcome. The adolescent-onset form, by contrast, tends to arise against a background of more normative peer relationships and is more often a reaction to the pressures and affiliations of adolescence than the expression of a long-standing trait. That contrast is the clinical heart of the distinction: conduct that is present from early childhood tends to reflect a more pervasive and enduring vulnerability, whereas conduct that emerges only in adolescence is

more often tied to a particular developmental phase and its social affiliations, and is correspondingly more likely to remit as that phase passes. Onset of the disorder is rare after age 16. A further high-risk subgroup, marked by the callous-unemotional presentation captured in the limited prosocial emotions specifier, is developed in its own right elsewhere in this chapter.

FEATURE	CHILDHOOD-ONSET	ADOLESCENT-ONSET
First symptom	Before age 10	Not before age 10
Peer relationships	Often disturbed	More often normative
Aggression	More prominent	Comparatively less
Course into adulthood	More likely to persist	More likely to remit

The onset subtypes set against the correlates that give the childhood-onset form its worse outlook; the adolescent-onset column is defined chiefly by the absence of early symptoms and by more normative peer relationships.

2.7 Grading the disorder: the severity specifier

The diagnosis is incomplete until it is graded. DSM-5-TR asks for a rating of **current severity** as mild, moderate, or severe. The grade reflects the number of conduct problems present beyond the minimum needed to make the diagnosis, together with how much harm those problems do to others. A young person who just clears the threshold, with problems that are minor and cause limited harm, sits at the mild end; one with many problems, or with conduct that inflicts considerable harm, sits at the severe end. Severity is rated independently of onset, so either developmental subtype can present in mild, moderate, or severe form. The grade is not cosmetic. It summarises the current burden and travels with the young person into every service and record that follows, shaping the urgency and intensity of the response.

> **GOOD TO REMEMBER**
>
> Always assign the severity, and treat it as clinical information rather than a formality. A mild adolescent-onset case and a severe childhood-onset case share a diagnostic label but are different clinical problems with different outlooks, and the grade you record is often the single most useful line in the entry for whoever reads it next.

2.8 The nearest neighbours: oppositional defiant disorder and normal limits

Most diagnostic errors cluster at the borders of the diagnosis. The first border is with oppositional defiant disorder. The two share defiance, anger, and a tendency to blame others, and childhood-onset conduct disorder is frequently preceded by it, but they part on a single decisive line: oppositional defiant disorder does not involve the violation of the basic rights of others or of major societal norms that defines conduct disorder. Argumentative, spiteful, and rule-refusing behaviour that stops short of aggression, destruction, deceit, and serious rule-breaking is the lesser diagnosis, which is developed in its own right elsewhere in this chapter. The second border is with ordinary development. Rule-testing, occasional lying, the odd fight, and adolescent risk-taking are common and usually self-limiting, and what counts as expected at a given age belongs to the Normal child development and milestones notes. The diagnosis is reserved for the persistent, impairing, rights-violating pattern, and not for any one behaviour taken alone.

- a single serious act, however grave, does not meet the pattern requirement;
- defiance and argumentativeness without violation of others' rights point toward oppositional defiant disorder, not conduct disorder;
- transient, situational rule-testing that fits the child's developmental stage is not a disorder;
- the behaviour must cause clinically significant impairment before it counts at all.

PITFALL

The commonest clinical error is over-reach: attaching the label to a defiant, delinquent, or simply hard-to-manage adolescent because the behaviour alarms the adults around them, rather than because a persistent pattern of rights-violating conduct has actually been established over time. A chaotic or depriving environment can generate rule-breaking that is adaptive to its setting; the diagnosis asks for a pattern in the young person's conduct, not a verdict on their circumstances.

2.9 The ICD-11 counterpart and how it differs

The manual that governs most Indian practice draws the same disorder with a different rule.

In ICD-11 the entity is **conduct-dissocial disorder**, coded 6C91. Its required feature is the same repetitive and persistent pattern of behaviour violating the basic rights of others or major age-appropriate social or cultural norms, sustained and recurrent over an extended period, for example 1 year or more, and it states explicitly that the mere commission of isolated delinquent acts is not sufficient. The behaviours fall into the same four groupings. The material difference is at the threshold: ICD-11 sets no fixed symptom count of the kind DSM-5-TR uses, treating the diagnosis as a clinical judgement about the pattern rather than a tally against a list. The same developmental split is preserved, as childhood onset (6C91.0, with one or more features before age 10) against adolescent onset (6C91.1, with none before that age).

AXIS	DSM-5-TR	ICD-11 (CONDUCT-DISSOCIAL DISORDER)
Symptom threshold	At least three of 15 criteria, with a recency rule	No fixed count; a persistent pattern over an extended period
Behaviour groupings	The same four	The same four
Onset subtypes	Childhood (F91.1) and adolescent (F91.2)	Childhood (6C91.0) and adolescent (6C91.1)
Diagnostic logic	Operationalised count and timeframe	Clinical judgement of the pattern

The same disorder under the two classifications; the load-bearing difference is that ICD-11 requires a persistent pattern but not a fixed count, which places the weight on documented clinical judgement.

IN INDIA

Because Indian services classify under ICD, the operative label in most case files, referrals, and official reports is conduct-dissocial disorder rather than the DSM term. The absence of a fixed symptom count is not a licence for looseness: it puts a premium on documenting the pattern, its persistence, and its impact carefully, since the diagnosis rests on the clinician's recorded reasoning rather than on a number that can be checked off. Where a young person's conduct intersects with the law, the juvenile-justice framework is taken up separately in this chapter, and the wider machinery of child protection and services in the Child psychiatry: assessment, treatment, services and protection notes.

3 of 15	age 10	4%
CRITERIA NEEDED FOR THE DIAGNOSIS	ONSET CUT DIVIDING THE SUBTYPES	MEDIAN ONE-YEAR PREVALENCE

3 · Limited prosocial emotions specifier (callous-unemotional traits)

The specifier answers a different question from the diagnosis itself. The diagnosis of conduct disorder tells you that a young person repeatedly violates the rights of others or the major rules of society; it says nothing about the emotional machinery driving that behaviour. The **limited prosocial emotions** specifier, the formal home of what the developmental literature calls **callous-unemotional traits**, is the manual's attempt to mark out the subgroup whose conduct problems are accompanied by a genuine deficit in the affective and interpersonal capacities that normally restrain antisocial behaviour. Marks accrue here because this is a high-yield discrimination that examiners love: the same headline behaviour, delinquency and aggression, can sit on top of two very different internal pictures, and the presence of this specifier changes what the clinician expects of severity, of persistence, and of response to the usual behavioural treatments.

This topic owns the specifier: its defining logic, its threshold, its four constituent features, the assessment demands it imposes, and the way it maps onto the international classification differently from the American one. The conduct disorder criteria and the onset subtypes, the wider etiology in which callous-unemotional traits sit as one developmental pathway, and the trajectory into adult antisocial outcomes for which these traits are a recognised predictor are each developed in their own right elsewhere in this chapter. Here they are named where the argument needs them, not reopened.

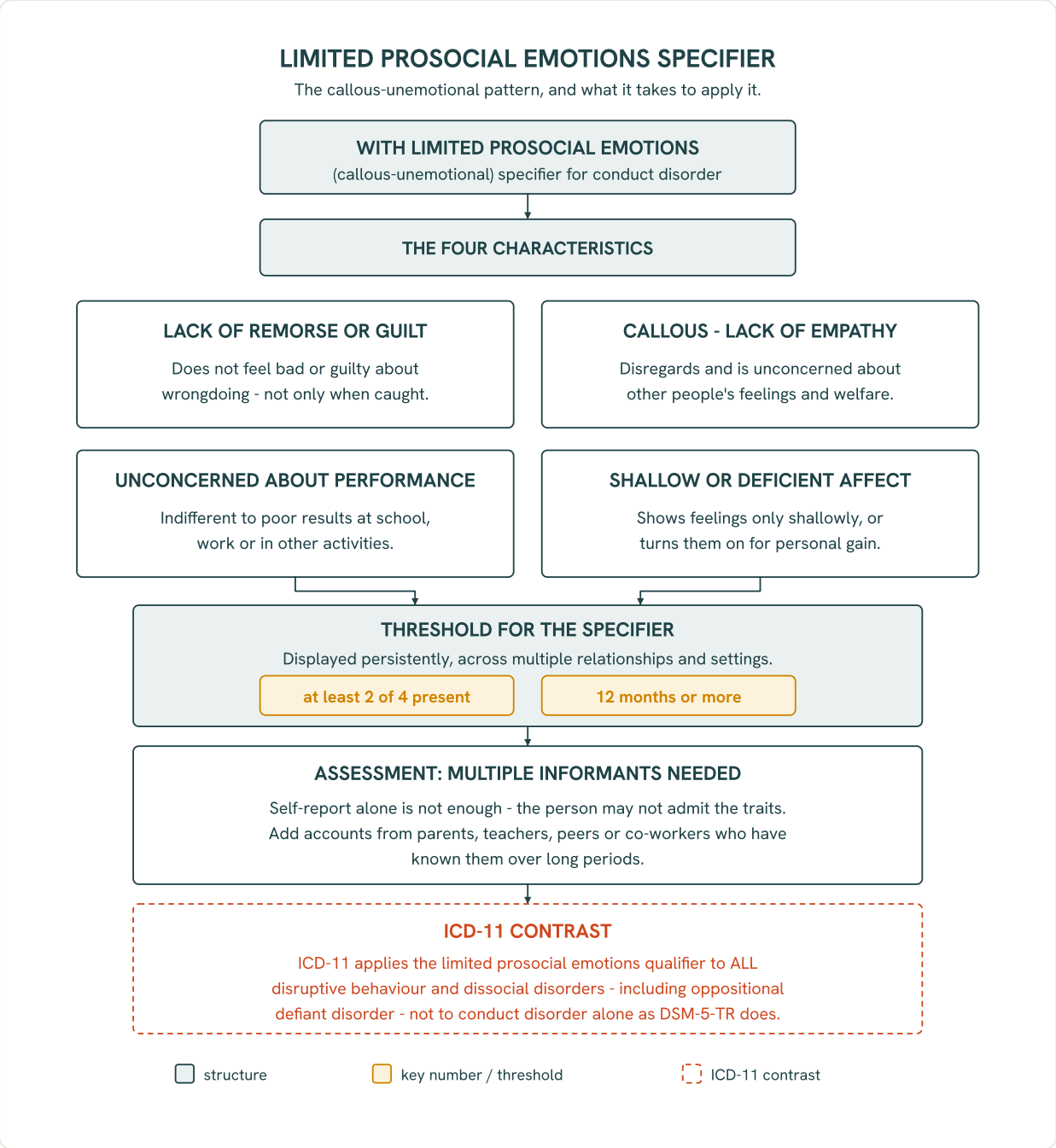

Fig 23.3. Limited prosocial emotions specifier: the callous-unemotional pattern for conduct disorder, its four characteristics, the threshold to apply it, and the ICD-11 contrast.

3.1 What the specifier marks, and why it is not the same as severe conduct disorder

It grades the emotional style of the behaviour, not its quantity. The commonest conceptual error is to treat this specifier as simply the top end of a severity scale, so that the most violent or most prolific offender automatically earns it. That is wrong, and understanding why is the whole point of the topic. Severity in conduct disorder counts how many behaviours are present and how much harm they do; the specifier instead asks about the affective-interpersonal quality that underlies the behaviour, the presence or absence of guilt, empathy, concern and genuine feeling. A frightened, reactive adolescent whose aggression is impulsive and who feels real remorse afterwards may carry a severe rating and still fall well short of the specifier. Conversely, a young person whose offending is more calculated and less florid may show exactly the cold, unemotional style the specifier captures. The two axes are orthogonal: one measures how much, the other

measures of what kind. Reading the specifier as a synonym for "worst" collapses that distinction and loses the clinical information the manual was trying to preserve.

RULE **The specifier describes the affective style of the conduct problem, not its magnitude.** It is applied on the presence of a callous, guilt-poor, empathy-poor interpersonal pattern, independently of how many symptoms or how much harm the severity rating records. A severe case need not qualify; a qualifying case need not be the most severe.

3.2 The threshold: a stable pattern, not an occasional coldness

Two of four features, sustained over time and across relationships. To apply the specifier the clinician must establish that the young person has displayed **at least two** of the **four** defining characteristics, and the qualifying words do more work than the count. The features must have been present persistently, over a period of **12 months** or more, and they must show themselves in multiple relationships and settings rather than in one strained situation. This is a deliberate guard against over-diagnosis. Every adolescent is capable of a cold, dismissive moment, particularly toward an adult they are in conflict with; what the specifier is reaching for is a typical way of relating and feeling, a trait rather than a state. Requiring the pattern across settings and informants means the clinician is describing how this young person characteristically operates in the world, and not simply how they presented in a single tense encounter. The threshold is low in number and high in stringency of pattern, and that combination is exactly what the examiner wants you to be able to state.

> The must-memorise threshold: at least two of the four features, present persistently for at least 12 months and shown across multiple relationships and settings, so that a typical pattern and not an occasional occurrence is being described.

3.3 The four features

Each feature names a specific missing capacity. The four characteristics are not interchangeable synonyms for coldness; each points at a distinct deficit, and being able to name and illustrate all four is standard examination fare. The first is a **lack of remorse or guilt**, an absence of feeling bad about wrongdoing that is genuine and not merely the sullen regret of having been caught. The second is being **callous, with a lack of empathy**, a disregard for and unconcern about the feelings of others that shows in how the young person treats the people they harm. The third is being **unconcerned about performance** at school, at work or in other activities, where the ordinary drive to do well and the discomfort of doing badly appear muted. The fourth is a **shallow or deficient affect**, an emotional life that seems thin, or feelings that are expressed only superficially or turned on and off in a way that looks instrumental. Read together they sketch the affective core of what, in the adult, becomes the interpersonal-affective dimension of antisocial pathology.

FEATURE	WHAT IT NAMES	THE DISCRIMINATING MARKER
Lack of remorse or guilt	No genuine bad feeling after wrongdoing	Regret only when caught does NOT count
Callous, lack of empathy	Disregard for others' feelings	Cold toward those they have harmed, not just detached generally
Unconcerned about performance	Muted drive to do well	Poor results provoke little discomfort or effort
Shallow or deficient affect	Thin or instrumental emotional expression	Feelings shown superficially or switched on for gain

The four features set against the marker that separates each from an ordinary adolescent equivalent; the "only when caught" qualifier on remorse is the one most often tested.

MNEMONIC

No Regret, No Feeling-for-others, No Drive, No Depth

The four features as four absences: **No Regret** (lack of remorse or guilt), **No Feeling-for-others** (callous, lack of empathy), **No Drive** (unconcerned about performance), and **No Depth** (shallow or deficient affect). Holding them as a quartet of deficits, rather than one vague idea of coldness, is what lets you demonstrate that at least two are present.

3.4 Assessment: why a single interview cannot make the call

The specifier is the one place where self-report is least trustworthy. There is a structural problem in assessing these traits, and the manual is explicit about it: the very features being sought, low guilt and a willingness to present a self-serving surface, make the young person an unreliable narrator of their own callousness. Some will minimise, some will manage the clinician's impression, and few will volunteer that they do not care about the people they hurt. For this reason the assessment cannot rest on the clinical interview or on self-report alone. It requires information from others who have known the individual over extended periods, and their observations are weighed alongside, and often above, what the young person says of themselves.

- parents and other caregivers, who see the child across the domestic and emotional life a clinic never observes;
- teachers, who see performance, peer conduct and response to correction over months;
- the extended family, peers and, in older adolescents, co-workers, who see the pattern in relationships the clinician has no access to;
- direct observation and history, set against self-report rather than replaced by it.

GOOD TO REMEMBER

Treat a bare clinic interview as insufficient to apply or exclude this specifier. If you cannot corroborate the affective pattern with someone who has watched the young person over time and in more than one setting, the honest position is that the specifier is not yet established, not that it is absent. Building the multi-informant picture into the assessment from the start saves you from a judgement the data cannot support.

3.5 Who qualifies, and why it matters for the outlook

The specifier identifies a smaller, harder subgroup. Only a minority of young people with conduct disorder meet the threshold for limited prosocial emotions, and those who do are not a random slice of the diagnosis. The specifier is more likely to be present in the childhood-onset presentation and in cases carrying a severe severity rating, which is to say it concentrates in exactly the group whose conduct problems are already the most entrenched. This is why it earns its place clinically rather than merely descriptively. Callous-unemotional traits function as one developmental pathway into persistent antisocial behaviour, and as a recognised predictor of the poorer long-term trajectory, both of which are developed where this chapter takes up etiology and prognosis. The traits also colour treatment expectations: interventions that lean on the child's attunement to others' distress and on a wish to please have less to work with when empathy and the drive to perform are precisely what is thinned, which is one reason the management of these cases is harder and the response often more limited. None of that lowers the therapeutic obligation; it sharpens what the clinician must plan for.

3.6 Where the two classifications part: scope of the qualifier

A material difference the Indian candidate must hold. The American manual attaches the limited prosocial emotions specifier to conduct disorder alone. The international classification takes a wider view: its equivalent **with limited prosocial emotions** qualifier may be applied across the whole grouping of disruptive behaviour and dissocial disorders, and in particular it may be applied to oppositional defiant disorder (**6C90**) as well as to conduct-dissocial disorder. This is not a cosmetic wording change. It reflects a genuine difference of view about where the callous-unemotional dimension belongs: as a modifier reserved for the full conduct syndrome, or as a cross-cutting affective specifier that can qualify the milder oppositional presentation too. For an examination governed by the international system, being able to state that the qualifier reaches oppositional defiant disorder in that scheme, and does not in the American one, is the discrimination that is actually asked.

AXIS	DSM-5-TR	ICD-11
Name of the marker	Specifier: with limited prosocial emotions	Qualifier: with limited prosocial emotions
Where it may be applied	Conduct disorder only	All disruptive behaviour and dissocial disorders
Reaches oppositional defiance?	No	Yes, including oppositional defiant disorder (6C90)

The same affective construct, differently scoped; the load-bearing contrast is that the international scheme lets the qualifier modify oppositional defiant disorder while the American one confines it to conduct disorder.

IN INDIA

Because Indian services classify under the international system, the operative form in case files and reports is the qualifier that may sit on any disorder in the disruptive-behaviour grouping, not the conduct-disorder-only specifier of the American manual. In practice this means the callous-unemotional pattern should be actively considered, and documented, even in a young person carrying only an oppositional defiant diagnosis, and not held back until the full conduct syndrome is reached. The wider scope is a prompt to look for the affective deficit earlier, which fits the pathway on which these traits are known to precede the more entrenched picture.

3.7 The traps and the near misses

Most errors here are errors of over-attribution or of mistaken equivalence. Three confusions recur. The first is reading regret-on-being-caught as remorse; the specifier explicitly requires that the absence of guilt be genuine, so contrition that appears only once consequences arrive counts toward the feature, not against it. The second is equating the specifier with a formal adult diagnosis: callous-unemotional traits in a child foreshadow, but are not the same as, the interpersonal-affective dimension of the adult antisocial personality, which is taken up in the Personality disorders notes, and applying an adult label to a child on the strength of these traits is a category error. The third is confining the assessment to the consulting room, where the young person controls the impression and the traits are least likely to declare themselves.

TRAP **Remorse only when caught is not remorse.** Candidates count a display of regret after discovery as evidence against the lack-of-guilt feature, and mark the specifier absent. The criterion is the opposite way round: guilt that surfaces only once the young person is caught does not rescue them from the feature, because the feature is about the absence of genuine, unprompted bad feeling about the wrongdoing itself.

PITFALL

A quieter clinical error is diagnosing the callous-unemotional pattern from a single hostile interview, in which a frightened or defended adolescent presents as flat and indifferent because that is how they cope with the encounter, not because it is how they characteristically feel. Detachment produced by the assessment situation is not the trait the specifier names, and treating one guarded meeting as sufficient evidence is how the label gets over-applied to children who do not warrant it.

2 of 4	12 months	all DBD
FEATURES NEEDED FOR THE SPECIFIER	MINIMUM PERSISTENT DURATION	SCOPE OF THE ICD-11 QUALIFIER

4 · Etiology and risk factors of conduct disorder

There is no single cause, and the examinable skill is holding several at once. The causes of conduct disorder are asked about precisely because a candidate who reaches for one explanation -

bad parenting, bad genes, bad company - has already failed. The disorder is the end-point of a developmental system in which an inherited liability, a temperament, a family, a peer group and a neighbourhood act on one another over years, and no term in that chain is sufficient on its own. What earns the marks is the ability to name the contributors at each level, to say how they combine rather than merely coexist, and to keep the honest uncertainty that the evidence itself carries: much of what is measured is a correlate of the disorder, not a proven cause of it.

This topic owns the etiology and the risk factors: the heritable liability and what is actually transmitted, the gene-environment interplay, the temperamental and neurocognitive substrate, the family and social adversities, the self-amplifying family process, the two developmental trajectories, the callous-unemotional route, and the neurobiological correlates. The diagnostic criteria and the onset subtypes belong to the definition of the disorder and are developed in their own right elsewhere in this chapter; the callous-unemotional specifier and its features have their own section; the trajectory into adult antisocial personality disorder, and the treatments that follow from these causes, are each taken up separately. Here those are named where the causal story demands them, not reopened.

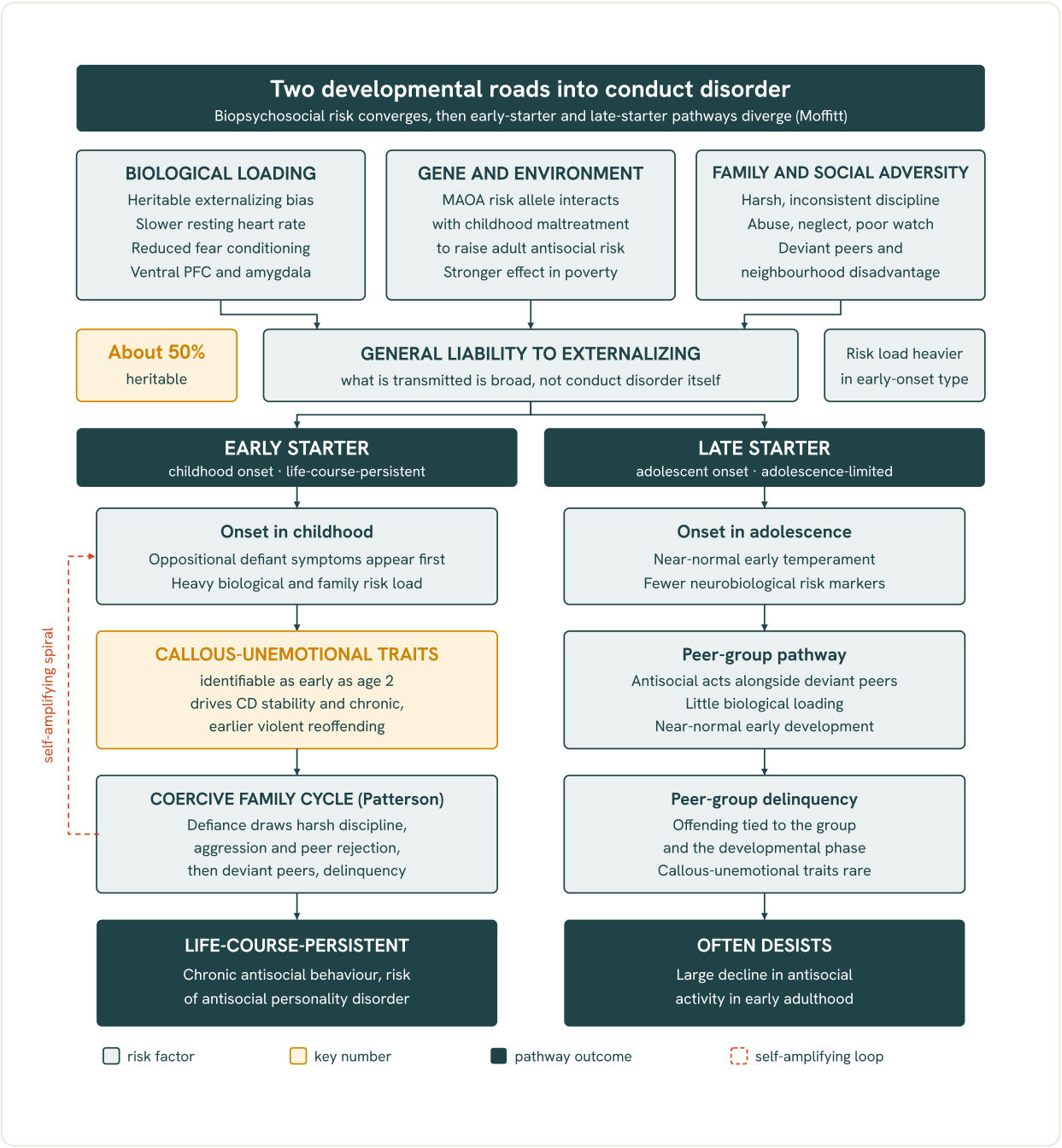

Fig 23.4. Developmental pathway to conduct disorder: biopsychosocial risk converges on a general externalizing liability, then diverges into an early-starter, life-course-persistent road and a late-starter, adolescence-limited road.

4.1 Etiology as a system, not a cause

The right frame is a developmental cascade, not a checklist of causes. Conduct disorder is best understood as the visible result of risk accumulating and compounding across development. A vulnerability present early, whether constitutional or temperamental, shapes how the child acts on the world; those actions shape how caregivers, teachers and peers respond; and those responses feed back to entrench or to soften the original vulnerability. The same starting liability can therefore end in very different places depending on what the environment does with it, which is why identical twins are discordant and why prevention is possible at all. This is not a soft statement that everything matters. It is the specific claim that the causes are probabilistic and interactive, so that any single factor raises risk without determining outcome, and the clinician's job is to read where in the cascade a given child sits and which links are still movable.

RULE **Conduct disorder has no single cause; it is the outcome of interacting risks across development.** Genetic liability, temperament, family process, peers and community each raise probability without any one being sufficient or necessary, so an etiological formulation names contributors at every level and, more importantly, says how they combine. A candidate who offers one cause has answered a different question.

4.2 The heritable liability, and what is actually inherited

Antisocial behaviour is substantially heritable, but heritability is not diagnosis-specific. Twin and adoption studies put the **heritability** of the antisocial behaviour underlying conduct disorder at **about 50%**, which sounds like a strong genetic case until the second half of the finding is added. What appears to be transmitted is not conduct disorder as a discrete entity but a broad, general liability to the externalizing disorders as a family, the shared disposition that also surfaces as oppositional behaviour, attention-deficit/hyperactivity disorder and, later, substance use and antisocial personality. The specifically conduct-disordered shape of that liability is contributed largely by the shared environment rather than by genes. This is why the same familial loading can express as several different externalizing outcomes in different children, and why a family history is a risk marker for the class of problems rather than a prediction of this one diagnosis. Reading heritability as if a gene for conduct disorder were being passed down is the commonest way to misuse the number.

> Carry this as the spine of the genetics: heritability of the underlying antisocial behaviour is about 50%, but what is inherited is a general liability to externalizing disorders, with the shared environment shaping it specifically into conduct disorder.

4.3 Genes and environment interact, they do not merely add

The genome sets a reactivity to experience, not a fixed destiny. The most instructive genetic findings in this field are not main effects of any gene but interactions in which a genotype changes how strongly an environmental adversity bites. The classic example concerns an allele of the **monoamine oxidase A (MAOA)** gene, which was found to raise the risk of adult antisocial behaviour specifically among individuals who had experienced childhood adversity, while carrying little consequence for those who had not. The gene did not code for antisociality; it conditioned the impact of maltreatment. The same logic appears at the population level, where genetic influences on problem behaviour are stronger in children growing up in poor families than in affluent ones - adversity does not swamp the genes, it unmasks them. For the exam this reframes the whole nature-nurture question: the two are not competing percentages to be apportioned but partners whose product is what matters, and an intervention that changes the environment can blunt a genetic risk that would otherwise have been expressed.

IN INDIA

The environmental half of that interaction is not evenly distributed. Overcrowding, large family size, harsh corporal discipline as an accepted norm, parental alcohol use, disrupted or absent caregiving and periods of institutional living are all more prevalent in the settings many Indian children grow up in, and the gene-environment finding implies that an identical inherited liability will be expressed more readily where such adversity is dense. The practical reading is optimistic rather than fatalistic: because so much of the operative risk is environmental and modifiable, the leverage sits in the family and the neighbourhood, and the wider machinery of child protection and welfare services relevant to these children is taken up in the Child psychiatry: assessment, treatment, services and protection notes.

4.4 Temperament and the neurocognitive substrate

The child brings something to the interaction from the start. Long before any conduct problem is diagnosable, some children show a temperament that raises risk: a **difficult, undercontrolled** infant style, marked by high negative emotionality, poor regulation of arousal and low behavioural inhibition, is one of the earliest measurable contributors. Alongside it sits a neurocognitive profile in which lower verbal IQ is characteristic - a relative weakness in language and verbal reasoning that impairs the capacity to talk problems down, to represent consequences in words, and to profit from purely verbal correction. What separates such a temperament from ordinary difficultness, and what counts as within the normal range of infant reactivity, belongs to the Normal child development and milestones notes; the point here is that these constitutional features are not the disorder but a soil in which it grows more easily. A hard-to-soothe, verbally weaker child evokes harsher and less consistent handling, does worse at school, and is more readily pushed toward the aggressive and rule-breaking solutions that the later diagnosis is built from. Temperament is thus both a direct risk and, more importantly, the child's opening move in the cascade that follows.

PITFALL

The clinical error here is to collapse into one side of the causal story. A family in crisis invites the verdict that the conduct is simply a product of bad parenting, which erases the child's heritable and temperamental contribution and the way a difficult child shapes the adults around them; the opposite error reads a slow-to-soothe, verbally weak child as constitutionally destined and quietly abandons the family work that is the most modifiable link in the chain. Both are reductions. The formulation has to hold the child's disposition and the family's response as reciprocal, because that is where the intervention actually acts.

4.5 Family and social adversity

The environmental risk factors are the densest and, mercifully, the most modifiable. The evidence identifies a consistent cluster of family-level adversities that raise the risk of conduct disorder and, importantly, are more common and more severe in the earlier-onset presentation. They are worth knowing as a family rather than as isolated items, because they tend to travel together and to reinforce one another.

- parental rejection and neglect, and inconsistent, unpredictable child-rearing;
- harsh or erratic discipline, and physical or sexual abuse;
- lack of supervision and monitoring of the child's whereabouts and company;
- early institutional living and frequent changes of caregiver, disrupting stable attachment;
- large family size and parental criminality in the home.

Beyond the household sit the community-level factors, which take over as the child moves outward into the social world: peer rejection in the early school years, subsequent affiliation with a delinquent peer group, neighbourhood disadvantage, and exposure to violence in the surrounding environment. The developmental sequence matters as much as the list: family adversity dominates the early years and peer and neighbourhood factors gather force through middle childhood and adolescence, which is why the same risk factors carry different weight at different ages.

DOMAIN	REPRESENTATIVE FACTORS	CLINICAL LEVERAGE
Temperamental / neurocognitive	Difficult undercontrolled temperament; lower verbal IQ	Largely fixed; guides how, not whether, to intervene
Family	Rejection, neglect, harsh or inconsistent discipline, abuse, poor supervision, caregiver instability, parental criminality	The most modifiable link; target of parenting work
Community	Peer rejection, delinquent peer group, neighbourhood disadvantage, exposure to violence	Partly modifiable; the focus of wider system work

The risk factors sorted by level and by how much the clinician can move them; the family row carries the most leverage, which is why the treatments that follow are built around it.

4.6 The coercive family process

Risk is not only a set of factors but a self-amplifying process between child and parent. The single most useful mechanistic model of how disruptive behaviour is built and maintained is the **coercive** family process described by Patterson. Its power is that it is dynamic rather than static: early defiance in the child provokes harsh and inconsistent discipline from the parent, which breeds further aggressiveness and, at school, peer rejection; the rejected, aggressive child then drifts toward association with deviant peers, and from there into antisocial acts, substance use and conflict with the law. Each step is both a consequence of the last and a cause of the next, so the chain tightens on itself over time. This model is not academic. It explains the observed dose-response relationship, in which the higher the number and variety of disruptive behaviours a child shows, the worse the adult outcome, because a busier, broader early pattern feeds a faster-turning coercive loop. It is also the explicit rationale for parent management training, the intervention that tries to interrupt the cycle at the parent-child step, whose detail belongs to the treatment section of this chapter.

MNEMONIC

Defiance draws discipline, discipline drives deviance

The coercive loop in three beats: a child's early **defiance** pulls harsh, inconsistent **discipline** from an overwhelmed parent, which breeds aggression and peer rejection and pushes the child toward **deviant** peers, antisocial acts and trouble with the law. Holding the loop as a cycle rather than a list is what makes the point that it can be interrupted.

GOOD TO REMEMBER

The etiology is not just background - it prescribes the treatment. Because so much of the operative risk lives in a modifiable family process rather than in fixed constitution, the first-line response to a young child with conduct problems works on the parent-child interaction, not on the child in isolation. When a formulation names the coercive cycle, it has already named where the intervention must act.

4.7 Two developmental trajectories

The same behaviour can sit on two very different developmental paths. One of the most robust organizing ideas in the field is Moffitt's distinction between a **life-course-persistent** path and a desisting, **adolescence-limited** one. The life-course-persistent group carries the heavier causal load: it is predicted by early onset, with oppositional defiant symptoms typically appearing first, by greater severity of the conduct problems, and by dense exposure to the risk factors already described. Its antisocial behaviour begins early, runs on across development, and is the pattern that carries forward. The adolescence-limited group looks similar at the peak but has a different origin and a different fate - it arises against a more normative background, is more a phenomenon of the adolescent phase and its affiliations, and desists. Consistent with this, a large decline in delinquent and antisocial activity is commonly seen as young people move into early adulthood, which reflects the adolescence-limited majority ageing out rather than everyone improving. Distinguishing the two prospectively is imperfect, but the predictors of persistence - early onset, severity, and the weight of accumulated risk - are exactly what the etiological assessment is trying to read.

AXIS	LIFE-COURSE-PERSISTENT	ADOLESCENCE-LIMITED
Onset	Early, oppositional symptoms first	In adolescence
Predicted by	Early onset, severity, dense risk exposure	Adolescent phase and peer affiliation
Background	Heavy constitutional and family risk	More normative
Course	Persists across the life course	Desists, with a large decline in early adulthood

The two pathways set side by side; the load-bearing difference is that early onset, severity and accumulated risk mark the persistent path, while the adolescence-limited majority accounts for the marked decline in antisocial activity in early adulthood.

4.8 The callous-unemotional pathway

A distinct early route runs through low affective empathy rather than through adversity. Not every child on the persistent path arrives there by the coercive, adversity-driven route. A separable developmental pathway runs through **callous-unemotional traits** - a style of low empathy, shallow affect and limited guilt that can be identified as early as **2 years of age** and appears to be largely responsible for the stability of conduct disorder in the children who show it. This pathway is more heritable and less obviously a product of harsh parenting than the coercive route, which is part of why it is worth separating out: adolescents with these traits are at higher risk of earlier and more chronic violent reoffending, so the pathway carries real prognostic weight. The trait cluster is formalised as a diagnostic specifier with defined features, which are taught in their own section of this chapter; the etiological point here is only that it names a second, affect-based road into persistent conduct disorder that sits alongside, and can combine with, the adversity-and-process road of the earlier subtopics.

4.9 Neurobiological and psychophysiological correlates

There are reproducible biological markers, and the trap is to over-read them. Several biological findings recur reliably in conduct disorder. The most striking is autonomic: a **slower resting heart rate** has been found consistently and is notable for not being characteristic of any other mental disorder, which makes it the field's most specific psychophysiological signature. Alongside it, reduced autonomic fear conditioning - indexed by low skin conductance responses - is well documented, fitting a picture of blunted responsiveness to cues of threat and punishment that would ordinarily restrain rule-breaking. Structural and functional neuroimaging points to differences in the ventral prefrontal cortex and the amygdala, regions central to the appraisal of emotion, threat and reward and to the inhibition of prepotent responses. These findings cohere into a plausible account of a nervous system that is under-aroused and under-deterred, and they matter for understanding mechanism. What they are not is diagnostic: none of them is sensitive or specific enough to identify the disorder in an individual, and they belong to the explanation of conduct disorder, not to its detection.

TRAP **The biological markers explain, they do not diagnose.** Candidates recite the slow resting heart rate, the low skin conductance and the ventral-prefrontal and amygdala findings as though they confirmed the disorder. They are group-level correlates, robust in the aggregate but neither sensitive nor specific in the individual; the diagnosis remains clinical, and presenting any of these as a diagnostic test is the error the examiner is watching for.

~50%	2 years	slower
HERITABILITY OF THE UNDERLYING ANTISOCIAL BEHAVIOUR	AGE FROM WHICH CALLOUS-UNEMOTIONAL TRAITS ARE IDENTIFIABLE	RESTING HEART RATE, A MARKER SEEN IN NO OTHER DISORDER

5 · Assessment and clinical evaluation of conduct disorder

A conduct disorder assessment is won or lost on where the information comes from, not on how the child behaves in the room. Assessing **conduct disorder** is a distinct clinical skill, separate from knowing the criteria, because the young person in front of you is the one

informant with the least incentive to describe the problem accurately. Children and adolescents with conduct disorder characteristically minimise their conduct problems, so a clinician who takes the interview with the child as the primary evidence base will systematically underestimate the disorder. The whole method is built to correct for that: it draws on several informants who each see the young person in a different setting, it treats disagreement between them as informative rather than as noise, and it anticipates that the young person, and sometimes the family, will not cooperate freely.

The marks here reward candidates who can describe a defensible process rather than recite a checklist. This topic owns the evaluation method: how a reliable diagnosis is assembled from multiple sources, how the purpose of the referral changes the way the assessment is conducted, what history domains and functional understanding must be covered, and the standardised instruments that quantify and track the picture. The diagnostic criteria and subtypes, the callous-unemotional specifier, the causes and risk factors, the treatments, and the long-term trajectory are each developed in their own right elsewhere in this chapter and are named here only where the assessment reaches for them.

5.1 The purpose of the referral shapes the whole assessment

Ask first who wants this assessment and why. Conduct disorder is unusual among child psychiatric presentations in how often the request originates outside the family. An evaluation may be requested by **parents, schools, courts, children's lawyers or social services**, and each of those origins carries a different question, a different standard of proof, and a different limit on confidentiality. A parent asking why their child is unmanageable wants a clinical formulation and a plan. A court or a child's lawyer wants an opinion that may be tested adversarially and disclosed, which changes what you can promise the young person about privacy before you begin. Establishing the purpose at the outset is therefore not a courtesy but a structural decision: it determines who you will speak to, what you will document, what you can keep confidential, and how you frame the evaluation to the child and family. A clinician who begins gathering history without settling this can find that the confidentiality they implied cannot be honoured, or that the assessment they conducted does not answer the question that was actually asked.

IN INDIA

A large share of conduct disorder assessments reach services through schools and through the statutory child-welfare and juvenile-justice route rather than through parents seeking help, which means the confidentiality and consent frame is frequently set by an institution before the child arrives. The psychiatric perspective on juvenile delinquency is taken up separately in this chapter, and the wider machinery of child protection, welfare committees and services sits in the Child psychiatry: assessment, treatment, services and protection notes. The practical lesson for the assessment itself is that the referral source is often a third party with its own agenda, so clarifying whose question you are answering, and what will be shared with whom, is the first task in the room.

5.2 The multi-informant, collateral method

No single informant is sufficient, and the child least of all. The reliable diagnosis of conduct disorder rests on **an evaluation by an experienced clinician drawing on several sources**, typically the child, the parents and the teachers, supplemented by questionnaire data. The reason this is doctrine rather than preference is the informant problem set out at the start: because the young person minimises, the clinician must actively seek out additional informants, and because behaviour is situation-specific, an informant who sees the child in only one setting gives only part of the picture. Parents see the home; teachers see the classroom and the playground; the child alone can report internal states, fears and motives that no observer can access. The skill lies in holding these accounts together. Reports from different informants frequently disagree, and the beginner reads that disagreement as one source being wrong. It is better understood as each source being right about a different context: a child who is compliant at school and defiant at home, or the reverse, is telling you something real about where the behaviour is triggered and maintained. The accounts complement each other precisely because they diverge.

RULE **Diagnose conduct disorder from collateral sources, never from the child's self-report alone.** The young person minimises the very behaviour under evaluation, so an experienced clinician assembles the picture from parents, teachers and the child together, supplemented by questionnaires. Discrepant reports are not a problem to resolve down to one true version; they map where the behaviour is and is not expressed, and that map is itself diagnostic and treatment-relevant.

Expect resistance and plan for it. Because the referral is often involuntary and the behaviour often brings the young person into conflict with authority, noncooperation and resistance are to be expected rather than treated as a complication that derails the assessment. A refusal to engage, a flatly minimising account, or open hostility is part of the presentation, not a failure of the interview, and the collateral method exists so that the evaluation does not collapse when the child will not talk. The experienced assessor builds the diagnosis from what the informants and instruments provide and reads the young person's reluctance as data about the therapeutic task ahead.

5.3 History domains and the functional understanding

Map the behaviour onto its context and its function. A conduct disorder history has to reach well beyond the catalogue of behaviours into the environment that shapes them. The domains that must be explored include **parenting style, the quality of parent-child interactions, inconsistent parenting, maternal depression, parental drug use, and the possibility of sexual abuse**. These are asked about here not to settle causation, which belongs to the discussion of etiology and risk factors elsewhere in this chapter, but because they are the modifiable context in which the behaviour is embedded and because some of them, notably abuse and a depressed or substance-using parent, are urgent findings in their own right. Alongside the content of the history sits its logic. A good assessment builds a **functional understanding** of the behaviour: what antecedents reliably precede it and what consequences follow and maintain it. Reading each problem behaviour as a link in an antecedent-behaviour-consequence chain turns a list of

complaints into an account of why the behaviour persists, and it is this functional account, not the diagnostic label, that points toward what an intervention should target.

PITFALL

The recurring clinical error is to assess the child and skip the context, producing a diagnosis that names the behaviour but explains nothing and guides no treatment. A behaviour that looks like pure defiance often turns out, once the antecedents and consequences are traced, to be maintained by an inconsistent contingency at home or by escape from an intolerable classroom demand. Stopping at the label, without the functional analysis and without probing parenting, parental mental illness and possible maltreatment, both misses treatable maintaining factors and risks leaving a safeguarding concern unaddressed.

5.4 Assessing across all symptom domains: comorbidity

Conduct disorder rarely travels alone, and the comorbidity is often the more treatable half. A competent evaluation deliberately covers all symptom domains so that comorbid disorders are not overlooked, because the conditions that accompany conduct disorder are frequently what respond best to treatment. The three that must be actively screened for are attention-deficit/hyperactivity disorder, mood disorders and substance misuse. The overlap with ADHD is close enough that the two are easily conflated, and its assessment and treatment belong to the ADHD and specific learning/communication disorders notes; the point for the conduct disorder evaluation is simply that it must be looked for rather than assumed absent. A depressive disorder can drive irritability and acting-out that mimics or amplifies conduct problems, and substance misuse both worsens and is worsened by the behaviour. An assessment that stops at conduct disorder because the disruptive behaviour is the loudest presenting feature will miss the disorder that is quietly doing much of the damage and is often more amenable to intervention.

- attention-deficit/hyperactivity disorder, closely overlapping and easily conflated, screened for and cross-referred rather than assumed;
- mood disorders, where irritability and acting-out may be depressive in origin;
- substance misuse, which both aggravates and is aggravated by the conduct problems.

5.5 Standardised rating instruments

Questionnaires quantify and track what the clinical interview establishes. The clinical evaluation is supplemented by structured questionnaires, chief among them the broad-band **Child Behavior Checklist**, together with narrower disruptive-behaviour scales such as the **Eyberg Child Behavior Inventory**, the **New York Teacher Rating Scale for Disruptive and Antisocial Behavior**, and the **Home and School Situations Questionnaire**, the last of which locates where the behaviour occurs. Their value is specific and bounded. Questionnaires provide quantifiable data, reliably supplement the interview, and are useful for measuring progress and outcome over the course of treatment, which is something a narrative interview does poorly. What they do not do is replace the multi-informant clinical evaluation. A score is a number attached to a rater's perception in a setting, and it acquires meaning only when the clinician

integrates it with the rest of the picture. The instruments are the measuring tape, not the diagnosis.

INSTRUMENT	TYPE	WHAT IT ADDS
Child Behavior Checklist	Broad-band	Wide behavioural and emotional coverage across informants
Eyberg Child Behavior Inventory	Disruptive-behaviour	Focused measure of conduct problems and their intensity
New York Teacher Rating Scale	Teacher-report	The classroom view of disruptive and antisocial behaviour
Home and School Situations Questionnaire	Situational	Where the behaviour occurs, across home and school settings

The instruments that supplement the interview, each contributing a different angle; none substitutes for the clinician's integration of the multiple accounts.

TRAP **A high questionnaire score is a screen, not a diagnosis.** Candidates treat a raised Child Behavior Checklist or a positive rating scale as if it made the diagnosis, when the instruments only quantify a rater's perception in one setting and are properly used to supplement the interview and to track outcome. The diagnosis remains the experienced clinician's integration of the collateral accounts; the number measures change, it does not replace judgement.

5.6 The Strengths and Difficulties Questionnaire

The workhorse behavioural screen of child mental health. The single instrument worth knowing in detail is the **Strengths and Difficulties Questionnaire**, developed by **Goodman**. It is a brief behavioural screening questionnaire of **25** items, each rated on three response options - Not True, Somewhat True and Certainly True - which makes it quick enough to use routinely. Its reach is one of its strengths: it is completed by parents and teachers, and by children and adolescents themselves as a self-report, so a single short instrument delivers the multi-informant, multi-setting perspective that the conduct disorder assessment demands. It spans roughly **ages 3 to 17**, is in the public domain, and has been translated into many languages, which is much of why it is so widely used. An optional impact supplement of up to about eight further items extends it beyond symptom counting to ask how much the difficulties actually burden the child and the family, which is often the more clinically decisive question.

5.7 SDQ structure: the five subscales and the Total Difficulties Score

The scoring architecture is examinable and elegant. The aptly named questionnaire carries both a strength scale and difficulty scales. It resolves into **five subscales of five items each**: emotional symptoms, conduct problems, hyperactivity and inattention, peer relationship problems, and prosocial behaviour. Four of these describe difficulties and one, prosocial behaviour, describes a strength, which is what the questionnaire's name announces. The scoring then does something that trips up candidates who assume all five simply add together. A **Total Difficulties Score** is generated by summing only the **four** problem subscales - emotional symptoms, conduct problems, hyperactivity and inattention, and peer relationship problems. The prosocial behaviour subscale is deliberately left out of that total and is scored separately against

its own cut point, because a low level of prosocial behaviour is conceptually a different kind of finding from a high level of difficulty. Keeping the strength scale out of the difficulty total is the design feature that distinguishes the instrument, and it is exactly the point an examiner probes.

MNEMONIC

Five by five, four make the total

Five subscales of **five** items each, and only the **four** problem scales sum to the Total Difficulties Score. The fifth scale, prosocial behaviour, is the strength and stands apart with its own cut point. The phrase fixes both the structure and the one scoring rule most often got wrong.

The must-memorise line: the SDQ Total Difficulties Score sums the four problem subscales only; the prosocial behaviour subscale is scored separately and is not part of the total.

5.8 Interpreting the scores and the banding caution

A screen produces a band, and the band is only as good as the threshold behind it. The subscale and total scores are interpreted by comparison against clinical cut points, and the familiar reporting frame sorts a child into a normal, borderline or abnormal band, with newer versions using a four-way categorisation from close-to-average through to very high. This is where discipline matters. The exact numeric thresholds that separate one band from the next are set by the instrument's own scoring instructions and are revised periodically, and they differ by informant and by version, so they are not something to reproduce from memory in a clinical or examination answer. The safe practice is to state the banding concept and to apply the current published thresholds from the questionnaire's own scoring guidance rather than to quote a boundary you cannot verify. The larger caution is the same one that governs every instrument in this topic: the SDQ, however well banded, is a screening and outcome-tracking tool. It flags children who warrant fuller evaluation and it measures change over time; it does not, on its own, make the diagnosis of conduct disorder, which remains the product of the multi-informant clinical assessment.

GOOD TO REMEMBER

Use the SDQ to open and to follow a case, not to close it. Its brevity, its public-domain availability, its coverage across parents, teachers and the young person, and its impact supplement make it an efficient front door and an honest outcome measure. Record a baseline, treat, and re-administer to show whether the difficulties have actually moved, but let the diagnosis rest on the integrated clinical picture and confirm any band you report against the questionnaire's current thresholds.

25	5 by 5	3 to 17
SDQ ITEMS	SUBSCALES, FIVE ITEMS EACH	AGE RANGE IN YEARS

6 · Management of conduct disorder (parent training, multisystemic therapy, pharmacotherapy)

The treatment of conduct disorder is where the diagnosis earns its keep, and where the commonest instinct is the wrong one. Faced with an aggressive, rule-breaking young person, the pull toward a sedating prescription is strong, and it is almost always the second move rather than the first. The management of conduct disorder is built on a settled principle: the disorder is a problem of behaviour embedded in relationships and systems, so the treatments that change it are the ones that work on those relationships and systems. Most guidelines concur that structured psychosocial and behavioural interventions are the first line of treatment for the disruptive behaviour disorders, oppositional defiant disorder and conduct disorder alike, and that these interventions continue even where medication is later added. Marks accrue on this topic because it rewards a candidate who can name the right intervention for the right age, explain why it works, and place medication precisely where it belongs, which is at the margin and never at the centre.

This topic owns the management of conduct disorder in full: the treatment hierarchy, parent management training as the core intervention for the younger child, multisystemic and community interventions for the adolescent, and the adjunctive, aggression-directed role of medication with its dosing and monitoring. The diagnostic criteria and subtypes, the callous-unemotional specifier, the causes and risk factors, the assessment, and the long-term trajectory into adult antisocial outcomes are each developed in their own right elsewhere in this chapter and are named here only where the treatment reasoning demands them.

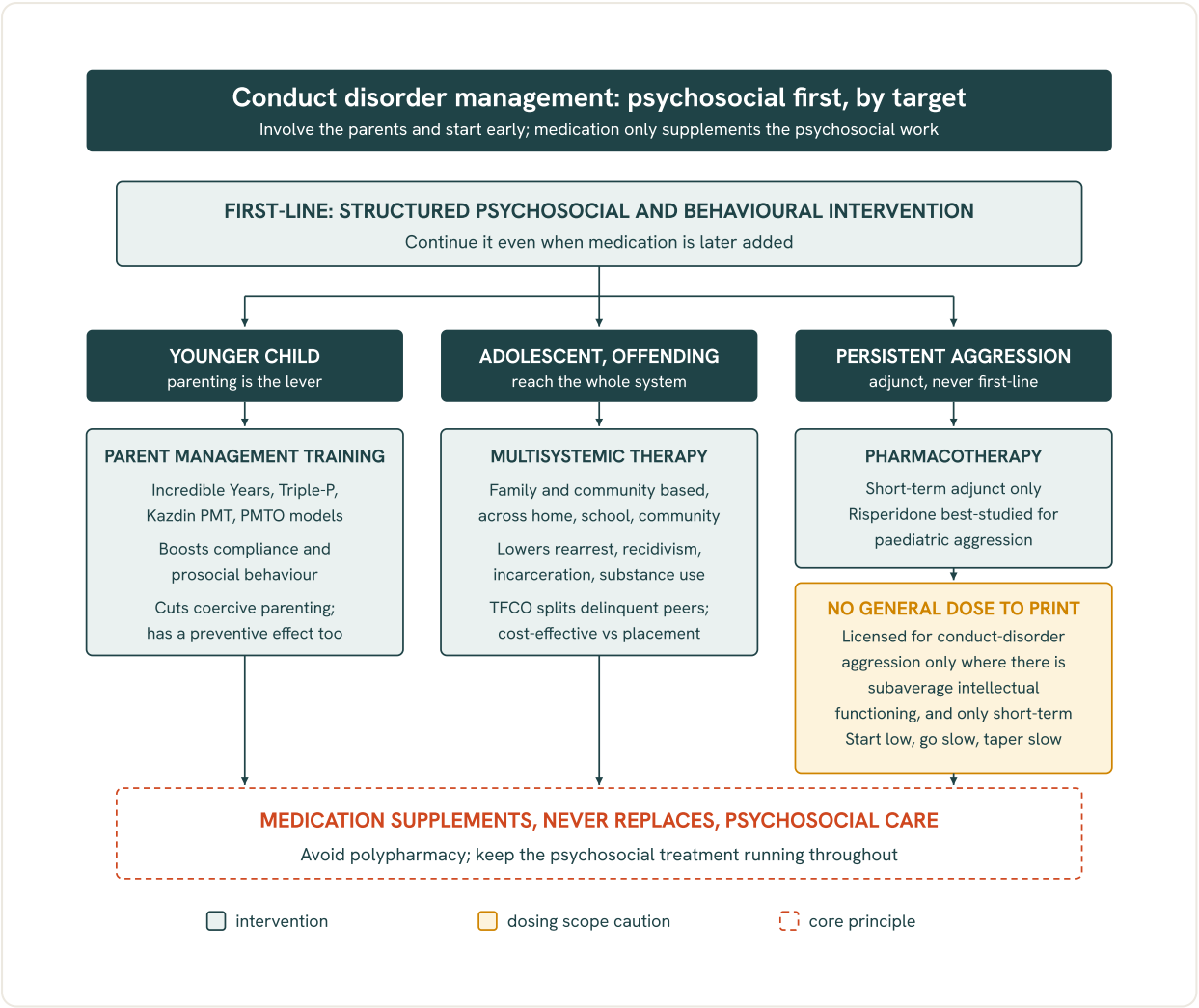

Fig 23.5. Conduct disorder management ladder: psychosocial intervention first, matched to the target - parent training for the younger child, multisystemic therapy for the offending adolescent, and short-term risperidone as an adjunct for persistent aggression. No dose is printed on the plate: the only licensed conduct-disorder dose is restricted to young people with subaverage intellectual functioning, so dosing is scope-bound and is set out in the text.

6.1 The treatment hierarchy: psychosocial first, medication as adjunct

The order of operations is itself examinable. The governing rule is that treatment leads with structured psychosocial and behavioural work and reserves pharmacotherapy for defined targets on top of it. Two features of that rule are easy to lose. The first is that the psychosocial intervention should be continued even if medication is subsequently initiated; the drug does not replace the behavioural programme, and the moment it is treated as a replacement the plan has already failed. The second is that this treatment should involve the parents, because improving parenting skills and the quality of parent-child interactions are core goals rather than incidental ones. There is also a timing effect that shapes prognosis and effort: intervention is more likely to be effective when administered early in the course, before coercive patterns and antisocial affiliations have consolidated. The clinical implication is direct. A young child brought in early is a genuine opportunity; an entrenched adolescent is harder work and the plan must be built accordingly.

RULE **Psychosocial and behavioural intervention is first-line for conduct disorder, and it continues even when medication is added.** Medication is layered onto the behavioural programme for a specific target; it never becomes the programme. An author who leads with a drug has inverted the hierarchy the evidence actually supports, and the earlier the psychosocial work begins, the more it achieves.

Carry this into the exam: for conduct disorder the first-line treatment is a structured, parent-involving psychosocial intervention, delivered early, and medication is a short-term adjunct for aggression, never a substitute for it.

6.2 Parent management training: the core intervention

The single best-evidenced treatment for the younger child works through the parent, not directly on the child. **Parent (behavioural) management training** programmes teach caregivers a set of concrete strategies that increase compliance and prosocial behaviour and reduce oppositional and aggressive behaviour. The logic is behavioural: much of the child's disruptive conduct is being inadvertently maintained by the family's own responses, and the intervention retrains those responses. Parents are coached to give clear, achievable instructions, to attend to and reinforce desirable behaviour, to use consistent and non-escalating consequences, and above all to interrupt the **coercive parenting** pattern in which harsh, inconsistent discipline and child noncompliance drive each other in an escalating loop. The change is achieved indirectly and deliberately so: rather than counselling the child, the clinician equips the parent to alter, session by session, the contingencies that surround the behaviour at home. Positive attention is shifted onto the behaviours the family wants to see, previously reinforcing responses to defiance are withdrawn, and predictable, proportionate consequences replace the cycle of threats that are not carried through. Because the parent is present for far more of the child's day than any clinician can be, a small, consistent change in the parent's response compounds across hundreds of daily interactions, which is why a treatment that never addresses the child directly can move the child's behaviour so reliably. Several programmes are well researched and share this backbone:

- Helping the Noncompliant Child, focused on younger children and instruction-following;
- Incredible Years, a structured video-based parent, child and teacher programme;
- Parent Management Training in the tradition of Kazdin, delivered to the parent as the agent of change;
- Triple-P (the Positive Parenting Programme), delivered across a tiered intensity of contact.

The evidence is not soft, and it is durable. The **Parent Management Training Oregon Model** (PMTO) reduces coercive parenting, and its benefit is measurable long after the sessions end: **nine years** post-intervention it was associated with lower teacher-rated delinquency and reduced arrest rates. Parent training is, on this evidence, an established evidence-based treatment for clinically significant disruptive and externalising behaviour, and it carries a preventive dividend as well, reducing later problems in children at risk rather than only treating those already

diagnosed. That combination, a treatment that both remediates and prevents, is why it sits at the head of the plan for the child in whom parental influence is still the dominant force.

6.3 Multisystemic therapy and the community interventions for the adolescent

When the adolescent's world has widened beyond the family, the intervention has to widen with it. By mid-adolescence the drivers of conduct problems are rarely confined to the home: school failure, delinquent peer groups, and neighbourhood factors all feed the behaviour, and a parent-only programme can no longer reach them. **Multisystemic therapy** is the intensive, family- and community-based intervention designed for exactly this situation, for antisocial behaviour in adolescent juvenile offenders. Its defining feature is that it works simultaneously across home, school and community settings rather than in a clinic room, treating the young person's behaviour as a product of the systems around them and intervening in each. The outcome data are among the strongest in this field: it is associated with decreased rates of rearrest, recidivism, incarceration, psychopathology and substance use. It is also, importantly for a resource-conscious service, a cost-effective alternative to out-of-home placement, keeping the adolescent in their own context while changing it. A related community intervention, **Treatment Foster Care Oregon** (TFCO), takes the complementary approach of placing the young person in a trained, structured foster setting that deliberately separates them from delinquent peers while the family situation is worked on. The peer emphasis is not incidental. Interventions that gather antisocial adolescents together can inadvertently strengthen the very behaviour they aim to reduce, as the group becomes a training ground for deviance and a source of mutual reinforcement, and both the multisystemic model and structured foster care are built to work with or around the peer network rather than to concentrate it. Both share the same premise that the peer group and the wider environment are not background but active ingredients of the disorder, and that an intervention confined to the individual or the clinic will not reach the systems that actually maintain the behaviour.

6.4 Matching the intervention to the developmental stage

The right treatment is a function of who still holds the levers of the child's behaviour. The two psychosocial pillars are not competitors but a developmental sequence. For the younger child, whose behaviour is still shaped chiefly within the family, the parent is the most powerful available lever, and so parent management training is the intervention of choice. For the adolescent, in whom peers, school and neighbourhood have taken on independent force and whose behaviour has often already drawn in the justice system, a family-only approach is necessary but no longer sufficient, and the multisystemic and community models that reach every system become the appropriate frame. This is why age of presentation changes the plan so decisively, and why the early-presenting child carries such a premium: the same effort applied while the family is still the dominant influence buys far more than it will a decade later. None of these interventions is a quick fix. Each asks sustained engagement of an often exhausted and demoralised family, and the clinician's task is to match intensity to the developmental reality rather than to reach reflexively for the most intensive option available.

6.5 The place of medication: adjunctive, aggression-directed, never first-line

There is no drug that treats conduct disorder; there are drugs that help a specific target within it. Medication in the disruptive behaviour disorders is deployed against aggression, and it is deployed as an addition to psychosocial treatment, not as a route around it. Antipsychotic medication for aggression in disruptive behaviour disorders is used as an adjunct in the short term and is never first-line. The framing that matters is that medication supplements, and does not replace, psychosocial treatment, and that polypharmacy should be avoided: a child accumulating agents is usually a child whose behavioural plan has been abandoned rather than one whose illness demands three drugs. Comorbidity is where pharmacotherapy earns a clearer place. Where attention-deficit/hyperactivity disorder coexists, psychostimulants help the oppositional and aggressive symptoms as the underlying ADHD is treated, which is one reason the comorbid picture must be actively sought and managed; the ADHD itself, its stimulant and non-stimulant treatment, belongs to the ADHD and specific learning/communication disorders notes.

TRAP **Treating pharmacotherapy as the primary treatment for conduct disorder.** Candidates reach for the antipsychotic as though it were the answer, because the aggression is the frightening part and a drug feels decisive. It is an adjunct for aggression only, layered onto a psychosocial programme that continues underneath it. Naming a medication before naming parent management training or multisystemic therapy is the inversion the examiner is looking for.

GOOD TO REMEMBER

Actively screen every child presenting with conduct problems for comorbid ADHD, because it is common in this group and it is treatable. Bringing the ADHD under control with a stimulant can reduce the oppositional and aggressive symptoms directly and can change the whole trajectory of the case, whereas leaving it unrecognised leaves a large, remediable driver of the behaviour untouched.

6.6 Risperidone: the best-studied agent, and the narrow scope of its licensed dose

When an antipsychotic is used there is a best-studied choice, a disciplined method, and a licence far narrower than the reflex assumes. Among the atypical antipsychotics, **risperidone** is the best-studied for aggression in paediatric populations, which is why it is the usual agent when the short-term adjunctive step is taken for severe disruptive behaviour and aggression in conduct disorder. What cannot be done is to quote a dose in the abstract, because the only licensed conduct-disorder dose in any labelling this chapter has read is tied both to a jurisdiction and to a population. The United Kingdom, the European Union and Australia license risperidone for the short-term symptomatic treatment of persistent aggression in conduct disorder, in children from five years of age and in adolescents, and only where there is subaverage intellectual functioning; the United States licenses no conduct disorder indication for risperidone at all, its paediatric approvals being schizophrenia, bipolar mania and irritability associated with autistic disorder. The restriction to subaverage intellectual functioning is part of the indication itself and can never be dropped when the dose is quoted.

Within that licensed population the labelled oral regimen is weight-banded and once daily, and it sits far below the antipsychotic doses used for paediatric schizophrenia. For a child under 50 kg the starting dose is **0.25 mg once daily**, increased in steps of 0.25 mg no more often than every other day, to an optimum of 0.5 mg once daily within a licensed range of 0.25 to 0.75 mg once daily. For a young person of 50 kg or more the starting dose is **0.5 mg once daily**, increased in steps of 0.5 mg no more often than every other day, to an optimum of 1 mg once daily within a licensed range of 0.5 to 1.5 mg once daily. The oral solution is the formulation that delivers the 0.25 mg and 0.75 mg doses, which the tablet cannot, and the licensed treatment period is **up to six weeks**, with any continuation evaluated and justified on an ongoing basis. The label itself carries the hierarchy this topic teaches: the drug is to be an integral part of a more comprehensive treatment programme including psychosocial and educational intervention, prescribing is recommended to be by a specialist in child neurology or child and adolescent psychiatry, and physical and social causes of the aggression, such as pain or an inappropriate environmental demand, are to be assessed before any prescription is written. In renal or hepatic impairment the starting and consecutive doses are halved and titration is slower. Risperidone is not recommended below five years of age, where there is no experience.

The gap that remains is the point, not a deficiency in the teaching. Outside that licensed population, which is to say for the young person who has conduct disorder with normal intellectual functioning and who is the subject of this topic, no label this chapter has read supports a risperidone dose; use there is off-label and unlicensed, no number is printed for it here, and the dosing of the antipsychotics themselves belongs to the antipsychotics chapter. Intellectual disability as a construct, and the separate risperidone indication for irritability in autism, are developed in the Intellectual disability and autism spectrum disorder notes; they are named here only because the licence for conduct-disorder aggression happens to be written around the first of them. What survives all of this unchanged is the method. The dose is reached, and later withdrawn, cautiously: begin low, increase gradually, and taper slowly when stopping, with routine monitoring throughout, so that both efficacy and adverse effects are tracked deliberately rather than discovered by accident. Because this is an antipsychotic in a growing child, the monitoring is specific: weight and metabolic parameters, and prolactin, are followed, since weight gain, metabolic disturbance and hyperprolactinaemia are the liabilities that most often force a change of plan.

MNEMONIC

Start low, go slow, taper slow

The dosing discipline for the aggression-directed antipsychotic in a child: **start low** at the bottom of the range, **go slow** in titrating upward against response and tolerability, and **taper slow** rather than stopping abruptly, all under routine monitoring. The phrase encodes the whole safety posture of adjunctive pharmacotherapy in this population.

PITFALL

Starting risperidone and then not monitoring for its metabolic cost. It is easy to prescribe and it does reduce aggression in the short term, so the follow-up drifts and the weight, metabolic indices and prolactin go unchecked. The commonest real-world harm from this agent in children is not treatment failure but unmonitored metabolic and endocrine adverse effects accumulating quietly on a drug that was only ever meant to be a short-term adjunct.

6.7 Assembling the plan, and the Indian reality

A defensible management answer names the whole staged plan, not one favourite piece of it. Put together, the plan begins with a thorough assessment of the young person and the family, leads with the psychosocial intervention matched to developmental stage, treats comorbidity actively, and adds aggression-directed medication only as a short-term adjunct with proper monitoring, keeping the behavioural work running throughout. The accompanying figure sets the modalities against the population each fits, the setting each works in, and the role each plays, and the table below does the same in summary form.

MODALITY	BEST SUITED TO	WORKS ACROSS	ROLE IN THE PLAN
Parent management training	The younger child	The family	First-line; retrains coercive interactions
Multisystemic therapy	The adolescent offender	Home, school and community	First-line for the older youth; a cost-effective alternative to out-of-home placement
Treatment Foster Care Oregon	The adolescent needing placement	A structured foster setting	Separates the young person from delinquent peers
Risperidone and related agents	Marked aggression on top of the above	Added to psychosocial care	Short-term adjunct only; never first-line

The four building blocks of management set against who each fits and what each contributes; the psychosocial rows lead, and the pharmacological row is deliberately last and conditional.

IN INDIA

The evidence base is Western, and the delivery gap is the exam-relevant point. Manualised parent management training programmes and, still more, multisystemic therapy and structured therapeutic foster care are largely unavailable in routine Indian services, which are thinly staffed and family-dependent. In practice the psychosocial first line is often delivered as pragmatic parent guidance, psychoeducation and school liaison built around the same behavioural principles rather than as a branded programme, and much of the work falls to families themselves. The mismatch this creates is a real risk: because an antipsychotic is cheap, quickly available and requires only a prescription, the temptation to lean on medication in place of the harder psychosocial work is greater here than in better-resourced settings, and that is precisely the inversion of the hierarchy the treatment principles warn against. One caution about scope belongs here rather than in a footnote: the licensing positions named above are those of the United Kingdom, the European Union, Australia and the United States, and the Indian labelling position for risperidone in conduct disorder is not stated in these notes because it was not verified for them. A prescriber here should read the current Indian package insert rather than assume that any of those licences transfers, and should not treat the drug's easy availability as evidence that a licence exists. Where conduct problems bring a young person into contact with the law, the child-protection and juvenile-justice machinery is handled in the Child psychiatry: assessment, treatment, services and protection notes.

nine years DURABILITY OF THE PMTO BENEFIT	**six weeks** LICENSED CAP ON ADJUNCTIVE RISPERIDONE, IN THE ONE POPULATION LICENSED FOR IT	**None** CONDUCT DISORDER INDICATION FOR RISPERIDONE IN THE UNITED STATES	**No dose** LICENSED FOR CONDUCT DISORDER WITH NORMAL INTELLECTUAL FUNCTIONING

7 · Prognosis and link to antisocial personality disorder

Prognosis is where conduct disorder stops being a description and becomes a prediction. Having established the diagnosis, its subtypes and its severity, the examiner now wants to know what becomes of these young people, and whether the diagnosis is the childhood face of a life-long antisocial trajectory or a phase that most will grow out of. The honest answer is that both outcomes are common, and the marks lie in being able to say which child is heading which way. The single most consequential fact about the natural history of conduct disorder is that its course is heterogeneous: it is not a uniformly grim sentence, and it is not a benign rite of passage either. A large proportion of affected young people improve, while a smaller high-risk subgroup carries the disorder into an adult life of persistent offending and personality pathology. The clinical and the examinable skill is the same one - reading, from features present in childhood and adolescence, where a given young person sits on that spread.

This topic owns the trajectory itself: the overall course into adulthood, the features that predict persistence against those that predict recovery, and the specific developmental bridge that links conduct disorder to **antisocial personality disorder**. The diagnostic criteria and the onset subtypes are established in their own right elsewhere in this chapter and are named here only as prognostic markers, not re-taught. Antisocial personality disorder as a full adult diagnosis, with

its own criteria and management, belongs to the Personality disorders notes; what is developed here is the one-way developmental corridor that runs from the childhood disorder toward it, and the fact that most children never travel down it.

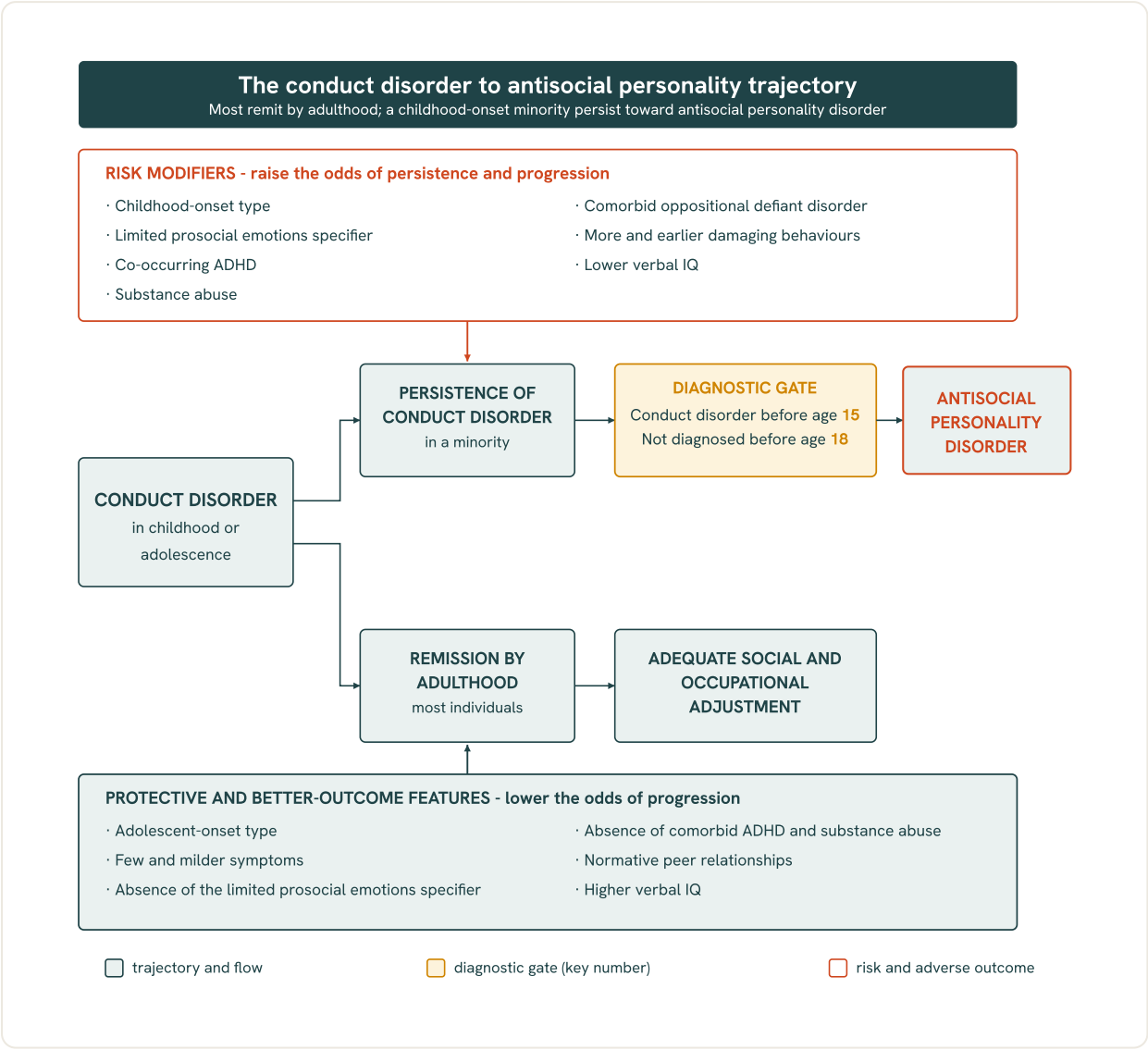

Fig 23.6. Conduct disorder to antisocial personality trajectory: most cases remit by adulthood, while a childhood-onset minority persist across the age 15 and age 18 diagnostic gates, with risk modifiers driving progression and protective features favouring remission.

7.1 The shape of the outcome: heterogeneity and remission

The default trajectory is improvement, not deterioration. The most important corrective to clinical pessimism is that in the majority of individuals the disorder **remits** by adulthood. Many young people, particularly those with the adolescent-onset course and with few and milder symptoms, go on to achieve adequate social and occupational adjustment as adults, holding down work and relationships that their teenage conduct would not have predicted. The clinical picture in early adult life is typically one of desistance: a large decline in delinquent and antisocial activity is commonly reported as the young person moves out of adolescence, matures, and acquires the stakes in conventional life - employment, a partner, responsibilities - that make offending costly and unrewarding. This does not mean the disorder is trivial, because the minority whose course does not follow this benign arc account for a great deal of harm, and

because even a self-limiting course can leave educational and legal scars. But the base rate matters enormously for how the clinician frames the problem to a family, and for resisting the pull toward writing a child off.

RULE **Conduct disorder is prognostically heterogeneous, and its commonest outcome is remission.** The disorder resolves in most affected individuals by adult life, with a marked fall in antisocial activity in early adulthood. Prognosis is therefore a question of identifying the high-risk minority who will not follow that path, not a foregone conclusion applied to everyone who carries the label.

7.2 Childhood onset as the pivot of prognosis

When the trouble began is the strongest single prognostic marker. The onset subtype is not a bookkeeping detail; it is the axis on which the outlook turns. The **childhood-onset type** predicts a worse prognosis and an increased risk of criminal behaviour, of persistent conduct disorder that does not remit on the usual schedule, and of substance-related disorders in adulthood. Alongside the age at which problems first appeared, the nature of those early problems carries weight of its own: engaging in more damaging behaviours at an early age is itself predictive of a worse outcome, so a young child whose conduct is already seriously harmful sits in a different prognostic band from one whose early problems are milder. The **adolescent-onset type**, by contrast, is the course most associated with the benign trajectory of the previous section - later onset, milder and fewer symptoms, and a stronger tendency to remit as adolescence passes. Reading onset age as a prognostic variable, and not merely as a descriptive specifier, is the reasoning a candidate is expected to show.

PITFALL

The mirror-image clinical error to over-diagnosis is therapeutic nihilism: treating a child with the childhood-onset, high-risk profile as already destined for an antisocial adult life and quietly withdrawing effort. Worse prognosis is a shift in probability, not a sealed fate. This is precisely the subgroup in which early, intensive, evidence-based intervention has the most to offer, and writing them off both misreads the evidence and forfeits the window in which the trajectory is most malleable.

7.3 What keeps the disorder going: predictors of persistence

Persistence clusters around a recognisable profile. The features that predict that conduct disorder will not remit but will run on into adult antisocial pathology are worth memorising as a set, because they recur in vignettes and in real referrals alike. **Persistence** is more likely when the young person's course meets the childhood-onset pattern and also qualifies for the limited prosocial emotions specifier - the callous-unemotional presentation whose four defining features are set out in their own right elsewhere in this chapter and are named here only as a prognostic amplifier. The risk of persistence is further increased by co-occurring **attention-deficit/hyperactivity disorder**, whose own assessment and treatment belong to the ADHD and specific learning/communication disorders notes, and by **substance abuse**, which both reflects and drives an escalating antisocial course. Comorbid ADHD and oppositional defiant disorder

each predict worse outcomes. The unifying theme is that the disorder persists when it is early, callous, comorbid and chemically fuelled: each of these adds to the load and lowers the chance of the spontaneous desistance that carries most young people out of the disorder.

- childhood-onset course rather than adolescent onset;
- the limited prosocial emotions (callous-unemotional) specifier present;
- co-occurring attention-deficit/hyperactivity disorder;
- co-occurring oppositional defiant disorder;
- substance abuse compounding the antisocial course.

MNEMONIC

Early, Callous, Hyperactive, Intoxicated

The persistence profile, in the order the features tend to appear: **E**arly onset (childhood-onset course), **C**allous (the limited prosocial emotions specifier), **H**yperactive (co-occurring ADHD, with oppositional defiant disorder alongside), and **I**ntoxicated (substance abuse). Each is a claim only about which conduct disorder runs on; none re-teaches the disorder it names.

7.4 The bridge to antisocial personality disorder

Conduct disorder is the mandatory childhood antecedent of the adult diagnosis, but it is far from a guarantee of it. The link that the examiner most wants stated precisely is the developmental corridor between the two disorders. Antisocial personality disorder cannot be diagnosed until adulthood, and it is given only where there is evidence of conduct disorder with onset **before age 15** years. That childhood requirement, written into the adult criteria themselves, is the structural reason the two disorders are taught together: conduct disorder before the mid-teens is a necessary building block of the adult diagnosis, so no one acquires antisocial personality disorder de novo in adult life without an antisocial childhood behind it. What makes the relationship so easily misremembered is that necessity runs in only one direction. A childhood history of conduct disorder is required for the adult diagnosis, yet the great majority of adolescents with conduct disorder do not go on to develop antisocial personality disorder at all. The corridor is real but narrow: most children who enter it turn off it, and only the high-risk minority identified by the persistence profile of the previous section travel its full length. The adult disorder in its own right - its criteria, its differential and its management - is the Personality disorders notes'; what belongs here is only the trajectory that connects it back to childhood.

The must-carry line: antisocial personality disorder requires evidence of conduct disorder with onset **before age 15** years, yet most adolescents with conduct disorder never develop it - necessary, not sufficient.

TRAP **The direction of the conduct-disorder-to-antisocial link is routinely reversed.** Candidates read the required childhood antecedent as though it ran the other way and assert that conduct disorder usually, or even always, becomes antisocial personality disorder. The correct statement is the opposite: conduct disorder before the mid-teens is a precondition of the adult diagnosis, but the majority of young people with conduct disorder do not progress to it. Stating the base rate as high is the single commonest error on this topic.

7.5 The other side of the ledger: protective and better-outcome features

The features that predict recovery are, for the most part, the mirror image of those that predict persistence. Prognosis is not read only from what is wrong; the examiner also expects the protective side. A better adult adjustment, and a lower likelihood of progression to antisocial personality disorder, are associated with the adolescent-onset course, with few and milder symptoms, with the absence of the limited prosocial emotions specifier, with the absence of comorbid ADHD and of substance abuse, and with **normative peer relationships** rather than an entrenched deviant peer group. Temperament contributes too: a higher verbal intelligence is protective, given that a lower verbal IQ is a recognised temperamental risk factor for the disorder. These are not a separate list to be learned in isolation but the same prognostic axis read from its favourable end, which is why they map cleanly onto the persistence predictors reversed. The clinical value of holding both columns in mind is that they convert prognosis from a vague impression into a structured judgement: the young person is placed by counting how many protective and how many adverse features are present, rather than by the emotional impression the presenting behaviour makes.

PROGNOSTIC AXIS	POINTS TOWARD PERSISTENCE AND ANTISOCIAL OUTCOME	POINTS TOWARD REMISSION AND GOOD ADJUSTMENT
Onset subtype	Childhood-onset	Adolescent-onset
Symptom load and severity	Many symptoms, damaging behaviours early	Few and milder symptoms
Limited prosocial emotions specifier	Present	Absent
Co-occurring ADHD	Present	Absent
Substance abuse	Present	Absent
Peer relationships	Deviant peer affiliation	Normative
Verbal intelligence	Lower verbal IQ	Higher verbal IQ

The prognostic ledger for conduct disorder read across shared axes; the protective column is largely the persistence column reversed, which is why the two are learned together rather than as separate lists.

7.6 Turning prognosis into practice

Prognosis is only useful if it changes what the clinician does. The reason the examiner cares about outcome prediction is that it is meant to be acted on. A structured reading of the prognostic profile does two things at once. It identifies the high-risk young person - early onset, callous-unemotional features, comorbid ADHD, emerging substance use - in whom the most

intensive, evidence-based intervention is justified and in whom the window for altering the trajectory is widest. And it gives the clinician language to hold a family's fear at the right level: neither the false reassurance that a seriously disordered child will simply grow out of it, nor the false verdict that a child is already lost. The prognostic features are also the natural targets of intervention, because several of them - the comorbid ADHD, the substance use, the deviant peer affiliations - are modifiable, and modifying them is one of the mechanisms by which good treatment shifts outcome.

GOOD TO REMEMBER

Record the prognostic profile explicitly in the formulation, not just the diagnosis. Whether the course is childhood or adolescent onset, whether the limited prosocial emotions specifier applies, and whether ADHD or substance use co-occurs, together place the young person on the outcome spread and set the intensity of the response. The same features that predict a poor outcome are the ones worth targeting, so the prognostic list doubles as an intervention checklist.

IN INDIA

Because Indian services classify under ICD, the operative label in case files is the conduct-dissocial disorder of that system, and the prognostic principles carry across unchanged: onset age, callous-unemotional features and comorbidity still sort the persistent minority from the many who remit. The practical constraint is continuity. The desistance seen in early adulthood is easiest to support where a young person can be followed over years, but fragmented follow-up, limited child mental health services, and loss to care after a first contact mean many are seen once at a crisis and not again. Where conduct intersects with the law, the juvenile-justice interface is taken up separately in this chapter, and the wider machinery of services and protection in the Child psychiatry: assessment, treatment, services and protection notes; the prognostic point for practice is that a single contact is rarely enough to change a trajectory that is defined by its persistence.

before 15	childhood-onset	majority
AGE OF CONDUCT DISORDER ONSET REQUIRED FOR THE ADULT ANTISOCIAL DIAGNOSIS	THE SUBTYPE CARRYING THE WORSE PROGNOSIS	OF AFFECTED YOUNG PEOPLE REMIT BY ADULTHOOD

Oppositional defiant disorder and the impulse-control disorders

8 · Oppositional defiant disorder (criteria, course, management)

The diagnosis lives in the space between a difficult temperament and a rights-violating illness. **Oppositional defiant disorder** is the syndrome of childhood and adolescence in which anger, argumentativeness, defiance and a streak of vindictiveness cohere into a pattern that is out of proportion to the child's developmental stage and that costs the child or the family something real. It is one of the commonest referrals to child mental health, and it is examined heavily for a reason that is easy to state and hard to apply: almost every young child is oppositional some of the time, so the whole art of the diagnosis is telling a disorder apart from an ordinary phase, a

temperamental extreme, or the friction of a coercive home. Marks are lost not on the symptom list, which candidates memorise, but on the thresholds, the developmental caveats and the borders with conduct disorder and the chronically irritable presentations, which is where the criteria actually do their work.

This topic owns the diagnostic architecture of the disorder: the essential feature, the symptom count and timeframe, the three symptom clusters and what each predicts, the age-graded frequency rule that separates symptom from normality, the severity grading, the local differential against conduct disorder, and the way the two major classifications part company over chronic irritability. The full conduct disorder criteria, disruptive mood dysregulation disorder, the cross-cutting normal-limits map and the attention-deficit/hyperactivity comorbidity are each developed in their own right elsewhere; here they are named where the reasoning demands it, not reopened.

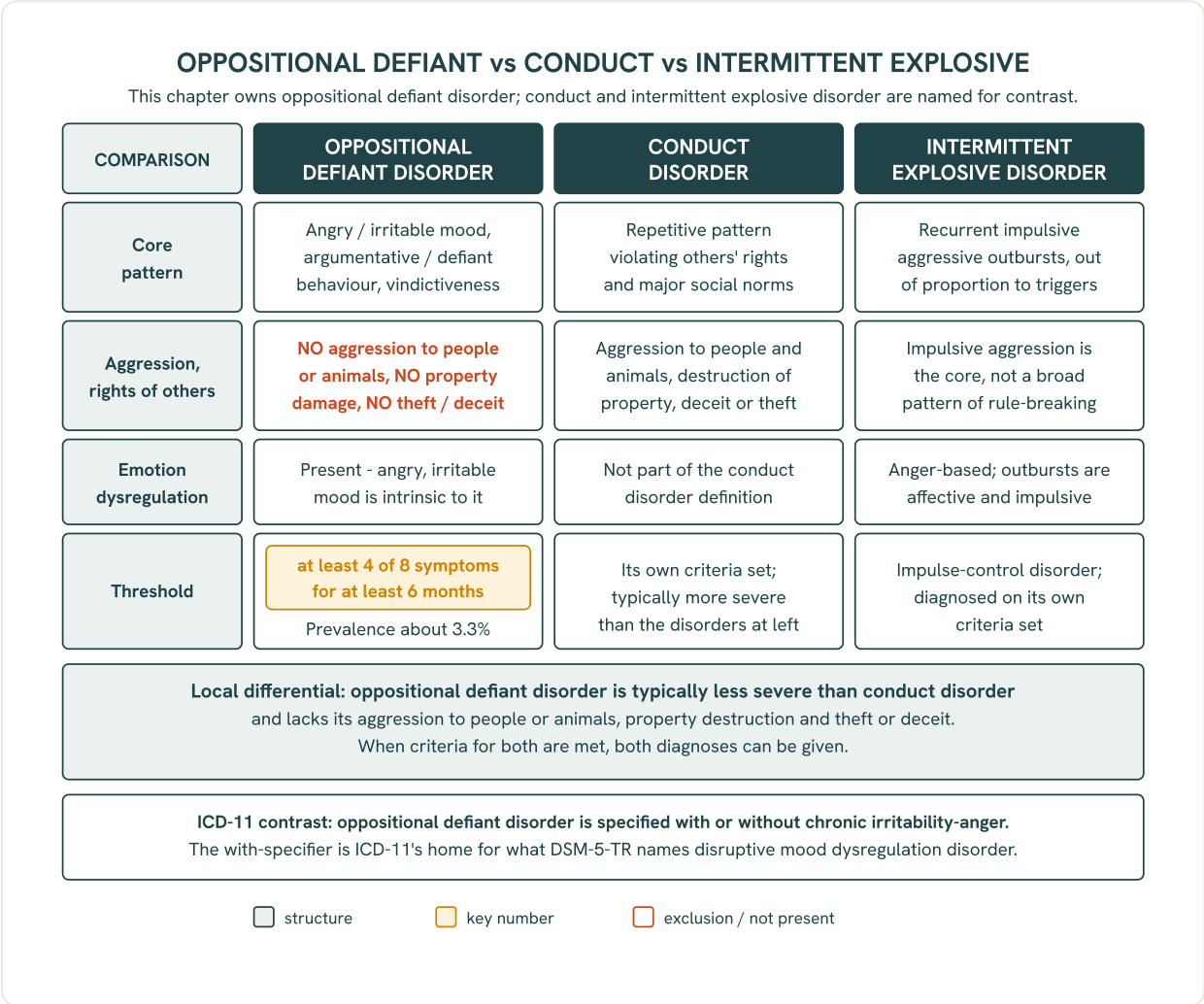

Fig 23.7. Oppositional defiant disorder against conduct disorder and intermittent explosive disorder: the same core-pattern, aggression, emotion and threshold axes that separate a persistent defiant pattern from rule-violating conduct and from impulsive aggressive outbursts.

8.1 The essential feature: a disposition, not a single scene

The unit of diagnosis is a durable pattern of three affective-behavioural dispositions. The essential feature is a recurring pattern of **angry/irritable mood**, **argumentative/defiant behaviour** and **vindictiveness**, sustained rather than sporadic and directed at the people around

the child. What makes it a disorder is not any one tantrum, argument or spiteful act, all of which are near-universal in childhood, but the consolidation of these tendencies into a stable way of relating. The framing is deliberately broader than pure behaviour: unlike the frankly antisocial syndromes, this disorder puts a mood dimension at its centre, so that the irritable, easily-provoked child who is not yet aggressive to others still belongs here. That affective core is what later separates the disorder from conduct disorder and links it to the mood and anxiety outcomes. The clinician's first task is to establish that a pattern sits behind the presenting complaint rather than be captured by the single episode that brought the family in.

8.2 Criterion A: the count, the clock and the non-sibling rule

The threshold, the timeframe and the target of the behaviour are the examinable core. The operational rule requires at least **four** symptoms drawn from a set of **eight**, present over a period of at least 6 months. Two features of the rule are quietly load-bearing. First, the four symptoms may be drawn from *any* of the three clusters, so a purely irritable child and a purely defiant child can both reach the threshold by different routes, which is why the clusters must be tracked separately rather than summed blindly. Second, the pattern must be shown during interaction with at least one individual who is not a sibling. This last clause is frequently missed and frequently tested: ordinary sibling conflict, however fierce, does not count toward the diagnosis, because rivalry and squabbling between brothers and sisters is developmentally expected and says little about a generalised oppositional disposition. The behaviour must reach beyond the sibling relationship before it becomes diagnostic.

> The core rule to carry into the exam: at least four of eight symptoms across the three clusters, for at least 6 months, shown with at least one person who is not a sibling.

8.3 The three symptom clusters and why the split predicts the future

Eight symptoms sort into three clusters that carry different prognostic freight. The criteria are not a flat list. They fall into an angry/irritable mood cluster (losing one's temper, being touchy or easily annoyed, and being angry and resentful); an argumentative/defiant behaviour cluster (arguing with authority figures, actively defying or refusing to comply with rules, deliberately annoying others, and blaming others for one's own mistakes); and a vindictiveness cluster. Grouping matters because the clusters point in different directions. The argumentative, defiant and vindictive dimension carries most of the risk for progression to conduct disorder, whereas the angry and irritable mood dimension carries most of the risk for later mood and anxiety disorders. This is the most useful prognostic reasoning the topic offers: two children who both satisfy the count can face different futures depending on which cluster dominates, so recording the profile rather than the bare tally is what lets the clinician anticipate an externalising or internalising trajectory.

CLUSTER	WHAT IT CAPTURES	WHAT IT CHIEFLY PREDICTS
Angry/irritable mood	Loses temper; touchy or easily annoyed; angry and resentful	Later mood and anxiety disorders
Argumentative/defiant behaviour	Argues with authority; defies rules; deliberately annoys; blames others	Progression toward conduct disorder
Vindictiveness	Spiteful or vindictive behaviour	

The three clusters set against what each chiefly forecasts; the affective cluster tilts toward internalising outcomes, the behavioural and vindictive clusters toward the externalising trajectory that ends in conduct disorder.

MNEMONIC

Mad, Bad and Vengeful

The three clusters in order: **Mad** (angry/irritable mood), **Bad** (argumentative/defiant behaviour) and **Vengeful** (vindictiveness). Holding them as separate axes rather than one undifferentiated pile of misbehaviour is what makes the prognostic split usable at the bedside.

8.4 The frequency rule: telling a symptom from ordinary development

Persistence and frequency, read against the child's age, are what convert a normal behaviour into a symptom. Because the raw behaviours are so common, the criteria supply an age-graded frequency floor. For a child younger than 5 years the behaviour should occur on most days across a period of at least 6 months; for a child of 5 years or older it should occur at least once per week over the same period, with an allowance for the vindictiveness item, which is expected less often. These frequencies encode a developmental truth: daily oppositionality is closer to the norm in the preschool years than later, so a higher bar is set for the younger child and a lower one for the older. The criteria also insist that the judgement be made against the individual's developmental level, sex and culture, which keeps the diagnosis from being read off behaviour in isolation from the child who produces it. Frequency, persistence and context together are what license the move from behaviour to symptom.

RULE **The threshold is set by frequency against age, not by the behaviour alone.** The same tantrum that is developmentally unremarkable on most days in a preschooler is a symptom when it recurs weekly in a school-aged child. The criteria pin the diagnosis to how often the behaviour occurs relative to what is expected at that age, which is precisely the safeguard that stops normal childhood oppositionality being pathologised.

8.5 Grading the disorder: pervasiveness across settings

Severity is read from how many settings the symptoms invade. The disorder is graded not by symptom count but by pervasiveness, on the principle that a child who is oppositional only at home is in a different clinical position from one who is oppositional everywhere. The grade is **mild** when symptoms are confined to a single setting, moderate when some symptoms appear in at least two settings, and severe when they are present in three or more. The clinical logic is sound. Symptoms limited to one relationship or place often say as much about that setting as about the child, and may respond to work targeted there; symptoms that generalise across home,

school and the wider world point to a more ingrained, more disabling disposition. Reading severity from pervasiveness also quietly instructs the assessor to gather information from more than one informant, because a grade cannot be assigned honestly from the parents' account alone.

GOOD TO REMEMBER

Because severity tracks the number of settings, a single-informant assessment can only ever establish the floor. If the parents describe defiance at home but the child has never been observed or reported on at school, the honest grade is provisional, and the next clinical move is to obtain the school's account before the disorder is called moderate or severe.

8.6 Epidemiology and the sex distribution

The frequency of the disorder is one of the reasons the threshold has to be strict. Cross-national estimates put the average prevalence at about **3.3%**, though the figure ranges widely, from roughly 1% to 11% across settings. Before adolescence the disorder is somewhat commoner in boys than girls, at a ratio of about **1.59 to 1**; the male preponderance is real but modest, and it attenuates with age, unlike the sharper and more persistent male excess seen in the frankly conduct-disordered group. The wide prevalence band is itself an examinable point: it is a direct consequence of how much the diagnosis depends on where the line between a difficult child and a disordered one is drawn, which varies between raters and cultures, and it is a standing argument for holding to the frequency and pervasiveness rules rather than to clinical impression.

8.7 The local differential: oppositional defiant disorder against conduct disorder

The two disorders share a family resemblance but part on rights-violation and on mood. The most consequential differential is against **conduct disorder**, whose full criteria are developed in its own right elsewhere in this chapter. Two lines separate them. First, the behaviours of oppositional defiant disorder are typically less severe and, crucially, do *not* include aggression toward people or animals, destruction of property, or a pattern of theft or deceit; those serious rights-violating and norm-violating acts are the province of conduct disorder. Second, oppositional defiant disorder additionally carries a component of emotion dysregulation, the angry and irritable mood, that has no place in the definition of conduct disorder. The two are not mutually exclusive, and when a young person genuinely satisfies the criteria for both, both diagnoses can be made.

- defiance, argument and spite without aggression, destruction, theft or deceit point to oppositional defiant disorder, not conduct disorder;
- aggression to people or animals, property destruction, or a pattern of theft or deceit moves the picture into conduct disorder territory;
- the angry and irritable mood dimension belongs to oppositional defiant disorder and is not part of the conduct disorder definition;
- when the criteria for both are met, both are diagnosed rather than one being forced to displace the other.

TRAP **Oppositional defiant disorder and conduct disorder are not mutually exclusive.** Candidates often treat them as a hierarchy in which the more severe diagnosis cancels the milder, and answer that a child meeting both sets of criteria has "only" conduct disorder. When both are genuinely satisfied, both diagnoses stand; the disorders are related but distinct, and the presence of one does not extinguish the other.

8.8 The classification split: chronic irritability and where it is housed

The two manuals agree on the disorder and disagree on what to do with chronic irritability. Under ICD-11 the entity is **oppositional defiant disorder**, coded 6C90, and the classification adds a decision the other manual makes differently. It allows the disorder to be specified **with chronic irritability-anger** (coded 6C90.0) or without it (coded 6C90.1). The with-specifier identifies a pattern of prevailing, persistent irritable and angry mood, and it is ICD-11's home for the very presentation that the other major classification carves out as a separate disorder, **disruptive mood dysregulation disorder**, which is developed in its own right elsewhere in this chapter. This is a genuine structural disagreement rather than a wording difference: one system treats chronic non-episodic irritability as a specifier hung on the oppositional diagnosis, the other as a distinct entity in its own right. Which manual a case file follows therefore decides whether a chronically irritable child is recorded as an oppositional defiant disorder with a specifier or as a separate mood-dysregulation diagnosis.

AXIS	ICD-11	THE OTHER MAJOR CLASSIFICATION
Base diagnosis	Oppositional defiant disorder (6C90)	Oppositional defiant disorder
Chronic irritable-angry presentation	A specifier: with chronic irritability-anger (6C90.0)	A distinct disorder, disruptive mood dysregulation disorder
Consequence for coding	The irritable child stays within the oppositional diagnosis	The irritable child receives a separate mood diagnosis

The same clinical presentation of chronic, non-episodic irritability placed differently by the two classifications; the load-bearing point is that ICD-11 keeps it inside the oppositional diagnosis as a specifier where the other manual gives it its own name.

IN INDIA

Because Indian services classify under ICD, the operative label in case files, referrals and official reports is oppositional defiant disorder under its ICD code, with the chronic irritability-anger specifier available for the persistently irritable child. A clinician trained on the other manual's categories should not go looking for a separate mood-dysregulation diagnosis in an ICD-coded record: the same child is recorded here as an oppositional defiant disorder with the specifier applied, and treating the two as different disorders rather than the same presentation filed differently is a recurring source of confusion in cross-referenced notes.

8.9 Course and management

Onset is early, the outlook is dimension-dependent, and the first-line treatment is delivered through the parents. The disorder typically declares itself in the preschool and early school

years, often before conduct problems proper, and for a subset it is the earliest link in a chain that runs toward conduct disorder and, beyond it, to antisocial outcomes in adulthood. But this trajectory is neither universal nor fixed, and which way a given child tends is what the cluster profile forecasts: the behavioural and vindictive dimension carries the risk of progression to conduct disorder, the irritable dimension the risk toward mood and anxiety disorders. Comorbidity is the rule rather than the exception, with attention-deficit/hyperactivity disorder the most frequent travelling companion; that disorder, its assessment and its treatment belong to the ADHD and specific learning/communication disorders notes, and its presence materially worsens the oppositional picture and complicates management.

Treatment is built on the parent-child relationship before it is built on medication. The first-line and best-supported intervention is structured **parent management training**, in which caregivers are coached to replace the escalating cycles of command, refusal and capitulation that maintain oppositional behaviour with consistent, contingent responses, clear expectations, and the deliberate reinforcement of prosocial behaviour. For an older child, individual work on problem-solving and anger management is added, and school-based consistency is enlisted so the same contingencies operate across settings, which also addresses the pervasiveness that defines severity. Pharmacotherapy has no primary role in the oppositional syndrome itself and is directed instead at treatable comorbidity, most often the accompanying attention-deficit/hyperactivity disorder, rather than at defiance as such. This is a relational and developmental problem first, so the family, not a prescription, is the main instrument of change; medication earns its place only where a comorbid disorder independently warrants it.

PITFALL

The commonest clinical error runs in both directions around the same axis: over-diagnosing the ordinary defiance of a tired, testing or newly-limit-tested young child as a disorder by ignoring the age-graded frequency floor, and, conversely, dismissing a genuinely disordered older child as "just naughty" because oppositionality is expected in the young. The corrective is the same in both cases: hold the behaviour against what is developmentally expected at that age, and against how often and in how many settings it actually occurs, rather than against the adults' exasperation.

4 of 8	3.3%	1.59:1
SYMPTOMS NEEDED, AT LEAST 6 MONTHS IN DURATION	AVERAGE CROSS-NATIONAL PREVALENCE	BOYS TO GIRLS BEFORE ADOLESCENCE

9 · Intermittent explosive disorder

The disorder is defined by the loss of control, not by the size of the violence. **Intermittent explosive disorder** is the diagnosis given to recurrent behavioural outbursts that represent a failure to control aggressive impulses. What is disordered is not that a person becomes angry, nor even that they become violent, but that the aggressive impulse is discharged in a sudden, poorly restrained explosion that the person themselves usually regrets. The picture is of a short fuse attached to a response grossly larger than whatever lit it: a trivial provocation, a spilled drink or a delayed reply, met with screaming, threats, a thrown object or a blow. Between the episodes the

person is often unremarkable, which is precisely why families describe someone who is "fine until they snap".

Marks accrue here for a specific reason. Aggression is a final common pathway of many disorders, and the examiner wants to know that a candidate can tell impulsive, anger-driven aggression apart from the cold, instrumental aggression of conduct disorder and antisocial personality, from the chronically irritable child, and from aggression that is merely a symptom of mania, intoxication or a frontal lesion. Intermittent explosive disorder is the residual, positive diagnosis of the impulsive aggressive outburst once those other explanations have been excluded. This topic owns that diagnosis in full: its essential feature, the two thresholds of its recurrence criterion, its qualifying and exclusion rules, its course, epidemiology and differential. The wider family of impulse-control disorders, and the conditions of firesetting and stealing, are developed in their own right and named here only where the differential demands it.

9.1 The essential feature: a failure to control aggressive impulses

The unit of diagnosis is the impulsive aggressive outburst. The core disturbance is an inability to resist aggressive impulses, expressed as recurrent outbursts that are out of proportion and out of the person's control. The aggression is **impulsive** or anger-based; it is not **premeditated**, and it is not committed to secure a tangible objective such as money, power or intimidation. That single distinction carries most of the clinical and examination weight. Aggression used as a tool, to coerce, acquire or dominate, belongs to conduct disorder or antisocial personality and not here, however dramatic it looks. The aggression of this disorder is that of a system that has lost its brakes, discharged in the heat of the moment and typically followed by genuine remorse once the arousal subsides. The outburst is the disease; the person's baseline, and their regret, are part of the picture.

RULE **Impulsive and disproportionate, never instrumental.** The diagnosis requires aggression that is anger-based and unplanned and that is grossly out of proportion to its trigger. The moment the aggression is revealed to be premeditated or committed for gain, the diagnosis is not intermittent explosive disorder, and the assessor should be looking at conduct disorder, antisocial personality or another explanation instead.

9.2 Criterion A: two ways in, frequency or severity

The threshold is a dyad, and either arm alone suffices. The recurrence criterion can be met by one of two mutually alternative patterns, and understanding that they are alternatives is the examinable core of the topic. The first arm captures frequent, low-severity outbursts: **verbal aggression or non-damaging physical aggression** occurring **twice weekly**, on average, over a period of three months. These outbursts, by definition, do not damage or destroy property and do not cause physical injury, the tantrum-like verbal explosion or the shove that harms nothing being the prototype. The second arm captures infrequent but destructive outbursts: **three** outbursts that involve damage or destruction of property, or physical assault causing injury to people or animals, occurring within a 12-month period. A person qualifies on either arm; they need not meet both. The design is deliberate, catching both the individual disrupted by constant low-grade verbal explosions and the one who erupts only rarely but does real harm.

ARM OF CRITERION A	SEVERITY OF THE OUTBURST	REQUIRED FREQUENCY AND WINDOW
High-frequency arm	Verbal or non-damaging physical aggression; no property damage, no injury	Twice weekly on average, for three months
High-severity arm	Property damage or destruction, or assault causing injury	Three outbursts within a 12-month period

The two alternative routes to the recurrence criterion; a person satisfies the requirement through either arm, and the two differ in the severity of the outburst and in the counting window rather than in the nature of the underlying impulse.

9.3 The qualifying and exclusion criteria

Recurrence alone is not the disorder; the qualifiers make it one. Several further conditions must hold before the outbursts count. The aggressiveness must be grossly out of proportion to the provocation or precipitating stressor, so that a measured, if forceful, response to a genuine threat does not qualify. The outbursts must cause the person real cost, whether marked personal distress, impairment of occupational or interpersonal functioning, or financial and legal consequences. There is a developmental floor: the person must be at least **6 years** of chronological age, or the equivalent developmental level, and the diagnosis is not made below that age, where explosive behaviour is developmentally common and non-specific. Finally, the outbursts must not be better explained by another mental disorder and must not be attributable to a substance or a general medical condition. This last exclusion is what makes the condition a diagnosis of careful exclusion rather than of first impression, since aggression is so often secondary.

- the response is grossly disproportionate to its trigger;
- the outbursts cause marked distress, functional impairment, or financial or legal harm;
- the person is developmentally at least six years old;
- the aggression is not better accounted for by another disorder, a substance, or a medical condition.

> Carry this into the exam: impulsive, disproportionate, distressing aggression, either twice weekly and non-damaging for three months or three damaging outbursts in a year, in a person aged at least 6, and not explained by anything else.

9.4 The phenomenology and course of the outburst

The anatomy of the episode is characteristic and worth knowing. The typical outburst has a rapid, almost explosive onset, often with little or no prodrome, and is usually short-lived rather than smouldering for hours. Many patients describe a mounting sense of tension or arousal before the act and a sense of relief during it, followed by fatigue and, characteristically, remorse, embarrassment or guilt once it has passed. It is this remorse, and the ego-dystonic quality of the aggression, that distinguishes the picture from the unrepentant, goal-directed aggression of the antisocial patient. The disorder tends to begin in late childhood or adolescence and to run a chronic course over many years, punctuated by discrete outbursts against a comparatively normal

inter-episode baseline. That episodic structure against a well background is the signature, and it is a large part of why the condition is under-recognised: the person seeking help usually does so between outbursts, when they appear entirely well.

> **GOOD TO REMEMBER**
>
> Ask specifically about the shape of the episodes, not just their occurrence. Rapid onset, short duration, a build-up of tension beforehand, relief during, and remorse afterwards, all against a normal baseline, is a pattern the patient will rarely volunteer but will readily confirm. Documenting that episodic architecture is often what separates this diagnosis from a persistently irritable temperament or a mood disorder.

9.5 Epidemiology

It is commoner than its low profile suggests. Population data put the one-year prevalence in the United States at about **2.6%**, with a lifetime prevalence of around **4.0%**, figures that make it far from a rarity even if it is seldom the presenting complaint. It is more prevalent among younger people, being concentrated in those under about 35 to 40 years, in keeping with its onset in youth and its attenuation with age, and it is somewhat commoner in males. The gap between how often the disorder occurs and how rarely it is formally diagnosed is itself an examination point: patients are labelled as having an anger problem, or seen only through the marital, occupational or forensic consequences of their outbursts, rather than recognised as having a discrete, treatable impulse-control disorder.

9.6 The differential diagnosis: a crowded field

Almost every disorder that produces aggression must be set aside first. Because aggression is nonspecific, the differential is the most demanding part of the topic. The chronically irritable child whose mood is persistently angry between outbursts is better captured by **disruptive mood dysregulation disorder**, developed in its own right elsewhere in this chapter; the diagnostic convention is that where that disorder's criteria are met in a child, the explosive-disorder label is not added on top. Conduct disorder and oppositional defiant disorder, also elsewhere in this chapter, involve a broader pattern of rule-breaking or defiance in which aggression is one element, and the explosive-disorder diagnosis is reserved for impulsive aggression that is in excess of, and not fully explained by, that broader pattern. Antisocial personality is separated by the instrumental, unremorseful quality of its aggression and belongs to the Personality disorders notes. Aggression arising only during manic, depressive, psychotic or intoxicated states is a symptom of those states, and the impulsivity of untreated attention-deficit/hyperactivity disorder, taken up in the ADHD and specific learning/communication disorders notes, must be weighed as well.

CONDITION TO EXCLUDE	WHAT SEPARATES IT FROM INTERMITTENT EXPLOSIVE DISORDER
Disruptive mood dysregulation disorder	Persistent angry or irritable mood *between* the outbursts; where its criteria are met in a child, this disorder takes precedence and the explosive-disorder label is not added
Conduct disorder and oppositional defiant disorder	A broad pattern of rule-violation or defiance; the explosive diagnosis needs impulsive aggression in excess of what that pattern explains
Antisocial personality disorder	Aggression that is premeditated and instrumental, pursued for gain and without remorse
Manic, depressive or psychotic states	Aggression occurs only during the mood or psychotic episode and remits with it
Substance intoxication or withdrawal, and medical or neurological conditions	Aggression is attributable to the substance or the lesion; excluded by Criterion F

The principal competitors for the aggressive presentation; intermittent explosive disorder is what remains once each of these has been considered and set aside, which is why it is best thought of as a positive diagnosis reached through disciplined exclusion.

TRAP **Calling instrumental aggression "explosive".** The classic error is to reach for this diagnosis for any patient with a history of violence. Candidates forget that aggression committed to obtain something, or that is planned, is specifically excluded: it points to conduct disorder or antisocial personality. The disorder is impulsive, disproportionate and regretted, never a calculated means to an end.

9.7 Principles of management

Treatment targets the impulse, not merely the incident. Management combines psychological and pharmacological strategies, with the emphasis on reducing the frequency and intensity of the outbursts rather than managing each crisis after the fact. Cognitive behavioural approaches, including anger management, relaxation and coping-skills training, cognitive restructuring of the appraisals that trigger the arousal, and relapse prevention, are the psychological mainstay and have the best-established evidence. Pharmacologically, serotonergic antidepressants are the most commonly used agents, on the rationale that impulsive aggression is linked to serotonergic dysregulation, with mood stabilisers and certain anticonvulsants where affective instability is prominent; specific agents and dosing lie outside what this topic establishes and should be confirmed against a pharmacology source. Coexisting conditions, particularly depression, substance use and attention-deficit/hyperactivity disorder, must be identified and treated in their own right, since untreated comorbidity both worsens the outbursts and undermines any intervention aimed at them.

PITFALL

The commonest clinical failure is to treat only the aftermath. Clinicians manage the injury, the property damage or the legal fallout of each outburst and never step back to recognise and treat the underlying impulse-control disorder, so the patient cycles through emergency departments, marital breakdowns and forensic contacts without anyone naming the condition that drives them. Screening a patient with recurrent, regretted, disproportionate aggression for the disorder itself is what breaks that cycle.

IN INDIA

Most Indian services classify under ICD rather than DSM, where the condition sits among the impulse-control disorders, and its wider framework is developed in the Somatic-symptom, sexual, impulse-control disorders and suicide notes. The practical reality is that these patients present through their consequences, in casualty after an assault, in the marital clinic, or via a medicolegal referral after a charge, far more often than they ask for help with anger. Holding the diagnosis in mind when assessing recurrent, out-of-proportion, regretted aggression can convert a repeat forensic or emergency presentation into a treatable psychiatric problem, which is the single most useful move the topic offers here.

age 6	2.6%	4.0%
DEVELOPMENTAL FLOOR FOR THE DIAGNOSIS	ONE-YEAR PREVALENCE (UNITED STATES)	LIFETIME PREVALENCE

10 · Juvenile delinquency (psychiatric perspective)

The label is a verdict, not a diagnosis. A young person becomes a delinquent the moment a court adjudicates them for an offence, and nothing about that legal event tells you whether a psychiatric disorder is present. This is the first and most important idea in the topic, because the word arrives in the examination looking like a clinical entity and is nothing of the sort. Juvenile delinquency is a **legal and administrative category**, a description of what a young person has done and how the state has responded to it, not a description of a mind. The psychiatrist is called into this world not to confirm the label but to answer a separate question underneath it: what, if anything, is wrong, and is it treatable.

Marks are here for a reasoning move rather than a fact list. The examiner wants to see that a candidate can hold the legal act and the mental-health need apart, and can then say what the child and adolescent psychiatrist actually contributes to a young offender: an understanding of the antisocial behaviour, and a disciplined search for the psychiatric disorders that so often sit behind it. This topic owns that psychiatric perspective in full and names the juvenile-justice framework around it. It does not own the disorders themselves, which are taught in their own right, and it deliberately does not teach the legal machinery in detail, which belongs to the statute and to the child-services notes named at the end.

10.1 A legal category, not a psychiatric diagnosis

Delinquency is defined by adjudication, not by symptoms. The defining feature of the term is administrative: a young person has committed an act that would be an offence, and a justice system has processed them for it. There is no symptom list, no duration criterion, no exclusion clause, because it is not a diagnosis at all. This matters clinically because the same legal label covers utterly different children. One delinquent has no psychiatric disorder and has offended out of circumstance, poverty or a single bad decision; another carries **conduct disorder**, or an untreated attentional disorder, or a substance problem, or the aftermath of trauma, and offends as one expression of it. The legal category flattens all of them into a single administrative fact, and the psychiatrist's task is to reopen it. Treating the label as though it were the diagnosis is the error the whole topic is built to prevent: it leads clinicians and courts alike to assume a disorder where there is none, or, worse, to miss a treatable one because the young person has already been filed under a legal heading that seems to explain everything.

RULE **Delinquency is what the court decides; the diagnosis is what the clinician finds.** These are two independent questions asked by two different systems, and the psychiatric perspective exists to answer the second without letting the first stand in for it. A young offender may have no psychiatric disorder, or may have one that has gone unrecognised precisely because the legal label arrived first.

10.2 The psychiatric perspective: finding the disorder inside the label

The clinician's contribution is comprehension and comorbidity. Child and adolescent psychiatry is asked to do two things with a delinquent young person: to understand the antisocial behaviour in developmental terms, and to assess the psychiatric disorders that are frequently present and that contribute to the offending. This second duty is where the marks concentrate, because rates of unrecognised psychiatric illness in young people who reach the justice system are high, and much of it is treatable. The disorders that recur are a familiar cluster. Conduct disorder is the commonest and most directly relevant, developed on its own terms elsewhere in this chapter; attention-deficit/hyperactivity disorder, taken up in the ADHD and specific learning/communication disorders notes, drives the impulsivity and poor self-regulation that turn a temptation into an offence; substance use both disinhibits behaviour and generates acquisitive crime; specific learning disorders and low scholastic attainment feed the school failure and truancy that precede much delinquency; and a history of maltreatment or trauma, with its post-traumatic sequelae, is disproportionately common. None of these is the legal category, and each changes what should happen next.

CONTRIBUTING DISORDER TO SCREEN FOR	WHY IT MATTERS IN A YOUNG OFFENDER
Conduct disorder	The most direct psychiatric correlate of persistent offending; taught in its own right in this chapter, not re-established here
Attention-deficit/hyperactivity disorder	Impulsivity and poor self-regulation convert opportunity into offence; treatable, and covered in the ADHD and specific learning/communication disorders notes
Substance use	Disinhibits behaviour and generates acquisitive and violent crime; often the reversible driver
Specific learning disorders and scholastic failure	School failure and truancy commonly precede delinquency and are frequently unrecognised
Trauma and its post-traumatic sequelae	Maltreatment histories are over-represented; the offending may be one expression of the aftermath

The psychiatric disorders that recur behind the legal label; the clinical value of the psychiatric perspective lies in detecting and treating these, none of which is the delinquency itself, and several of which are directly modifiable.

GOOD TO REMEMBER

Treat entry into the justice system as a screening opportunity that would otherwise be missed. Many of these young people have never had a psychiatric assessment, and the disorders most worth finding, an attentional disorder, a substance problem, a treatable mood or post-traumatic picture, are exactly the ones that a purely legal response will overlook. The single most useful thing the psychiatric perspective offers is a low threshold for looking, because the disorder that is found is often the one that changes the trajectory.

10.3 The juvenile-justice framework and its rehabilitative orientation

The system that handles young offenders was built to rehabilitate, not to punish. The **juvenile-justice framework** is the specialised court and justice system that deals with young offenders separately from adults, and its founding logic is what a candidate must be able to state. The first juvenile court was created in **1899**, deliberately set apart from the adult criminal process so that the response to a delinquent child could be corrective rather than retributive. That rehabilitative orientation has persisted, and modern practice within the framework increasingly emphasises evidence-based psychosocial treatment over incarceration, and in particular over placements that gather delinquent young people together. The interventions that carry the best evidence are worth knowing by name:

- **multisystemic therapy**, an intensive family- and community-based treatment;
- therapeutic foster care, a structured alternative to group placement;
- diversion away from formal processing where appropriate;
- juvenile mental-health and drug courts, which route treatment needs into the justice response.

The common thread is that each keeps the young person embedded in a corrective relationship, family, foster placement or treatment programme, rather than in a peer group of other offenders.

There is also growing recognition within the framework of how much untreated psychiatric illness this population carries, which is precisely the door through which the psychiatric perspective enters. The full machinery of the framework, its statutory structure and the child-protection services that sit alongside it, is taught in the Child psychiatry: assessment, treatment, services and protection notes, and this topic names it rather than reproducing it.

PITFALL

The intuitive response, gathering delinquent adolescents together in a group placement or programme, can make antisocial behaviour worse rather than better. Placing young offenders among deviant peers tends to reinforce the very behaviour it is meant to correct, which is why the evidence-based interventions deliberately keep the young person in a family or individually structured setting. A clinician who recommends "a place with others like him" may be recommending the one thing the framework has learnt to avoid.

10.4 The Indian statutory frame, named but not quoted

The legal specifics belong to the statute, not to a textbook paraphrase. Everything above describes the general, largely Western juvenile-court model and the psychiatric role within it. The Indian legal machinery is governed by its own statute, the **Juvenile Justice (Care and Protection of Children) Act**, and this is where a candidate must be disciplined. The exact definitions, the age thresholds that determine who is treated as a child, the provisions dealing with serious offences, and the bodies constituted to decide these cases are statutory specifics that must be read from the primary Act itself, not carried over from a Western textbook or a second-hand summary. Quoting an age or a provision from memory or from a paraphrase is precisely how an error enters a candidate's answer, because the numbers differ across jurisdictions and across revisions of the law. The safe and correct move is to name the Act, to state that it governs the Indian juvenile-justice response, and to say that its provisions are read from the statute when a specific is genuinely needed.

IN INDIA

The Indian juvenile-justice response operates under the Juvenile Justice (Care and Protection of Children) Act, which establishes the dedicated bodies that adjudicate and provide for young persons in conflict with the law and children in need of care and protection. The statutory detail, the operative age definitions and the provisions for serious offences, should be read from the current Act rather than reproduced here, because these are the specifics most likely to be misquoted. For the full child-protection and juvenile-justice machinery, and for the child psychiatric services that interface with it, see the Child psychiatry: assessment, treatment, services and protection notes.

A delinquent is a young person a court has adjudicated, not a young person a clinician has diagnosed; the marks are for finding, and treating, the psychiatric disorder that so often hides inside the legal label.

TRAP **Equating delinquency with conduct disorder.** Candidates routinely treat the two as synonyms and lose the point of the topic. Delinquency is a legal status conferred by a court; conduct disorder is a clinical diagnosis conferred by a clinician. Most delinquents are not simply "conduct disorder by another name": some have no psychiatric disorder at all, and others have an attentional, substance-related, learning or post-traumatic disorder that the label conceals. Answering as though every young offender has conduct disorder both over-diagnoses and misses the treatable illness the perspective exists to find.

1899	Legal	Comorbid
FIRST JUVENILE COURT ESTABLISHED, ON A REHABILITATIVE MODEL	DELINQUENCY IS AN ADJUDICATED CATEGORY, NOT A DIAGNOSIS	CONDUCT DISORDER, ADHD, SUBSTANCE USE, TRAUMA COMMONLY PRESENT

11 · Pyromania and kleptomania (impulse-control disorders)

These are diagnoses of a captured will, not of the behaviour it produces. **Pyromania** and **kleptomania** are the classical impulse-control disorders of deliberate fire setting and repeated stealing. What makes each a psychiatric diagnosis is not the act, which is also the stuff of ordinary crime, but the internal machinery driving it: a rising, uncomfortable tension that the act discharges, followed by pleasure or relief, in the absence of any everyday motive that would explain the same behaviour in anyone else. The person does not set fires to collect insurance or steal what they cannot afford; they set fires because the setting relieves something, and they steal objects they neither need nor want.

Marks accrue here because the examiner is testing a discrimination the untrained eye gets wrong almost every time: the difference between a crime and a disorder that happens to be enacted through a criminal behaviour. A candidate who can recite that the fire setting must be purposeless, tension-driven and unexplained by gain or anger has understood the topic; one who labels every arsonist a pyromaniac and every shoplifter a kleptomaniac has missed the point. This section owns both disorders in full: their essential features, exclusion rules, rarity and differential from their criminal mimics. The wider family of impulse-control disorders is developed in the Somatic-symptom, sexual, impulse-control disorders and suicide notes and is named here only as context.

11.1 The shared architecture: tension, act, relief

Both disorders run on the same three-beat cycle, and recognising it is half the topic. Before the act there is a mounting sense of **tension or affective arousal**; the act discharges it; and in its aftermath there is pleasure, gratification or relief. This is the signature of an **impulse-control disorder**: the behaviour is not a means to an external end but a way of managing an internal state, which is why it feels compelling to the person and senseless to the observer. The impulse is difficult to resist, often ego-dystonic, and the relief is short-lived, so the cycle repeats. Holding this architecture in mind is what lets a clinician ask the right questions, because the tension-then-relief pattern is rarely volunteered but readily confirmed once specifically sought.

MNEMONIC

Tension - Act - Relief

The common spine of both disorders: an uncomfortable build-up of arousal before the behaviour, its discharge in the act of fire setting or stealing, and a transient sense of pleasure, gratification or relief afterwards. If that cycle is absent, and the behaviour is fully explained by an external motive, neither diagnosis applies.

RULE **The diagnosis is the impulse, never the act.** Fire setting and stealing are behaviours, not disorders. Pyromania and kleptomania are made only when the behaviour is driven by an irresistible, tension-relieving impulse and is not accounted for by profit, ideology, concealment, anger, need or another psychiatric condition. The moment an ordinary motive is found, the impulse-control diagnosis falls away.

11.2 Pyromania: the core criteria

Four positive features must be present together. The behavioural core is **deliberate and purposeful fire setting** on more than one occasion, so a single episode never suffices. Around it sit three affective features that make it a disorder: the tension or affective arousal that precedes the act; a genuine **fascination with, interest in, curiosity about or attraction to fire** and its contexts, uses and paraphernalia; and the pleasure, gratification or relief experienced when setting fires or witnessing their aftermath. The person is often a familiar face at fires, drawn to alarms, equipment and the fire service. The internal state carries the weight: the fire is set not to destroy a target but because the setting and its spectacle satisfy something in the person.

11.3 Pyromania: the exclusions and the arson differential

The exclusions do most of the diagnostic work, because instrumental fire setting is common and pyromania is not. The fire setting must not be done for a rational or comprehensible external reason: not for monetary gain, not in the service of a sociopolitical ideology, not to conceal a crime, not to express anger or vengeance, not to improve one's living circumstances, not in response to a delusion or a hallucination, and not as a product of impaired judgement such as in a major neurocognitive disorder or intellectual disability. Beyond these motive-based exclusions, the fire setting must not be better explained by a broader disorder in which it is one symptom among many.

- excluded if set for monetary gain, ideology, concealment of crime, anger or vengeance, or to improve living circumstances;
- excluded if set in response to a delusion or hallucination, or as a result of impaired judgement;
- excluded if better explained by **conduct disorder, a manic episode, or antisocial personality disorder**.

These exclusions are precisely what separate the diagnosis from ordinary or **instrumental arson**, and from fire setting that is a symptom of conduct disorder, developed in its own right elsewhere in this chapter. The great majority who set fires do so for a reason, which is itself the disqualifier. Antisocial personality disorder, where fire setting can be one facet of a broader exploitative

pattern, belongs to the Personality disorders notes; fire setting only during a manic episode is a symptom of that episode.

TRAP **Equating fire setting with pyromania.** The classic examination error, and the classic clinical one, is to call any repeated fire setter a pyromaniac. Candidates forget that the diagnosis is one of exclusion: it requires that every ordinary motive be absent and that the behaviour not be part of conduct disorder, mania or antisocial personality. Most fire setting is instrumental or symptomatic, and true pyromania is a small residue left once those are stripped away.

11.4 How rare pyromania really is

The number examiners want is a warning against over-diagnosis. Pyromania as a primary diagnosis appears to be very rare, and its true population prevalence is simply not known. The most quoted figure comes from a forensic study of repeated fire setters, among whom only **3.3%** met full criteria for the disorder; even in the population most enriched for fire setting, the overwhelming majority did not qualify. It is essential to separate this from fire-setting behaviour in general, whose lifetime prevalence is of the order of **1.0% to 1.1%** and which is only one component of the disorder and never sufficient for it. In children and adolescents, fire setting is usually associated with conduct disorder, attention-deficit/hyperactivity disorder or an adjustment disorder rather than pyromania, so the paediatric diagnosis should be made with great caution.

11.5 Kleptomania: the core criteria

The defining feature is theft of the useless. Kleptomania is a recurrent failure to resist impulses to steal objects that are **not needed for personal use or for their monetary value**. As with pyromania, an increasing sense of tension precedes the theft, and pleasure, gratification or relief accompanies the act of committing it. The tell is in what is taken and what becomes of it: the objects are typically of little value to the person, who could usually have afforded them, and who then gives them away, discards, hoards or even returns them. The stealing is not driven by want but by the impulse, and the goods are almost incidental. Between episodes the person is frequently distressed and ashamed, the ego-dystonic quality that marks the impulse-control disorders and separates them from a settled criminal disposition.

11.6 Kleptomania: exclusions and the ordinary-theft differential

The single most important discrimination is from ordinary theft. The stealing must not be committed to express anger or vengeance and must not occur in response to a delusion or a hallucination, and it must not be better explained by conduct disorder, a manic episode or antisocial personality disorder. The pivotal contrast is with **ordinary theft**, which is deliberate and motivated by the usefulness or monetary worth of the object. In kleptomania the object is not wanted; in ordinary theft it is the whole point. Shoplifting driven by need, resale value or peer pressure is not kleptomania; nor is the calculated stealing of the antisocial patient, which belongs to the Personality disorders notes, nor the disinhibited acquisitiveness of a manic episode, which remits with the mood state.

- not committed to express anger or vengeance, and not a response to a delusion or hallucination;
- not better explained by conduct disorder, a manic episode, or antisocial personality disorder;
- distinguished from ordinary theft, where the object is taken for its use or its worth.

PITFALL

The commonest clinical failure is to stop at the label of thief and never look for what travels with kleptomania. These patients carry a heavy burden of comorbid mood, anxiety, eating and substance-use disorders, and present through shame and legal jeopardy rather than a request for help. Treating the person as merely delinquent, without screening for the treatable picture behind the stealing, misses both the diagnosis and its companions.

11.7 Kleptomania: epidemiology and the sex ratio

Rare in the population, over-represented among the caught. Kleptomania is very rare in the general population, with a prevalence of about **0.3% to 0.6%**. It shows a female preponderance, with women outnumbering men by a ratio of about **3 to 1**, one of the few impulse-control disorders that is commoner in women. Its visibility is greatest at the point of apprehension: it is found in roughly 4% to 24% of people arrested for shoplifting, a wide band that reflects how variably it is sought. That combination, genuine rarity in the community but a real minority presence among shoplifters, is the examinable shape of its epidemiology, mirroring pyromania, where the disorder is a small fraction even of fire setters.

AXIS	PYROMANIA	KLEPTOMANIA
The behaviour	Deliberate, purposeful fire setting on more than one occasion	Recurrent stealing of objects not needed for use or value
The internal cycle	Tension before, fascination with fire, relief after	Tension before, pleasure or relief at the theft
Its criminal mimic	Instrumental or ideological arson	Ordinary, need-driven or profit-driven theft
Sex distribution	Not established by an owned figure here	Female preponderance, about 3 to 1
Frequency	Very rare even among fire setters (3.3%)	Very rare in the population (0.3% to 0.6%)

The two disorders side by side; they share an identical internal architecture and differ chiefly in the behaviour through which the impulse is discharged and the criminal act each must be told apart from.

11.8 Assessment and management principles

The task is to find the impulse behind the offence and to treat what surrounds it. Because both present far more often through their consequences than a request for help, assessment begins with a deliberate search for the tension-relief cycle and a careful exclusion of every ordinary motive, followed by a systematic screen for the accompanying comorbidities. Management is not established here by an owned protocol, and specific agents and doses should be confirmed against a pharmacology source; in broad terms it combines psychological work

aimed at the impulse, such as cognitive and behavioural strategies that interrupt the tension-to-act sequence, with treatment of coexisting mood, anxiety and substance-use disorders, which frequently improves the impulse-control behaviour as a byproduct. The evidence base for both is thin, a consequence of their rarity, and treatment is individualised rather than protocolised.

GOOD TO REMEMBER

When a patient reaches you through a charge for fire setting or shoplifting, ask three things first: what they felt in the minutes before the act, what they felt during it, and what they did with what they took or what target they chose. A build-up of tension, relief in the act, and a fire set at nothing in particular or an object neither needed nor kept is what turns a medicolegal referral into a treatable diagnosis. The motive question is the whole assessment in miniature.

IN INDIA

Most Indian services diagnose under ICD, where both conditions sit among the impulse-control disorders and their broader framework is developed in the Somatic-symptom, sexual, impulse-control disorders and suicide notes. In practice these patients almost never present asking for treatment; they arrive through the criminal-justice route, after a fire or a shoplifting charge, often for a medicolegal opinion on responsibility. Holding the diagnosis in mind in that forensic setting, and enquiring carefully into motive and the tension-relief cycle, is what distinguishes a treatable disorder from ordinary crime.

Carry this into the exam: pyromania and kleptomania are diagnoses of a tension-relieving impulse, made only when fire setting or stealing is purposeless and every ordinary motive - gain, anger, ideology, need - and every competing disorder is excluded.

3.3% OF REPEATED FIRE SETTERS MET FULL PYROMANIA CRITERIA	**0.3-0.6%** KLEPTOMANIA PREVALENCE, GENERAL POPULATION	**3 to 1** FEMALE-TO-MALE RATIO IN KLEPTOMANIA

Childhood anxiety, OCD and trauma disorders

12 · Separation anxiety disorder (criteria, course, management)

Almost every toddler protests when a parent leaves the room, so the whole clinical art of this diagnosis is knowing where an ordinary developmental protest ends and a disorder begins. **Separation anxiety disorder** is the syndrome in which fear or anxiety about being parted from those to whom the child is attached becomes developmentally inappropriate, excessive and persistent, and starts to cost the child a normal life. It is the most frequent anxiety disorder of early childhood, and it earns its marks not from the symptom list, which candidates memorise easily, but from the two discriminations that sit on either side of it: telling the disorder apart from the heightened separation anxiety that is a normal, even reassuring, feature of early

development, and recognising that the same disorder no longer stops at the nursery door but runs on into adolescence and adult life.

This topic owns the diagnostic architecture of the disorder: the essential feature and its symptom threshold, the eight anxieties the criteria enumerate, the duration and impairment rules, the way the disorder is graded against the child's developmental stage, its epidemiology across the lifespan, its heritable and temperamental roots, the split between the two major classifications over how long symptoms must last, and the screening instrument most used to detect it. School refusal, the childhood anxiety family, selective mutism and the somatisation of distress each belong to their own accounts elsewhere in this chapter and are named here only where the reasoning demands, never reopened.

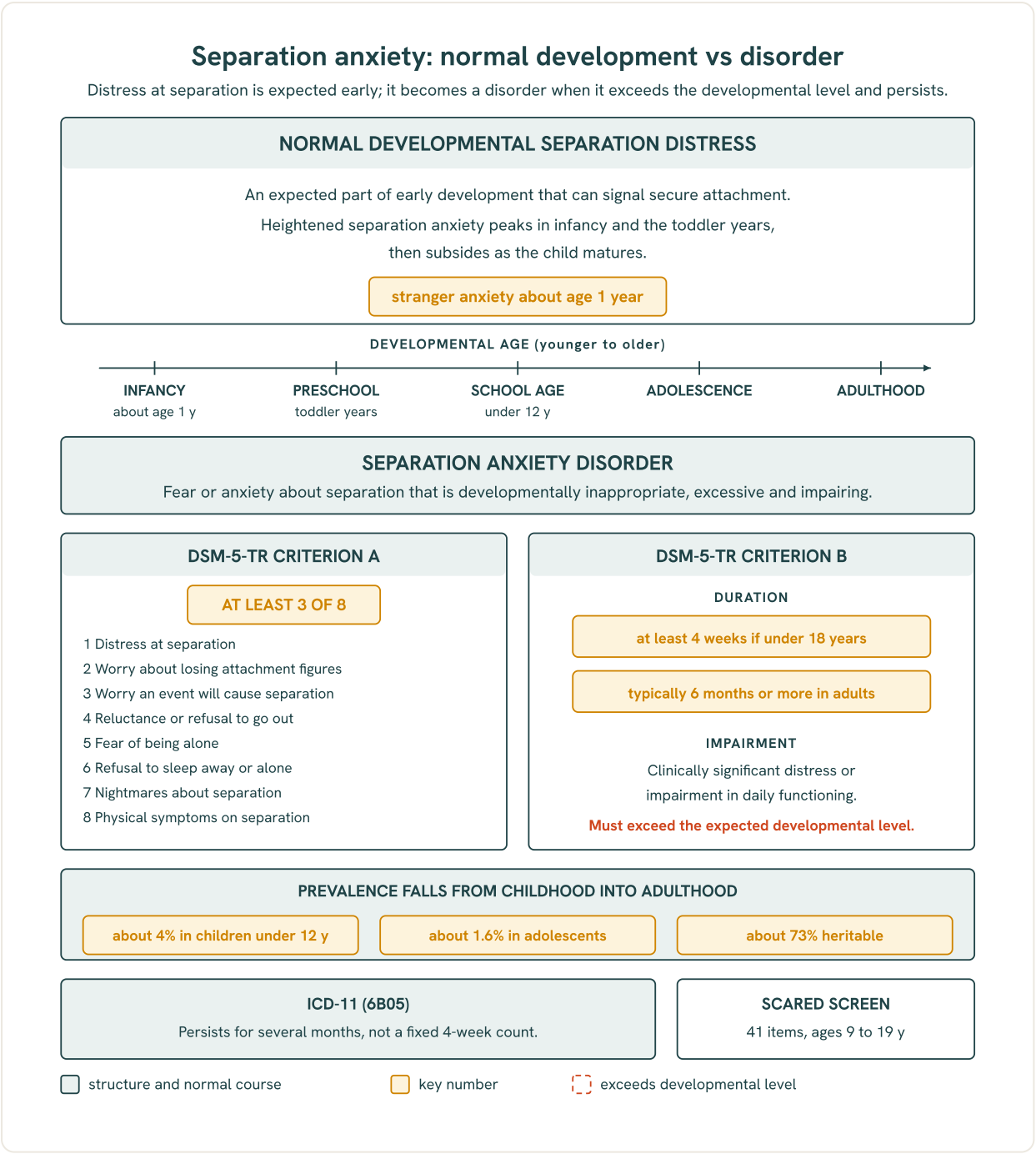

Fig 23.8. Separation anxiety across development: normal early distress that signals secure attachment versus separation anxiety disorder, defined by the DSM-5-TR count and duration thresholds, with prevalence falling from childhood into adulthood.

12.1 Normal separation anxiety and where the disorder begins

Separation fear is a normal developmental achievement before it is ever a disorder. A period of heightened distress on separation is an expected part of early development, appearing as **stranger anxiety** around the age of one year and reflecting the very cognitive and attachment gains that let an infant hold a caregiver in mind and register their absence. Far from being pathological, its presence can signal a securely formed attachment. The disorder is diagnosed only when the anxiety exceeds what is developmentally expected for the child's age and, in addition, meets the duration and impairment thresholds the criteria set. That two-part test - out of proportion to development, and functionally costly - is the safeguard that keeps a normal

phase from being pathologised, and it is the SAD-specific corner of the broader question of when childhood behaviour is within normal limits, which is drawn together as a cross-cutting map in its own right elsewhere in this chapter.

PITFALL

The commonest clinical error runs in both directions around the same axis. A clinging, tearful preschooler at the school gate is over-called a disorder when the distress is age-appropriate and transient; conversely, a genuinely impaired older child is dismissed as merely "attached" or "shy" because separation fear is assumed to belong only to the very young. The corrective is to read the anxiety against the child's developmental level and against how much it actually derails schooling, sleep and family life, rather than against the parents' worry alone.

12.2 Criterion A: the count and the eight anxieties

The diagnosis requires a developmentally excessive fear of separation evidenced by a defined minimum of symptoms. The operational rule is that the fear or anxiety concerning separation from attachment figures be shown by **at least three** of eight enumerated symptoms. Those eight are not a random inventory: they cluster around a small number of themes. Three are forms of anticipatory **worry** - distress at actual or anticipated separation, persistent worry about losing attachment figures or that harm will befall them, and worry that some untoward event will bring about separation. Two are forms of refusal and avoidance - reluctance or refusal to leave home, go to school or go out because of the separation it entails, and reluctance or refusal to be alone. Two concern sleep - reluctance or refusal to sleep away from home or without being near an attachment figure, and repeated nightmares whose theme is separation. The eighth is bodily: repeated **physical complaints** such as headaches, stomachaches, nausea or vomiting when separation occurs or is anticipated. The last of these is easy to miss because it presents to the paediatrician, not the psychiatrist; the recurrent abdominal pain and headache through which anxiety and low mood so often speak in children is developed as a presentation in its own right elsewhere in this chapter, but here the point is narrower - somatic complaints tied specifically to separation are one of the eight diagnostic symptoms and must be counted as such.

THEME	DIAGNOSTIC SYMPTOMS TIED TO SEPARATION
Anticipatory worry	Distress at separation; worry about losing or harm coming to attachment figures; worry about an event causing separation
Refusal and avoidance	Reluctance or refusal to go out or to school; fear of being alone
Sleep	Reluctance or refusal to sleep away or without an attachment figure near; nightmares of separation
Bodily	Physical symptoms - headaches, stomachaches, nausea, vomiting - on separation

The eight Criterion A symptoms grouped by theme; the child needs at least three of the eight, and the bodily symptoms are the ones most likely to be routed to a physician before a psychiatrist.

MNEMONIC

Worry, Won't-go, Won't-sleep, and the body

The eight anxieties fall into four holdable groups: three **worries** about separation and harm, two forms of **won't-go** refusal to leave or be alone, two forms of **won't-sleep** away from an attachment figure, and one **bodily** cluster of physical complaints. Carrying the themes rather than a flat list of eight is what makes the count usable at the bedside.

12.3 Criteria B to D: duration, distress and the differential

Duration is where the criteria are age-graded, and it is the most examinable single rule. The fear, anxiety or avoidance must be persistent, lasting **at least four weeks** in children and adolescents younger than eighteen years, and typically six months or more in adults. The four-week floor is deliberately short because childhood distress on separation can be so intense that a longer wait would leave an impaired child untreated; the longer adult convention reflects that a disorder first surfacing or persisting in adulthood must be distinguished from transient reactions to real life stress. Beyond duration, the disturbance must cause clinically significant distress or impairment in social, academic or other important areas, and it must not be better explained by another disorder. Several conditions mimic it and must be excluded:

- the refusal to leave home in autism, driven by a need for sameness rather than separation fear;
- the diffuse worry of generalised anxiety, which is not anchored to separation;
- a fear driven by delusion in a psychotic illness;
- the reluctance to go out in agoraphobia, where the fear is of the situation, not the parting.

The diagnosis, in other words, is not made from the separation fear alone but from separation fear that persists, disables and is not a better fit somewhere else.

The core rule to carry into the exam: at least three of eight symptoms of developmentally excessive separation fear, persisting at least four weeks in the under-eighteens, with clinically significant impairment.

RULE **The threshold is developmental inappropriateness plus duration plus impairment, never the fear alone.** Separation anxiety is normal; separation anxiety that exceeds the child's developmental level, lasts beyond the criterion period and demonstrably impairs schooling, sleep or family function is the disorder. Each of the three conditions is load-bearing, and dropping any one of them is how normal childhood distress is over-diagnosed.

12.4 A lifespan disorder: epidemiology and course

The single most important shift in how this disorder is now understood is that it does not end with childhood. It is the most common anxiety disorder in children younger than twelve years, with a six-to-twelve-month prevalence of **approximately four per cent**. Prevalence then falls steadily from childhood through adolescence and into adult life; by adolescence the twelve-month figure is down to **about 1.6 per cent**. The older teaching that the condition is purely a

disorder of childhood has been abandoned: it can persist across development, can wax and wane with periods of stress and transition, and can have its first onset in adulthood, which is why the criteria carry a separate adult duration convention at all. The typical childhood course is one of exacerbation around developmental hurdles - starting school, a family move, a parental illness or a bereavement - and the disorder is a recognised antecedent of later anxiety and depressive disorders, so it is worth treating rather than waiting out.

12.5 Etiology: temperament, attachment and a strongly heritable core

The disorder sits at the meeting point of a fearful temperament, the quality of attachment and a substantial genetic loading. Behavioural inhibition - the temperamental tendency to withdraw from, and to react with distress to, the unfamiliar - is the constitutional soil, and an anxious or overprotective parenting style, parental anxiety disorder, and stressful life events involving loss or threat of separation are the environmental accelerants. Onto that clinical picture the genetics map cleanly: in a community sample of six-year-old twins, heritability was **estimated at 73 per cent**, with higher rates in girls. That is a high figure for a childhood emotional disorder: it signals that the condition is not simply a learned response to an anxious home but carries a strong constitutional contribution, and it fits the observed familial aggregation, in which children of parents with anxiety and panic disorders are over-represented. Etiology here is genuinely biopsychosocial: temperament sets the threshold, attachment and parenting shape the expression, and life events involving separation pull the trigger.

12.6 The classification split: a fixed count against a clinical judgement of duration

The two major manuals agree on the disorder and disagree on how much numerical precision to demand. One system fixes an explicit symptom count and an explicit minimum duration, the four-week floor for the under-eighteens being the clearest example. The other, the ICD-11 clinical descriptions and diagnostic requirements, deliberately avoids that kind of artificial precision, asking instead that symptoms persist for at least several months rather than for a fixed four-week count, and leaving the clinician to judge severity and persistence against the whole picture. In ICD-11 the disorder is coded **6B05**, and, unlike its predecessor, it is no longer confined to childhood. Where the individual is under six years of age, the ICD-11 mortality and morbidity statistics map the condition to the older category of separation anxiety disorder of childhood, F93.0. The disagreement is not cosmetic: a count-and-clock system prizes reliability and reproducibility between raters, while the descriptive system prizes clinical validity and warns against reifying an arbitrary threshold, and knowing which philosophy a given case file follows tells the reader why one record insists on four weeks and another speaks of several months.

AXIS	FIXED-CRITERIA SYSTEM	ICD-11 CLINICAL DESCRIPTIONS
Symptom count	An explicit three-of-eight threshold	No fixed count; a clinical judgement
Duration	At least four weeks in the under-eighteens	At least several months, not a fixed count
Code	Separation anxiety disorder	6B05 (mapping to F93.0 under age six)

The same disorder handled by two classificatory philosophies; the load-bearing contrast is the fixed four-week count against a clinical judgement that symptoms have lasted several months.

IN INDIA

Because Indian services classify under ICD, the operative label in case files, referrals and official reports is the ICD entity, and the clinician is not obliged to satisfy an exact four-week count so much as to judge that the separation anxiety has persisted, is developmentally excessive and is impairing. A trainee schooled on the count-and-clock manual should not treat a record that speaks of several months rather than four weeks as incomplete: it is following the classification actually in force here, and the two systems are describing the same child.

12.7 Assessment and the screening instruments

Assessment is multi-informant, developmentally framed and helped, but never replaced, by a rating scale. The history is taken from the child and, separately, from the parents, because a young child's account and a caregiver's account of the same anxiety frequently diverge, and school information anchors the impairment. Among structured aids the most widely used is the **Screen for Child Anxiety Related Emotional Disorders**, the SCARED, developed by Birmaher and colleagues. It is a 41-item questionnaire yielding five factors - panic or somatic symptoms, generalised anxiety, separation anxiety, social phobia and school phobia - so that it screens not only for this disorder but across the childhood anxiety family, whose members are developed in their own account elsewhere in this chapter. It has matched child and parent report versions, is intended for roughly ages nine to nineteen, is freely available, and takes under fifteen minutes to complete; a short five-item version also exists for rapid screening. Its value is precisely that it captures the separation factor within a broader anxiety screen, which suits a disorder that rarely travels alone.

GOOD TO REMEMBER

Use the SCARED as a screen and a way of structuring the interview across the anxiety factors, not as a diagnostic verdict. A scale flags where to look; the diagnosis still turns on the developmental-inappropriateness, duration and impairment judgement made from the history, and a high separation-anxiety factor score in a child whose function is intact is a prompt to reassess, not a diagnosis.

12.8 Management and the borders that matter

Treatment is psychological first, pharmacological second, and always delivered partly through the parents. The best-supported first-line intervention is a structured, exposure-based **cognitive behavioural therapy**, in which the child is helped to face graded separations while the accompanying catastrophic cognitions about harm and loss are tested and revised, and in which caregivers are coached out of the accommodation - the reassurance, the sleeping-in, the staying at home - that inadvertently maintains the fear. For more severe or refractory presentations, pharmacotherapy is added, with the serotonergic antidepressants the usual choice; the specific agents, doses and the youth-specific safety monitoring that any antidepressant in a young person requires are the province of the paediatric mood and antidepressant accounts and are not

reproduced here. The clinically decisive point is that medication supplements, and does not replace, the psychological and family work, because the disorder is sustained by a learned cycle of avoidance and accommodation that only exposure can unwind.

The management borders are as examinable as the treatment itself. Separation anxiety disorder is the single commonest engine of **school refusal**, but the two are not the same thing - school refusal has other causes, from social anxiety and depression to bullying and learning difficulty, and its assessment and management are developed in their own right elsewhere in this chapter. It must also be held apart from selective mutism, another childhood anxiety condition treated separately here, and from the frank school truancy of the conduct-disordered child, who stays away from school without anxiety and usually without the parents' knowledge, rather than clinging to a caregiver in fear. The anxious child who cannot get to school and the defiant child who will not are two different clinical problems, and the exam reliably rewards the distinction.

TRAP **School refusal is not a diagnosis, and separation anxiety disorder is not its only cause.** Candidates conflate the two, answering "separation anxiety disorder" whenever a child will not attend school. The anxious, home-clinging non-attender whose parents know exactly where he is points to this disorder; the child who leaves for school and never arrives, without anxiety and without the parents' knowledge, is truant, which belongs to the disruptive rather than the anxious spectrum.

3 of 8	4%	73%
SYMPTOMS, WITH A FOUR-WEEK MINIMUM DURATION	PREVALENCE IN CHILDREN UNDER 12	HERITABILITY IN A TWIN SAMPLE

13 · School refusal and school phobia (assessment and management)

The single most useful thing this topic teaches is that "not going to school" is a behaviour, not a diagnosis. A child who is repeatedly absent has told you almost nothing about why, and the entire clinical and examination value of the subject lies in taking that one observable fact apart into the very different problems that produce it. Absenteeism sits at the meeting point of psychiatry, paediatrics, the family and the school, and the marks are here because the assessor wants to see a candidate move from the presenting behaviour to the mechanism underneath it, and then to a management plan that follows from the mechanism rather than from the behaviour.

The vocabulary is where candidates lose easy marks, because the words are used loosely in ordinary speech and precisely in the exam. This topic owns the terminology of school non-attendance, the assessment that separates its varieties, and the management that flows from that separation. The individual anxiety disorders that so often lie beneath school refusal are each developed in their own right elsewhere in this chapter and are named here only at the point where the differential needs them, because the skill being tested is not the re-recitation of separation anxiety criteria but the ability to recognise which disorder is driving the child to the door and turning them back.

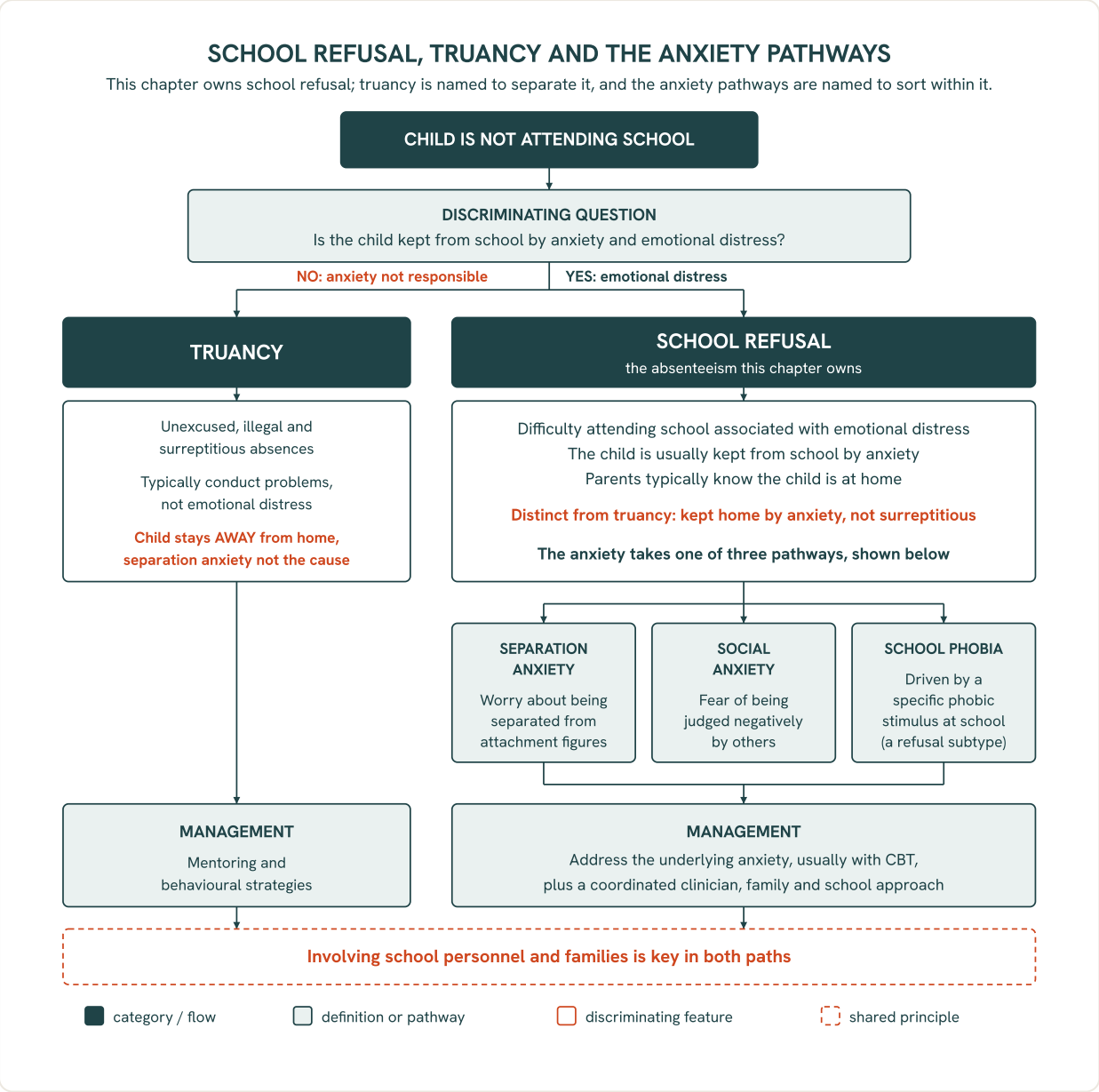

Fig 23.9. School refusal against truancy and the three anxiety pathways: one discriminating question splits anxiety-driven school refusal from surreptitious truancy, school refusal then sorts into separation anxiety, social anxiety or school phobia, and the management path diverges - mentoring and behavioural strategies for truancy, addressing the underlying anxiety for school refusal - while school and family involvement stays key in both.

13.1 Three problems wearing one presentation

The same empty desk has at least three quite different explanations. The first is **truancy**, which is absence that is unexcused, concealed and often against the rules, in a child who characteristically does not want to be in school and shows no particular emotional distress about staying away. Truancy is typically embedded in a wider pattern of conduct problems rather than in anxiety, and the child usually hides the absence from parents. The second is **school refusal**, difficulty attending school that is bound up with emotional distress: the child is not skipping school for something more enjoyable but is being kept from it by fear, dread or misery, and the distress is real and visible. The third and narrowest is **school phobia**, which is difficulty attending because of a specific phobia, a phobic stimulus located in or on the way to the school itself. These three are not points on a single spectrum of severity; they are different problems with different engines, and the first task of assessment is to say which one is in front of you.

RULE **Distress is the pivot on which the differential turns.** The absence that is comfortable, concealed and tied to rule-breaking points to truancy and its conduct-problem context; the absence that is distressing, known to the parents and driven by fear points to school refusal and an anxiety disorder beneath it. Every later decision, assessment and management alike, follows from which side of that line the child falls.

13.2 Why "school refusal" is a symptom and not a disorder

School refusal is a final common pathway, and the diagnosis lives one layer down. No classificatory system carries "school refusal" as a disorder, and that is the conceptual heart of the topic. It is a presenting behaviour produced by any of several anxiety disorders, and the clinical job is to identify which one. Three pathways account for most cases. In the youngest children the driver is most often separation anxiety, the fear not of school itself but of being apart from an attachment figure, so that the child would be equally reluctant to go anywhere that meant separation; separation anxiety disorder is developed in its own section of this chapter and is only named here. In older children and adolescents the driver is frequently social anxiety, where the avoidance is generated by **fear of being judged negatively by others** rather than by worry about leaving home, so that the feared object is the classroom, the corridor and the crowd rather than the parent's absence. The third pathway is the true school phobia, in which a discrete phobic stimulus connected with the school compels avoidance. Social anxiety disorder and specific phobia are themselves taught elsewhere in this chapter; what belongs here is the recognition that each can surface as the identical behaviour of a child at the school gate who cannot go in.

PATHWAY BENEATH THE REFUSAL	WHAT THE CHILD IS ACTUALLY AFRAID OF	WHERE THE FEAR POINTS
Separation anxiety	Being apart from an attachment figure; harm befalling them or the self while apart	Away from the parent, not at the school as such; reluctance to sleep away or be left anywhere
Social anxiety	Being negatively evaluated by others rather than separation from home	The peer group, being watched, performing, the corridor and the classroom
Specific phobia (school phobia)	A discrete phobic stimulus located in or en route to the school	The particular object or situation, avoidance narrowing onto it

The three anxiety routes to the same behaviour. Naming the route is the diagnosis; the behaviour of non-attendance is shared and by itself distinguishes none of them.

13.3 Truancy versus school refusal: the fork that decides everything

The two look alike from the school register and diverge on almost every clinical axis. Distinguishing conduct-driven truancy from anxiety-driven school refusal is the examinable core of assessment, and it is done from a handful of discriminating features rather than from the fact of absence. The most reliable single discriminator concerns where the child is while not in school. In truancy, and in the school avoidance that goes with conduct disorder, the child or adolescent typically stays away from the home rather than returning to it, and separation anxiety is not what is keeping them out; the absence is spent elsewhere, often with a peer group, and is concealed

from the parents. In school refusal the opposite holds: the child is usually at home, and the parents usually know it, because the child cannot bear to leave and the distress is directed at going, not at being caught. Conduct disorder, within which truancy characteristically sits, is developed in its own section of this chapter and is named here only as the context that truancy points towards.

FEATURE	TRUANCY (CONDUCT-LINKED)	SCHOOL REFUSAL (ANXIETY-LINKED)
Emotional state about attending	Little distress; the child prefers not to attend	Marked distress, fear or dread centred on going
Where the child is during absence	Away from home, often with peers	At home, close to the attachment figure
Parental knowledge	Usually concealed from parents	Usually known to the parents
Associated pattern	Broader conduct problems and rule-breaking	An underlying anxiety disorder
Schoolwork attitude	Often accompanied by loss of interest in schoolwork	Often a conscientious child who wants to keep up

The clinical axes that separate the two great categories of absence. No single row is infallible, but the pattern across them is what the assessor is testing.

TRAP **Treating "school phobia" as a synonym for all school refusal.** The older literature used "school phobia" loosely for any anxious non-attendance, and candidates inherit the habit. In precise use it is the narrow subtype in which a specific phobia drives the avoidance; most school refusal is not a true phobia of the school at all but separation or social anxiety wearing the school as its stage. Answering that a young, separation-anxious child has "school phobia" misnames the mechanism and, worse, points management in the wrong direction.

13.4 Assessment: reading the pattern, not just the absence

The history is built to expose the mechanism, and its shape is characteristic. A useful assessment establishes the onset and its triggers, the daily pattern, the somatic accompaniments and the family response. Onset is often tied to a transition or a discrete stressor: starting a new school, moving up a stage, returning after an illness or a holiday, a bereavement, bullying, or a humiliation in class. The daily pattern is a strong clue, with distress and physical symptoms mounting on school mornings, on Sunday evenings and at the start of term, and easing markedly at weekends, in the holidays and once the child is allowed to stay home, a Monday-to-Friday rhythm that anchors the problem to school rather than to a generalised illness. A meticulous account of what the child does during the day, and of whether the parents know where they are, does much of the work of separating refusal from truancy on its own.

The somatic presentation deserves particular emphasis because it is how many of these children first reach a doctor. Recurrent abdominal pain, headache, nausea and non-specific malaise, appearing on school mornings and resolving when attendance is not required, are the common currency of anxious school refusal, and the child is frequently worked up for a physical illness before the pattern is recognised. This somatisation of distress is itself developed elsewhere in this

chapter, and the point to carry here is only that a negative physical work-up in a child whose symptoms keep the timetable of the school week should redirect the assessment towards anxiety rather than prolong the search for organic disease. Corroborating history from the school, and from several informants, is essential, and the fuller apparatus of child psychiatric assessment and the involvement of services is developed in the Child psychiatry: assessment, treatment, services and protection notes.

- onset and its trigger: a school transition, an illness, a bereavement, bullying or a classroom humiliation;
- the weekly rhythm: worse on school mornings and at term's start, better at weekends and in holidays;
- somatic symptoms timed to attendance and clearing when the child stays home;
- where the child spends the day and whether the parents know, the discriminator from truancy;
- the family's response, and whether accommodation has quietly become the pattern.

GOOD TO REMEMBER

Ask the parents to narrate a single school morning in detail, from waking to the point at which the child either leaves or does not. That one concrete account usually reveals the mechanism faster than any checklist: the pleading and the physical complaints, the exact moment the distress peaks, whether it is the parting or the classroom that is dreaded, and how the household has learned to respond. The pattern of who does what at that moment is also the raw material for the behavioural plan that follows.

13.5 Management follows the mechanism

The management splits exactly where the diagnosis split, and the two paths are not interchangeable. Because truancy and school refusal have different engines, they call for different interventions. Truancy is best met with mentoring and behavioural strategies, whereas school refusal is best managed by treating the anxiety underneath it, usually with cognitive behavioural therapy and a coordinated approach across clinician, family and school. In both, the involvement of school personnel and the family is decisive, because the behaviour is enacted in the space between home and school and cannot be shifted from the clinic alone. For the anxious school refuser the therapeutic centre of gravity is a **graded return to school**: a planned, stepwise re-exposure that begins below the point of overwhelming anxiety and advances as tolerance builds, so that avoidance is not reinforced by escape and the child relearns that the feared situation can be survived. The longer a child is out, the harder re-entry becomes, which is why an early, structured, sympathetic push to return generally serves the child better than an open-ended wait for the anxiety to pass on its own.

The work is necessarily done by a triad. The clinician formulates the driving disorder and delivers or directs the anxiety treatment; the family is coached to hold the plan at the difficult morning moment and to stop inadvertently rewarding non-attendance; and the school arranges the graded

timetable, a point of safety within the building and the flexibility that a stepwise return requires. Where the underlying disorder is severe, medication for the anxiety disorder may be added to therapy, but the specific agents and their dosing belong with the pharmacology of the individual anxiety disorders and are not settled by this topic. Comorbid conditions and the family's own difficulties are addressed in parallel, since an unaddressed parental anxiety or an unrecognised depression in the child will quietly undo the best-laid return plan.

PITFALL

The recurring clinical failure is to misread the category and then apply the wrong lever. An anxious school refuser handled as a truant, with sanctions, threats and a purely disciplinary response, is punished for a fear they cannot simply choose to drop, and the anxiety and the avoidance both deepen. The mirror error, treating a conduct-driven truant with gentle, anxiety-focused accommodation, rewards the very absence it means to cure. Naming the mechanism first is not an academic nicety; it is what decides whether the intervention helps or harms.

IN INDIA

Several features of Indian practice sharpen this topic. These children very often present first to a paediatrician or a general practitioner with recurrent abdominal pain or headache, and the psychiatric mechanism is reached only after a physical work-up, so the index of suspicion has to be carried by the first doctor. The intense weight placed on academic performance, tuition and board examinations can both precipitate anxious refusal and make families read it as laziness or defiance, which pushes them towards a punitive response and away from the graded, collaborative plan that actually works. Coordinated clinician, family and school management, straightforward to prescribe, is harder to deliver where schools have little counselling provision and mental-health literacy is low, so a large part of the clinician's task is patient liaison and psychoeducation with parents and teachers who may never have heard the behaviour named as anxiety.

13.6 Course, complications and why the marks are here

Left unaddressed, the problem compounds. School refusal tends to entrench: avoidance is self-reinforcing, each absence makes the next one harder, and prolonged absence brings secondary losses that outlast the original fear. Academic slippage, social withdrawal from a peer group the child no longer sees daily, erosion of confidence and, in the family, a slow accommodation that hardens around the absence are the characteristic complications, and the anxious school refuser who never returns is at risk of carrying anxiety disorders and impaired functioning into adult life. The prognostic levers are the ones assessment and management have already identified: the earlier the recognition and the shorter the time out of school, the better the outlook, while long-standing refusal in an older child with a well-established pattern is harder to reverse. This is why the topic rewards a candidate who can move cleanly from the shared behaviour to the specific mechanism and then to a mechanism-matched plan, because that sequence is exactly what changes the trajectory.

Not-going-to-school is a behaviour, not a diagnosis: separate the concealed, distress-free absence of truancy from the distressing, parent-known absence of school refusal, find the anxiety disorder driving the refusal, and manage each along its own path, with a graded return to school at the centre of the anxious child's plan.

Distress	At home	Graded
THE PIVOT: ITS PRESENCE MARKS REFUSAL, ITS ABSENCE MARKS TRUANCY	WHERE THE REFUSER WAITS AND THE PARENTS KNOW; THE TRUANT IS AWAY	THE STEPWISE RETURN TO SCHOOL AT THE CENTRE OF ANXIOUS-REFUSAL MANAGEMENT

14 · Anxiety disorders in children (generalized, social, specific phobia)

Anxiety is the commonest form of childhood psychopathology, and the marks here reward the candidate who can hold three disorders apart while remembering what binds them. This topic covers the childhood presentations of generalized anxiety disorder, social anxiety disorder and specific phobia. These are ordinary human states of worry and fear that have crossed a threshold: they have become excessive for the child's developmental stage, persistent rather than passing, and costly to functioning at home, at school or among peers. The examinable difficulty is not the existence of these disorders, which every candidate can name, but the child-specific modifications the classifications make to their adult criteria, the developmental content of the fear, and the border between a disorder and the fears and worries that are normal in growing up.

What is owned here is the child-specific criteria and epidemiology of these three disorders. The wider family of childhood anxiety - separation anxiety disorder, selective mutism, and the anxiety that drives school refusal - is developed in its own right elsewhere in this chapter, as are paediatric obsessive-compulsive disorder and childhood post-traumatic stress disorder; here they are named as relatives, not reopened. The general adult anxiety disorders, their neurobiology and their pharmacology are not the business of this chapter. The accompanying figure places these disorders as one anxiety family so that their overlap and comorbidity can be seen at a glance.

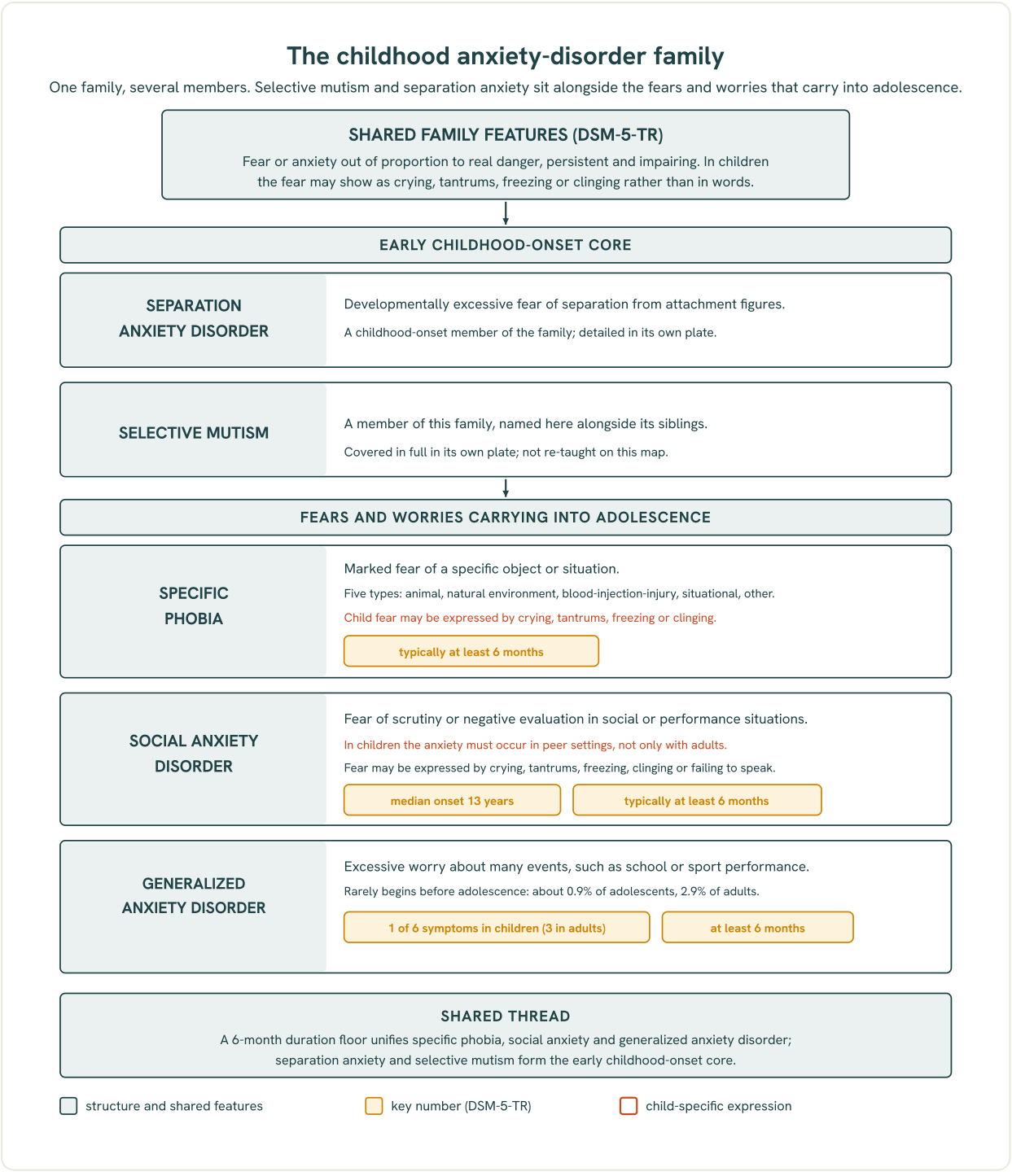

Fig 23.10. The childhood anxiety-disorder family: an early childhood-onset core (separation anxiety disorder and selective mutism, the latter named only) alongside specific phobia, social anxiety disorder and generalized anxiety disorder, unified by a fear out of proportion to danger, a typical 6-month duration floor and a child-specific expression through crying, tantrums, freezing or clinging.

14.1 The shared architecture: what makes childhood fear a disorder

Across all three disorders the diagnosis turns on the same three qualities: the fear is excessive, it is persistent, and it impairs. A young child who is frightened of the dark, shy with strangers or worried before an examination is not thereby ill; fear and worry are adaptive and developmentally expected. The classifications convert these normal states into disorders only when the response is out of proportion to any real danger, endures well beyond the situation that provoked it, and interferes with the child's life. This is why each of the three carries a minimum

duration and an impairment requirement, and why the clinician's first move is always to weigh the presentation against what is normal for that age. The core pathology is not different from the adult form; what differs is how a developing child expresses fear and how the thresholds are set for someone who cannot yet regulate or articulate distress. Holding this shared frame in mind is what stops a candidate from memorising three unconnected symptom lists and losing the reasoning that actually earns the marks.

RULE **Childhood anxiety disorders differ from their adult forms in expression and threshold, not in essence.** The fear is the same fear; the child shows it through behaviour rather than words, and the criteria are modified to fit a developing person. Diagnose when the fear or worry is excessive for the child's stage, persistent rather than transient, and genuinely impairing, and not before.

14.2 Generalized anxiety disorder: the worry and its childhood content

The essential feature is excessive, hard-to-control worry, and in children its content is characteristically about performance. **Generalized anxiety disorder** requires excessive anxiety and worry, occurring more days than not, about a number of events or activities, sustained for at least **6 months**. What marks the childhood form is the subject matter of the worry. Affected children **worry excessively about their own competence and the quality of their performance** at school or in sport, and they do so even when their performance is not being evaluated by anyone. Such children are often overly conforming, perfectionistic and unsure of themselves, and they tend to seek repeated reassurance about their work and their worth. This reassurance-seeking is diagnostically useful and clinically double-edged: it briefly relieves the child but draws the parent into providing it again and again, maintaining the very worry it is meant to settle. The worry is not tied to a single feared object, which separates it from specific phobia, nor to separation or to social scrutiny specifically.

14.3 The associated-symptom criterion and the child modification

The single most tested point in this disorder is that children need far fewer associated somatic-cognitive symptoms than adults. The worry must be accompanied by physical and cognitive symptoms drawn from a fixed set of six. In adults, at least three of the six must be present; in children, however, only **one** of the six is required. This is a deliberate developmental concession: a worried child may not yet report or recognise the full cluster of arousal symptoms an adult can describe, so the bar is set lower to avoid missing genuine cases. The **six associated symptoms** are the familiar mix of arousal and its consequences.

- restlessness or feeling keyed up or on edge;
- being easily fatigued;
- difficulty concentrating or the mind going blank;
- irritability;
- muscle tension;
- sleep disturbance.

MNEMONIC

Restless, Run-down, Distracted, Irritable, Tense, Sleepless

The six associated symptoms in order: **R**estless (restlessness or feeling keyed up), **R**un-down (easily fatigued), **D**istracted (difficulty concentrating or mind going blank), **I**rritable, **T**ense (muscle tension) and **S**leepless (sleep disturbance). In a child, remember that any single one of these, alongside the worry, satisfies the criterion.

14.4 Generalized anxiety disorder: epidemiology and course

The disorder is uncommon before adolescence, and that fact carries clinical weight. Twelve-month prevalence among adolescents is around **0.9%**, and the disorder rarely occurs before adolescence, with rates rising as the child moves through the teenage years toward the higher figures seen in adulthood. The developmental point behind the numbers is that the sustained, future-oriented, self-evaluative worry that defines this disorder depends on cognitive capacities - anticipating, comparing oneself against a standard, projecting consequences forward - that mature through late childhood and adolescence. A genuinely generalised, chronic worry in a very young child is therefore unusual and should prompt a careful look for another explanation: a more circumscribed anxiety disorder such as separation anxiety, a response to an ongoing stressor, or worry anchored to a specific feared situation. The disorder tends to be chronic and to fluctuate, and it is a frequent forerunner of, and companion to, depressive disorders as the young person grows.

14.5 Social anxiety disorder: the fear and its child-specific expression

Two child-specific rules define this disorder in the young: the fear must reach peers, and it may be shown as behaviour rather than spoken as fear. **Social anxiety disorder** is a marked fear of social situations in which the child is exposed to possible scrutiny by others, driven by a fear of acting in a way, or showing anxiety symptoms, that will be negatively evaluated. The first modification is a safeguard against overdiagnosis: in children the anxiety must occur in settings with peers and not only during interactions with adults. Many children are reticent with unfamiliar grown-ups while being entirely comfortable among other children, and that pattern is not this disorder; genuine social anxiety disorder shows itself with age-mates, in the playground and the classroom, not merely in the consulting room. The second modification concerns how the fear surfaces. A young child rarely says that they fear negative evaluation; instead the fear may be expressed by crying, tantrums, freezing, clinging, shrinking, or failing to speak in social situations. The clinician must read these behaviours as the child's language of fear rather than as defiance or as a separate problem.

GOOD TO REMEMBER

A child who freezes, clings or falls silent when peers are present is telling you about fear, not staging a protest. Because the same child may speak freely at home, the diagnosis is easily missed if the assessment relies only on the parents' account of a "shy" or "stubborn" child; ask specifically about behaviour in the presence of other children, and gather a report from school before the shyness is dismissed.

14.6 Social anxiety disorder: duration, onset and trajectory

The duration requirement is what separates the disorder from the ordinary and often intense social fears of childhood. The fear, anxiety or avoidance must be persistent, typically lasting 6 months or more. This floor matters more in the young than in adults precisely because transient social fears - a phase of clinginess, a spell of reluctance to perform, a wariness after a change of school - are so common in children and so often self-limiting. The persistence requirement forces the clinician to wait past the passing phase before affixing a diagnosis. In its course, social anxiety disorder has a characteristically early onset: the median age at which it begins is about **13** years, and it frequently emerges out of a childhood history of **behavioural inhibition** or marked shyness, the temperamental tendency to withdraw from the unfamiliar that can be observed from the earliest years. That developmental continuity, from an inhibited temperament in the toddler to a diagnosable disorder in the adolescent, is a favourite theme of examiners and a real target for early intervention.

14.7 Specific phobia: the phenomenon, the childhood expression and the five types

A specific phobia is fear out of proportion to a circumscribed object or situation, and in children it too speaks through behaviour. **Specific phobia** is a marked, disproportionate fear or anxiety about a specific object or situation, which is actively avoided or endured with intense distress, and which is out of keeping with any actual danger. As in social anxiety, the young child's fear may be expressed by crying, tantrums, freezing or clinging rather than described. The phobias are grouped by the nature of the feared stimulus into **five** specifier types.

SPECIFIER TYPE	TYPICAL FEARED STIMULUS
Animal	Dogs, insects, snakes
Natural environment	Heights, storms, water
Blood-injection-injury type	Needles, injections, the sight of blood
Situational	Enclosed spaces, aeroplanes, the dark
Other	Choking, vomiting, costumed figures

The five phobic-stimulus specifiers by which specific phobia is coded. The blood-injection-injury type is set apart by its distinctive physiology.

One type behaves unlike the rest. The blood-injection-injury type is associated with a vasovagal fainting response: rather than the sustained sympathetic arousal of the other phobias, exposure provokes an initial rise and then a sharp fall in heart rate and blood pressure, and the person faints. This is why the child who passes out at the sight of a needle or blood belongs in a

physiological category of their own, and why the applied relaxation used for other phobias is combined here with applied tension to counter the drop in blood pressure.

14.8 Specific phobia: duration and the border with normal childhood fear

Because circumscribed fears are near-universal in childhood, the persistence requirement is the safeguard that keeps a normal fear from being called a disorder. Fear of the dark, of animals, of strangers and of imaginary creatures follows a well-described developmental sequence, each fear appearing and then fading as the child matures; these are part of normal development, not pathology. What converts such a fear into a specific phobia is that it is persistent, typically lasting 6 months or more, disproportionate to real danger, and productive of real impairment or avoidance. The threshold is therefore not the presence of the fear but its persistence and its cost. The normative sequence of childhood fears belongs to the Normal child development and milestones notes; the diagnostic line drawn here is simply that a fear which has outlasted its developmental window, and which is now distorting the child's life, has become a disorder. This is the specific threshold this topic owns; the broader map of normal-versus-disordered childhood behaviour is drawn elsewhere in this chapter.

> **PITFALL**
>
> The error runs both ways. On one side, an anxious parent and clinician pathologise the developmentally ordinary fear of the dark or of dogs in a preschooler, ignoring that the fear is age-appropriate and will pass; on the other, a genuinely impairing, persistent phobia in an older child is waved away as "a phase" long after the phase should have ended. The corrective in both directions is the same: measure the fear against the developmental norm and against the twin tests of persistence and impairment, not against the family's level of alarm.

14.9 Assessment, comorbidity and management

Find one childhood anxiety disorder and look for the others, because they cluster, and treat first with graded exposure. These three disorders are highly comorbid with one another, with the rest of the anxiety family, and with depression as the child grows, so the discovery of one should trigger a screen for the rest. Assessment is necessarily multi-informant: child, parents and school each see a different slice of the anxiety, and the criterion that the fear reach peers, or that worry persist across settings, cannot be judged from one source. Childhood anxiety also hides behind other complaints. It commonly presents through recurrent physical symptoms such as abdominal pain and headaches, and through reluctance or refusal to attend school; those presentations are developed in their own right elsewhere in this chapter and are flagged here only so the anxiety behind them is not missed. The first-line treatment across all three is **exposure**-based cognitive behavioural therapy, in which the child, supported by parents, is helped to approach the feared situation in graded steps while the avoidance that maintains the fear is dismantled; parental accommodation, the well-meaning reassurance and avoidance that families build around an anxious child, is a specific target. Medication is reserved for more severe or refractory presentations and is layered onto the psychological work, never a substitute for it.

IN INDIA

Two realities shape presentation here. First, services classify under ICD, so these disorders appear in case files under their ICD labels rather than the DSM ones, and the clinician reads across the two systems. Second, childhood anxiety is under-detected: it surfaces in paediatric and general clinics as unexplained abdominal pain, headache or school refusal rather than as a complaint of worry, and the intense cultural emphasis on academic performance can both feed the performance-focused worry of generalized anxiety disorder and cause it to be normalised as ordinary diligence. Trained delivery of exposure-based cognitive behavioural therapy remains scarce outside specialist centres, which places a premium on equipping parents and schools to carry the graded-exposure approach.

FEATURE	GENERALIZED ANXIETY	SOCIAL ANXIETY	SPECIFIC PHOBIA
Focus of fear	Diffuse worry about competence and performance	Scrutiny and negative evaluation by others	A single circumscribed object or situation
Child modification	Only one associated symptom required	Must occur in peer settings, not only with adults	Fear shown as crying, tantrums, freezing or clinging
Behavioural expression in the young	Reassurance-seeking, perfectionism, conforming	Crying, freezing, clinging, mutism	Avoidance, distress on exposure
Duration floor	at least 6 months	at least 6 months	at least 6 months

The three disorders on shared axes; the shared six-month persistence floor sits beneath all of them, and each carries its own developmental modification to the adult criteria.

TRAP **Candidates apply the adult criteria to the child and lose the modifications.** The two most frequently missed are that generalized anxiety disorder in a child requires only *one* associated symptom rather than the adult three, and that social anxiety in a child must be present in *peer* settings and not only with adults. Reproducing the adult thresholds verbatim either excludes a genuinely anxious child or counts ordinary adult-directed reticence as a disorder.

Carry into the exam: all three disorders share a persistence floor of about 6 months plus impairment, and each modifies the adult criteria for the child - generalized anxiety needs only one associated symptom, social anxiety must reach peers, and specific phobia and social anxiety may be shown as crying, freezing or clinging rather than spoken.

6 months SHARED PERSISTENCE FLOOR	**1 of 6** ASSOCIATED SYMPTOMS NEEDED IN A CHILD (GAD)	**0.9%** 12-MONTH PREVALENCE IN ADOLESCENTS (GAD)	**13 yrs** MEDIAN ONSET OF SOCIAL ANXIETY

15 · Selective mutism

The child can speak, and that single fact is the whole diagnosis. **Selective mutism** is the condition of a child who talks fluently and comfortably in the settings where they feel safe, most often at home with immediate family, yet consistently fails to speak in particular social situations

where speech is expected of them, the classroom being the prototype. The speech apparatus is intact, the vocabulary is present, and the child demonstrates both every evening at home; what fails is the ability to produce speech under the specific social pressure of being watched, addressed or expected to answer. This is not shyness that a warm teacher can coax away in a week, nor a refusal that a firm one can command out of the child. It is a persistent, situation-bound silence that the child cannot simply choose to break, and understanding it as an anxiety phenomenon rather than a wilful or a linguistic one is the key to the entire topic.

The marks here are discriminative. An examiner uses this diagnosis to test whether a candidate can separate an anxiety-driven, situational mutism from the several conditions it superficially resembles: ordinary developmental shyness, the silent settling-in of a child learning a new language of instruction, a primary communication or language disorder, a hearing impairment, and the social muteness that can accompany autism spectrum disorder. This topic owns selective mutism in full: its defining criterion, its duration and exclusion rules, its standing as an anxiety disorder, its epidemiology, onset and course, and its management. The wider childhood anxiety family of which it is a member is developed in its own right elsewhere in this chapter and named here only where the reasoning needs it.

15.1 The essential feature: a situational, not a global, failure of speech

The diagnosis lives in the contrast between the two settings. The core requirement is a consistent failure to speak in specific social situations in which there is an expectation to speak, present at the same time as normal speech in other situations. It is the coexistence of the two that defines the disorder: a child who is mute everywhere has a different problem, but a child who chatters at home and falls silent at school shows the characteristic split. The failure must be consistent, not the occasional off day, and it must occur where speech is genuinely expected, so that a child who happens to be quiet in a setting that makes no demand on them is not captured. Because the child can and does speak elsewhere, the clinician has direct evidence that the machinery of speech is unimpaired; the deficit is one of performance under a particular kind of social threat, not of competence.

RULE **Speech preserved in one setting, absent in another, is the disorder.** Selective mutism requires demonstrated normal speech in a comfortable setting alongside a consistent failure to speak where speech is expected. If the child is mute in every setting, the diagnosis is not selective mutism, and the assessor should be considering a language disorder, a hearing deficit, autism spectrum disorder or a global mutism instead.

15.2 The duration rule and why the first month of school does not count

The one-month floor exists to exclude normal settling-in. The disturbance must last for **at least one month**, and, importantly, that qualifying month is explicitly not the first month of school. This is not an arbitrary threshold. It is entirely normal for a child to be reticent, subdued or briefly silent in the first weeks of a new class, a new school or a new language environment, and that transient reserve settles on its own as the child habituates. Requiring the mutism to persist beyond that initial settling-in period screens out this benign, self-limiting reticence and reserves the diagnosis for a silence that has proven itself durable. A candidate who forgets the

exclusion of that first month will over-diagnose the ordinarily anxious new entrant, which is precisely the error the criterion is written to prevent.

15.3 The exclusion criteria: language, communication and pervasive disorders

The silence must be anxiety, not an artefact of language, speech or a broader disorder. Two exclusions do most of the diagnostic work. First, the failure to speak must not be attributable to a lack of knowledge of, or comfort with, the spoken language the situation demands. A child newly arrived into a school taught in an unfamiliar tongue may pass through a normal **silent period** while they acquire it, and that child does not have the disorder however complete their classroom silence. Second, the mutism must not be better explained by a **communication disorder** such as a childhood-onset fluency disorder, and it must not occur exclusively during the course of autism spectrum disorder or a psychotic disorder. Where the muteness is fully accounted for by one of those conditions, that condition is the diagnosis. These exclusions are what convert selective mutism from a description of a silent child into a specific, anxiety-based entity.

- not due to a lack of knowledge of, or comfort with, the required spoken language;
- not better explained by a communication disorder such as a fluency disorder;
- not occurring only within autism spectrum disorder or a psychotic disorder;
- separable, clinically, from ordinary shyness and from a hearing impairment.

CONDITION TO DISTINGUISH	WHAT SEPARATES IT FROM SELECTIVE MUTISM
The silent period of a new-language learner	The muteness reflects unfamiliarity with the language of the setting, not anxiety; the language-knowledge exclusion removes it
Communication or fluency disorder	A primary difficulty producing speech itself, present across settings, rather than a setting-bound silence with normal speech elsewhere
Autism spectrum disorder	The muteness sits within a pervasive social-communication deficit and a restricted, repetitive repertoire; selective mutism is not diagnosed if the silence occurs only within autism
Ordinary developmental shyness	Transient, coaxable and not functionally impairing; selective mutism is consistent, durable and disabling

The conditions the situational silence is most often confused with; each is separated by the source of the muteness rather than by its appearance, which is why the exclusion criteria carry the diagnosis.

TRAP **Diagnosing the bilingual child in a silent period.** The classic examination error is to label a recently immigrated or newly enrolled child, silent in a classroom taught in a language they are still acquiring, as having selective mutism. The language-knowledge exclusion exists precisely for this child: until they are comfortable in the language of the setting, their silence is not the disorder, and time and exposure, not treatment, are what they need.

15.4 An anxiety disorder in its own right

The company it keeps settles what kind of disorder it is. Children with selective mutism seen in clinical settings are almost always given an additional anxiety diagnosis, and the one that accompanies it most often is **social anxiety disorder**. That near-constant coincidence is the strongest argument that the mutism is a manifestation of anxiety rather than a communicative or an oppositional problem: the child who cannot speak at school is very often the same child who is intensely frightened of scrutiny and negative evaluation. Both major classifications now place the condition accordingly. It sits among the anxiety disorders in current DSM classification, and ICD-11 lists it as an **anxiety or fear-related disorder** under code **6B06**. This is not a filing detail; it directs treatment, because it tells the clinician to target the anxiety and not to treat the silence as a speech problem or a behaviour to be disciplined.

> Carry this into the exam: a child who speaks freely at home but consistently not at school for at least a month, and not explained by language or a communication disorder, is showing an anxiety disorder, almost always alongside social anxiety.

15.5 Epidemiology

A rare disorder, and commoner in the young. Selective mutism is uncommon. Point-prevalence estimates across clinic and school samples fall in the range of **0.03 to 1.9 per cent**, the width of that range reflecting how much the figure depends on the setting sampled and on the age of the children studied. It is more likely to manifest in young children than in adolescents and adults, in keeping with its early onset and its tendency to attenuate with age. The rarity has a practical consequence: because most clinicians see very few cases, the condition is easily missed or misattributed, and the child who is silent at school but never at home is often written off as merely shy long before anyone names the disorder.

15.6 Onset and course

Early to begin, late to be noticed, and it leaves something behind. Onset is usually before **age five**, but the condition frequently goes unrecognised until the child enters school, where the demand to speak in front of others suddenly exposes a silence that at home had looked like nothing more than a quiet temperament. School entry, in other words, does not cause the disorder so much as reveal it. The course is instructive: in many children the mutism itself fades with time, but the underlying social anxiety commonly persists, so that the disorder is better understood as an early, visible expression of an anxiety diathesis than as a self-contained childhood problem that resolves without trace. That residual anxiety is why early, anxiety-directed intervention matters, rather than simply waiting for the child to grow out of the silence.

15.7 Principles of management

Treat the anxiety, lower the pressure, and let speech be shaped, not forced. Because the disorder is anxiety-based, management is built on behavioural and family-and-school interventions that reduce the anxiety around speaking rather than demand speech directly. The mainstay is graded behavioural work: techniques such as stimulus fading, in which a person the child already speaks to slowly brings a new listener into the setting, and shaping, in which any

communication is rewarded and successively closer approximations to speech are encouraged, together with graded exposure to the feared speaking situations. Close collaboration with the school is essential, since the classroom is usually where the silence lives and where any gain has to be generalised. Where the disorder is severe or does not respond to behavioural work, a selective serotonin reuptake inhibitor is the pharmacological option most commonly added, on the same anxiety rationale that governs its use across the childhood anxiety disorders; specific agents and dosing lie outside what this topic establishes and should be confirmed against a pharmacology source.

GOOD TO REMEMBER

The single most useful instruction to give the parents and the teacher is to stop asking the child to speak. Direct pressure, coaxing, and rewards made contingent on an immediate spoken answer all raise the very anxiety that produces the silence. Removing the spotlight, accepting non-verbal responses at first, and letting a trusted adult widen the circle gradually does more than any amount of encouragement to talk.

PITFALL

The commonest clinical failure is to read the silence as oppositionality. A child who speaks fluently at home but says nothing to the teacher looks, to an untrained eye, as though they are choosing not to cooperate, and the response, more pressure, discipline or a battle of wills, predictably worsens matters. The child is not refusing; they are unable to speak under that specific anxiety, and treating it as defiance both misses the diagnosis and deepens the problem.

IN INDIA

Two features of Indian practice are worth holding in mind. Most services diagnose under ICD, where the condition now sits among the anxiety or fear-related disorders, which usefully reinforces the anxiety-directed approach. And the multilingual classroom makes the language-knowledge exclusion especially live: a child schooled in English, or in a state language different from the mother tongue spoken at home, may be silent simply because they are still acquiring the language of instruction. Establishing which languages the child speaks freely, and where, is the first step before the diagnosis is even entertained, and it is often what separates a genuine case from a child in a normal silent period.

0.03 to 1.9%	1 month	age 5	6B06
POINT-PREVALENCE RANGE, CLINIC AND SCHOOL SAMPLES	MINIMUM DURATION, BEYOND THE FIRST MONTH OF SCHOOL	ONSET USUALLY BEFORE	ICD-11 ANXIETY OR FEAR-RELATED CODE

16 · Paediatric OCD and PANDAS

Obsessive-compulsive disorder in a child is the same disorder as in an adult, but it wears a developmental disguise. A young child rarely arrives complaining of intrusive thoughts. The family notices the behaviour first: the handwashing that will not stop, the bedtime routine that must be repeated until it feels right, the questions asked again and again for reassurance. Marks

accrue here because the examiner wants to know that a candidate can recognise the disorder through that behavioural surface, grade its severity with the standard paediatric instrument, and hold a disciplined, sceptical line on the one piece of this territory that generates more heat than light, the proposed link between streptococcal infection and abrupt-onset childhood OCD. The general adult disorder, its full criteria set and its pharmacology, are developed in the Anxiety, OCD and trauma-related disorders notes; this topic owns the child-specific presentation, the standard rating scale, and the PANDAS and PANS story in full.

The structure to carry into the exam is simple. First, what OCD is and how its two building blocks behave in a child. Then how it is measured and how common it is. Then the post-infectious hypothesis, taught as a named, contested proposal rather than as settled fact, because that scepticism is itself the examinable point.

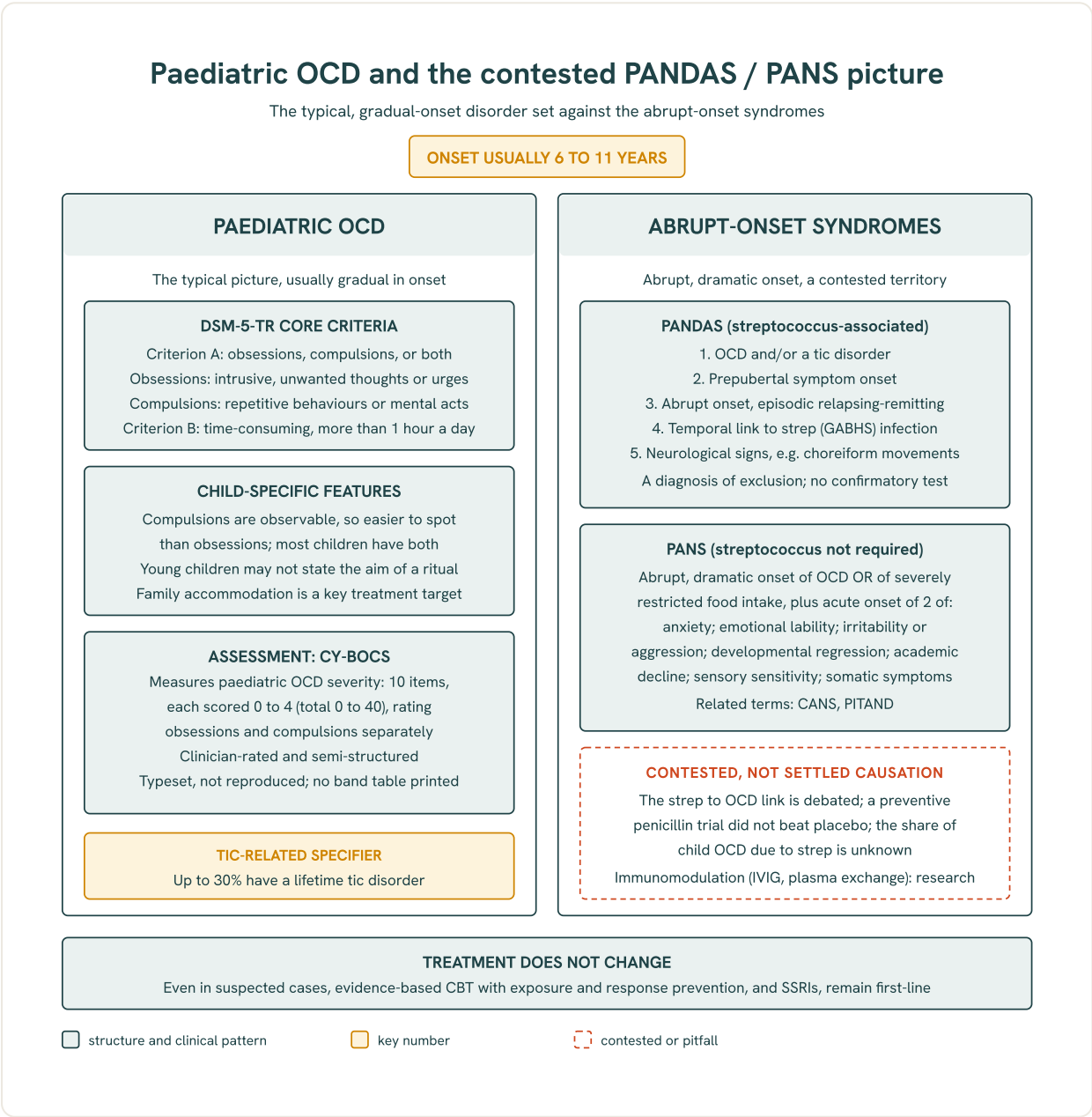

Fig 23.11. Paediatric OCD and the contested PANDAS and PANS abrupt-onset picture: the typical gradual-onset disorder with its DSM-5-TR criteria and its CY-BOCS severity assessment, set against the abrupt-onset syndromes whose streptococcal cause remains contested and whose treatment stays evidence-based CBT with exposure and response prevention, and SSRIs.

16.1 The defining features and how they present in a child

The disorder is built from two elements, and either can stand alone. The core diagnostic requirement is the **presence of obsessions, compulsions, or both**. Obsessions are recurrent, intrusive and unwanted thoughts, urges or images that the person experiences as distressing; compulsions are the repetitive behaviours or mental acts performed in response to an obsession, or according to rigid self-imposed rules, that are meant to reduce that distress or to prevent some feared event. The compulsion is functionally tethered to the obsession even when the child cannot articulate the link. The second requirement is that these symptoms exact a real cost. They must be time-consuming, taking **more than one hour a day**, or else cause clinically significant distress or impairment of functioning. That threshold is what separates a diagnosable disorder from the ordinary superstitions, rituals and preoccupations of normal childhood, which the reader will find mapped against their pathological counterparts elsewhere in this chapter.

RULE **Obsession drives, compulsion relieves.** The compulsion is not a random tic or habit; it is performed to neutralise the anxiety of an obsession or to satisfy a rigid rule. When you cannot see an obsession, ask what the child fears will happen if the ritual is stopped, and the obsession usually appears.

16.2 Why the child looks different from the textbook adult

In children the compulsion is the visible half of the disorder. Younger children often cannot describe the aims of their compulsions, so the **compulsions**, being observable, are more readily diagnosed than the obsessions that drive them, even though most affected children in fact have both. This inverts the adult stereotype of a patient who can narrate a tormenting thought. A second developmental feature carries genuine clinical weight: **family accommodation**, the involvement of parents or siblings in the child's rituals, providing the reassurance, performing the checks, or reorganising the household around the symptoms. Accommodation feels kind in the moment and is almost universal, but it maintains the disorder by removing every opportunity for the anxiety to fall on its own, and it is therefore an important treatment target, more so in children than in adults.

PITFALL

The commonest management failure is to leave family accommodation untouched. Parents who have spent months answering the same reassurance-seeking question, or supervising a two-hour bedtime ritual to keep the peace, are unwittingly holding the disorder in place. Unless the treatment plan explicitly coaches the family to withdraw accommodation in a graded, agreed way, exposure work with the child alone will keep failing at home.

16.3 Epidemiology, onset and sex

It is neither rare nor a purely adult disease. The most widely accepted six-month prevalence of OCD in the general paediatric population is **0.5 to 1 per cent**. The disorder is strongly developmental in its origins: **up to 80 per cent** of adults with OCD had their onset in childhood or adolescence, so the paediatric presentation is not a minor tributary of the adult disorder but its headwater. Mean age at onset falls between 6 and 11 years, with a bimodal pattern peaking

around puberty and again in young adulthood. The prepubertal, early-onset group has a distinctive profile: it shows a male preponderance and a higher rate of family history, whereas the sex ratio tends to even out by adolescence. That early-onset, male-predominant, familial, often tic-associated form is the one to keep in mind when the post-infectious hypothesis is raised.

16.4 The tic-related specifier and OCD-tic comorbidity

Childhood-onset OCD and tics travel together. DSM-5-TR carries a **tic-related specifier**, applied when the individual has a current or past tic disorder, precisely because the overlap is substantial: **up to 30 per cent** of people with OCD have a lifetime tic disorder, an association most common in males with childhood onset. The clinical importance is that tic-related OCD tends to present with a particular symptom flavour, with more just-right, symmetry and touching compulsions than pure contamination-and-washing pictures, and it sits at the crossroads of OCD and Tourette syndrome. The tic disorders themselves, their classification and management, are developed in their own right elsewhere in this chapter; here the specifier matters because it flags the early-onset, familial subtype and because it is the same subtype invoked by the streptococcal hypothesis below.

16.5 Measuring severity: the CY-BOCS

The standard paediatric instrument is worth knowing in structural detail. The **Children's Yale-Brown Obsessive Compulsive Scale** is a ten-item clinician-administered, semi-structured scale, each item anchored on an ordinal scale from 0 to 4. It precedes the rated items with a broad symptom checklist and then rates obsessions and compulsions separately across five dimensions each: the time occupied, the interference with life, the subjective distress, the internal resistance mounted against the symptom, and the degree of control the child retains over it. What it measures is paediatric OCD severity across those two halves, and severity is read off the summed total. No interpretive band table is printed here: the exact cut-offs separating mild from moderate from severe illness could not be confirmed against a primary source, and a wrong cut-off is worse than none, so learn the structure and the dimensions rather than a number line. The instrument is a licensed one, typeset from its published description rather than reproduced as a form. Because it is administered by the clinician rather than self-reported, it sidesteps the very problem that defines paediatric OCD, the child who cannot put the obsession into words, and it is the scale used both to grade severity at intake and to track response over treatment.

> **GOOD TO REMEMBER**
>
> Use the CY-BOCS as a change measure, not a one-off label. A total at intake, repeated at review, converts a vague impression that the child is "a bit better" into a defensible figure that tells you whether exposure therapy and, where used, a serotonergic agent are actually working, and whether family accommodation is falling as it should.

16.6 PANDAS: the proposed post-streptococcal syndrome

PANDAS names a hypothesised subgroup, not an established disease. **PANDAS**, paediatric autoimmune neuropsychiatric disorders associated with streptococcus, denotes OCD-like and tic symptoms proposed to be triggered by group A beta-haemolytic streptococcal infection. The

proposed mechanism is **molecular mimicry**: antibodies raised against streptococcal antigens are held to cross-react with, and inflame, the basal ganglia, producing an acute neuropsychiatric picture. The model is deliberately built on Sydenham chorea, the recognised post-streptococcal movement disorder of rheumatic fever, and it was first described by Swedo and colleagues in a small putative subgroup of children. The concept is attractive because it offers a mechanism and, tantalisingly, a treatable cause; the difficulty, taken up below, is that the evidence has never firmly closed the loop from infection to symptoms.

16.7 The working diagnostic features, and PANS

The clinical description is a checklist of features and a diagnosis of exclusion. The working features proposed for PANDAS are the presence of OCD or a tic disorder; prepubertal onset; an abrupt onset with an episodic, relapsing-remitting course; a temporal association with streptococcal infection; and associated neurological signs such as motoric hyperactivity and choreiform movements. There is no confirmatory test, so the diagnosis rests on this pattern and on excluding other causes.

- OCD or a tic disorder is present;
- onset is prepubertal;
- onset is abrupt and the course is episodic;
- there is a temporal link to streptococcal infection;
- neurological abnormalities, such as choreiform movements, accompany the presentation.

A broader term was later coined to escape the demand for a proven streptococcal cause. **PANS**, paediatric acute-onset neuropsychiatric syndrome, requires an abrupt, dramatic onset either of OCD symptoms or of severely restricted food intake, together with the acute onset of at least two of a wider list, anxiety, emotional lability, irritability or aggression, developmental regression, academic deterioration, sensory sensitivity, or somatic symptoms. The crucial contrast is that PANS, unlike PANDAS, does not require any link with or presumed cause by streptococcal infection; it is defined by the abrupt neuropsychiatric onset alone, and it too has no definitive test.

FEATURE	PANDAS	PANS
Streptococcal link	Required: a temporal association with streptococcal infection	Not required; no presumed streptococcal cause
Core symptom	OCD or a tic disorder	Abrupt OCD, or severely restricted food intake, plus acute onset of at least two further neuropsychiatric symptoms
Onset and course	Prepubertal, abrupt, episodic and relapsing-remitting	Abrupt and dramatic
Confirmatory test	None; diagnosis of exclusion	None; diagnosis of exclusion

PANS is the wider, streptococcus-agnostic term: it keeps the abrupt neuropsychiatric onset of PANDAS but drops the requirement for a streptococcal trigger, which is precisely the requirement that has proved hardest to substantiate.

16.8 The controversy, and why it does not change the treatment

This is the part the examiner is really testing. The causal claim behind PANDAS is **contested and not established**. The association between streptococcal infection and childhood OCD remains debated; efforts to establish a reliable biological marker have been inconclusive; the proportion of paediatric OCD actually attributable to streptococcus is unknown; and a prophylactic penicillin trial failed to separate from placebo, while several groups could find no dependable temporal association between infection and symptom flares. In the current literature PANDAS is described as one of the most **controversial** conditions in paediatric practice, with epidemiological data still lacking and few randomised studies to anchor any recommendation. The honest position, and the one to write, is that this is a proposed, contested autoimmune mechanism, never settled causation.

The practical consequence is reassuringly clear. Whatever one believes about the trigger, the treatment of a child who presents with OCD or tics does not change: evidence-based cognitive behavioural therapy with exposure and response prevention, together with a serotonergic agent where indicated, remains the mainstay even in suspected post-infectious cases. Immunomodulatory treatments such as intravenous immunoglobulin and plasmapheresis remain experimental and belong to research protocols, not to routine care.

TRAP **Presenting PANDAS as a settled, antibiotic-treatable cause of OCD.** The seductive error is to hear "abrupt-onset OCD after a sore throat" and reach for a streptococcal work-up and antibiotics as if the diagnosis were proven. It is not. Reproduce the contested status, keep the child on exposure-based therapy and, where indicated, a serotonergic agent, and reserve immunomodulation for research settings.

Paediatric OCD is diagnosed through observable compulsions and treated with exposure and response prevention plus an SSRI; PANDAS and PANS are contested, test-free hypotheses that never displace that treatment.

IN INDIA

Two local realities sharpen this topic. First, Indian services classify predominantly under ICD, and the CY-BOCS remains the workhorse severity measure across child psychiatry units. Second, the background rate of streptococcal disease and rheumatic fever is higher here than in the Western data that framed the PANDAS debate, which makes it tempting to over-attribute abrupt childhood OCD to infection. The discipline is the same as everywhere: a positive throat swab or a raised antistreptococcal titre in a child with new OCD proves exposure, not causation, and it must not divert the family from exposure-based therapy and, where indicated, a serotonergic agent.

0.5 to 1%	up to 80%	up to 30%	1 hour/day
SIX-MONTH PAEDIATRIC PREVALENCE	ADULT OCD WITH CHILDHOOD ONSET	LIFETIME TIC DISORDER IN OCD	TIME THRESHOLD, CRITERION B

17 · Post-traumatic stress disorder in children

A child is not a small adult with the same symptoms writ small. Post-traumatic stress disorder after a traumatic event is recognisable in a school-aged child or adolescent along broadly the same lines as in an adult, but in the youngest children it hides. A traumatised two-year-old does not describe a flashback; a traumatised four-year-old does not report emotional numbing. They repeat the event in their play, wake screaming from dreams whose content no one can trace, lose words they had already learned, and cling to a parent as though the world has become unsafe. Applying the standard adult symptom list to such a child systematically under-diagnoses them, and that under-recognition is the whole reason this topic carries marks.

The general trauma-related disorders, including the adult form of post-traumatic stress disorder, acute stress disorder, adjustment disorders and the underlying neurobiology of the fear circuitry, are developed in the Anxiety, OCD and trauma-related disorders notes; they are named here and not re-taught. What this topic owns is the paediatric problem: how trauma presents developmentally in young children, and the separate, developmentally modified diagnostic set that the current classification provides for them. The examinable core is that a child of a certain age is read against a different rulebook, and knowing which criteria change, and why, is what a candidate must show.

17.1 A separate rulebook for the youngest children

The main criteria are written for anyone older; the very young get their own set. The current classification splits the diagnosis by developmental stage. The principal post-traumatic stress disorder criteria are intended for adults, adolescents and children older than six years, and are read as they would be for an adult. For children who are **six years and younger**, a distinct and developmentally modified **preschool subtype** is applied instead. This is not a mere footnote softening the adult criteria; it is a genuinely separate set, with fewer required symptoms and with several clusters rewritten to match what a preverbal or barely verbal child can actually show. The single most useful thing to carry into the assessment room and the examination hall is the age boundary itself, because it decides which of two different diagnostic templates you are entitled to use.

RULE **Age decides the template.** A child of six years or younger is diagnosed on the preschool subtype, which lowers and re-shapes the symptom requirements to fit developmental expression. Reading such a child against the adult criteria, which demand more symptoms and more verbal reporting, is how a genuinely traumatised toddler is wrongly cleared.

17.2 Criterion A: the exposure, seen through the caregiver

For a young child the caregiver is the centre of the traumatic world. The exposure criterion in the preschool subtype is deliberately narrower and more caregiver-focused than the older-than-six version. A young child qualifies by directly experiencing the event, by witnessing it in person as it happened to others (particularly to primary caregivers), or by learning that the event happened to a parent or other caregiving figure. The logic is developmental: a small child's sense of safety is organised almost entirely around the attachment figure, so a threat to that figure is a

threat to the child, whereas the more abstract routes that count for older individuals do not yet have the same force. This differs from the criterion used for anyone older than six, which additionally counts learning of violent or accidental events happening to any close relative or friend, and repeated or extreme exposure to aversive details of trauma. Naming that contrast is often the mark: the young child's exposure is anchored to the people who care for them.

17.3 Criterion B: intrusion expressed as play

Reexperiencing in a young child is enacted, not narrated. The intrusion cluster requires one or more symptoms, but the crucial point is how they show. Intrusive memories in a young child **may appear as repetitive play** that refers, directly or symbolically, to the trauma, and this play need not look distressing to an observer; the child may seem absorbed or even calm while re-enacting a car crash or an assault with toys. Frightening dreams are common, but their content often cannot be tied to the event, so a new pattern of night terrors without an obvious theme still counts. Trauma-specific re-enactment may spill into behaviour and games. Because the child cannot say "I keep seeing it happen", the clinician must infer the intrusion from what the child does, which is why detailed collateral about play, sleep and behaviour is not optional here but is the actual data of the criterion.

17.4 Criterion C: avoidance and negative cognition, merged

Two adult clusters become one, and a single symptom will do. This is the structural heart of the subtype and the point most likely to be tested. In the version for anyone older than six, persistent avoidance and negative alterations in cognitions and mood are two separate clusters, each requiring its own symptoms. In the preschool subtype these are collapsed into a single Criterion C, and only one symptom from the combined list is needed, whether that is avoidance of reminders or a negative change such as social withdrawal, restricted play, or a marked reduction in the expression of positive emotions. The consequence to memorise is arithmetic: the preschool set is built on **three** symptom clusters rather than the four of the older version. The merge exists because a young child cannot reliably report the cognitive content, the persistent distorted blame or the sense of a foreshortened future, that fills out the adult negative-cognition cluster, so the classification asks only for the observable behavioural change.

FEATURE	OLDER THAN SIX YEARS	SIX YEARS AND YOUNGER (PRESCHOOL SUBTYPE)
Exposure (A)	Includes learning of events to any close person, and repeated exposure to aversive details	Direct experience, witnessing in person (especially to caregivers), or learning it happened to a parent or caregiving figure
Intrusion (B)	Reexperiencing, often reportable	One or more; often expressed as play re-enactment, not overt distress
Avoidance and negative cognition	Two separate clusters	Merged into a single Criterion C requiring one symptom
Arousal and reactivity (D)	Full cluster	Two or more symptoms, tantrums count as irritability
Number of symptom clusters	Four	Three
Duration	More than one month	More than one month

The preschool subtype set against the version used for everyone older; the changes all move in one direction, lowering and re-shaping the requirements to fit what a developmentally young child can show.

TRAP **Counting a preschooler against the four-cluster adult set.** Candidates apply the older-than-six structure to a young child, look for two separate avoidance and negative-cognition clusters and the full adult symptom counts, find them absent, and conclude there is no disorder. The preschool child is diagnosed on three clusters with lower thresholds, so the adult arithmetic wrongly excludes a genuine case.

17.5 Criterion D: arousal, and the tantrum as a symptom

Hyperarousal in a young child wears the face of behaviour problems. The **arousal and reactivity** cluster requires two or more symptoms, and here the developmental translation matters as much as the count. Irritable behaviour and angry outbursts may be expressed as extreme temper tantrums, and this is precisely where a traumatised preschooler is misread as merely oppositional or as having a behavioural disorder. Alongside the tantrums the cluster includes hypervigilance, an exaggerated startle response, problems with concentration, and disturbed sleep. The reason this cluster is a trap in daily practice is that every one of its manifestations, the tantrums, the poor attention, the broken sleep, is nonspecific and common in early childhood, so the arousal cluster is only informative when it is new, when it clusters with the other criteria, and when it dates from an identifiable trauma.

PITFALL

The commonest clinical failure is to stop at the presenting behaviour. A young child brought in for new tantrums, defiance, poor sleep and clinginess is labelled with a behavioural or oppositional problem, medicated or sent for behaviour management, and never asked what happened to them. Because young children do not spontaneously report trauma and may not even have shown fear at the time of the event, the history has to be sought actively from caregivers; without that question, the arousal cluster reads as naughtiness and the diagnosis is missed.

17.6 Duration, delayed expression and the developmental picture

The clock is the same, but the symptoms may take time to declare themselves. The duration requirement does not change with age: symptoms must persist for **more than one month** before the diagnosis is made, which separates the disorder from the expected, self-limiting distress of the first weeks after a frightening event. Where the full criteria are not met until **at least six months** after the event, the delayed-expression specifier applies, a pattern that is clinically real in children whose symptoms crystallise only as development proceeds. The wider developmental picture rounds out the assessment: before the age of six, children more often express reexperiencing through play that refers to the trauma directly or symbolically, may develop new frightening dreams without trauma-specific content, and may show developmental regression such as loss of previously acquired language. Young children may not have shown a fearful reaction at the moment of exposure at all, and their negative alterations tend to be changes in mood rather than the articulated guilt or self-blame of an older patient, simply because the capacity to label and attribute emotion is still forming.

- reexperiencing enacted in repetitive, trauma-referring play rather than described;
- new frightening dreams whose content cannot be tied to the event;
- developmental regression, including loss of language or of toileting skills;
- mood change and withdrawal in place of verbalised negative beliefs.

> In a child of six or younger, diagnose on the modified set: three symptom clusters not four, one intrusion symptom, one merged avoidance-or-negative-cognition symptom, two arousal symptoms, persisting for more than one month.

17.7 Assessment, management and the Indian setting

The diagnosis is only useful if it changes what happens next. Assessment leans heavily on caregiver report and on structured observation of the child's play, because the child is not a reliable narrator of internal states. The clinician gathers a clear trauma history, maps the current symptoms onto the developmental criteria, and screens the caregiving environment, since ongoing threat or a distressed, unavailable caregiver both perpetuates the disorder and limits any treatment. The first-line treatment is a **trauma-focused psychotherapy**, delivered in a developmentally graded and caregiver-inclusive form; the general evidence base and the pharmacological questions for post-traumatic stress disorder belong to the Anxiety, OCD and trauma-related disorders notes and are not restated here. Where the trauma is abuse, neglect or exposure to violence, safeguarding and the statutory child-protection response are inseparable from treatment, and the machinery for that is set out in the Child psychiatry: assessment, treatment, services and protection notes.

GOOD TO REMEMBER

Interview the caregiver as your primary instrument and watch the child's play as your second. Ask specifically about new fears, sleep, tantrums, clinginess, regression and any change dated to a frightening event, because none of these will be offered spontaneously by a young child. A single well-placed question about what happened often converts a vague "behaviour problem" referral into a treatable, developmentally-typed post-traumatic presentation.

IN INDIA

Young children in Indian practice carry a heavy and often unspoken trauma load, from road-traffic and burn injuries, natural disasters, domestic and community violence, and physical or sexual abuse, yet the preschool presentation is routinely read as a phase or a discipline problem and reaches services late, if at all. Most public services classify under ICD rather than DSM, and access to a trained trauma-focused child therapist is scarce outside a few centres, so the practical yield of this topic is recognition: asking the trauma question of a regressed or newly tantrumming young child, and routing genuine cases into whatever child mental-health and protection services exist locally.

6 and younger	3 clusters	over 1 month	6 months
AGE FOR THE PRESCHOOL SUBTYPE	PRESCHOOL SET, NOT FOUR	REQUIRED SYMPTOM DURATION	DELAYED-EXPRESSION SPECIFIER

Childhood mood disorders, disruptive mood dysregulation, early-onset psychosis and paediatric suicidality

18 · Childhood depression (presentation, masked depression, management)

The whole difficulty of the topic is that the illness rarely announces itself the way it does in an adult. **Childhood depression** is the same major depressive syndrome recognised in later life, but its surface presentation is bent by development: a young child cannot always name low mood, is more likely to show it as irritability or bodily complaint, and depends on the adults around to notice that something has changed. This is why the marks cluster where they do. Candidates who can recite the adult symptom list still lose ground on the two questions that actually decide the diagnosis in a child, which is how the presentation shifts with age, and how a depressive disorder is told apart from the disruptive and behavioural pictures it can hide behind.

This topic owns the developmental presentation of the disorder - the irritable-mood variant, the age-dependent symptom expression, and the older idea of a depression that is masked behind other complaints - together with the assessment instruments used to detect and track it and the shared minimal duties of its management. The general neurobiology of depression, the detailed suicide-risk tools, the paediatric antidepressant-suicidality warning, disruptive mood dysregulation disorder and the somatic presentation of childhood distress are each developed in their own right elsewhere; here they are named where the reasoning demands it, not reopened.

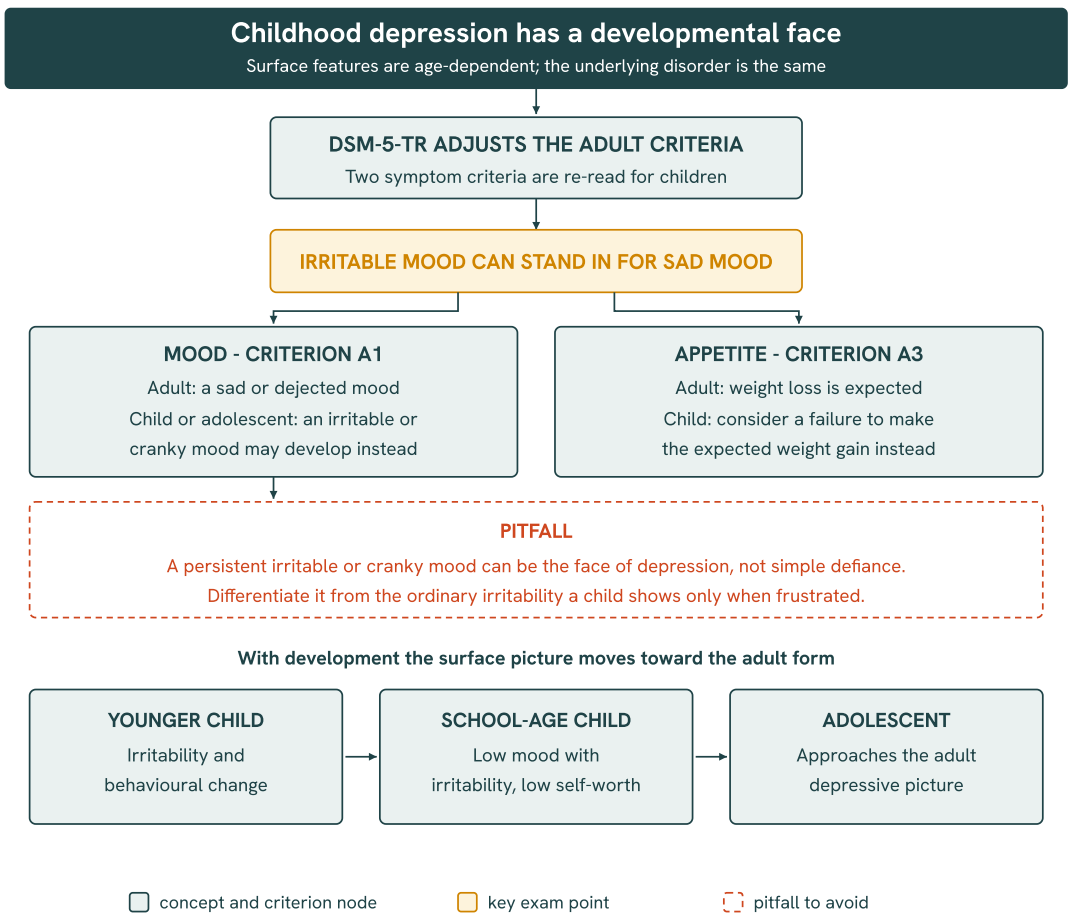

Fig 23.12. Childhood depression, developmental presentation: irritable mood and age-adjusted criteria as the picture shifts toward the adult form with development.

18.1 Why the disorder is missed: development conceals the mood

The core clinical problem is under-recognition, and it is a developmental problem before it is a diagnostic one. Depression in children was for a long time thought not to exist, on the reasoning that an immature personality could not sustain the sense of guilt and hopelessness that defines the adult illness. That view has been abandoned - the disorder is real, impairing and recurrent in the young - but the reason it was ever plausible remains clinically live. A child's capacity to identify and report an internal state is limited, so the depression tends to be inferred from what the adults around the child observe rather than declared by the child. Pervasive **anhedonia**, a loss of the ordinary pleasure in play, friends and activities, is often the most reliable observable sign, precisely because it does not depend on the child being able to articulate sadness. The practical consequence is that the assessor must actively look for the disorder in a child whose difficulty is showing up as falling grades, withdrawal, somatic complaint or a change in temper, rather than wait for a spontaneous complaint of low mood that a young child may never produce.

18.2 Irritability as the depressed mood

The single most examinable adaptation of the criteria is that irritability can stand in for sadness. The diagnostic rules for a major depressive episode carry an explicit developmental

note: **in children and adolescents the depressed-mood criterion can be satisfied by an irritable mood** rather than by a subjectively sad or dejected one. This is not a softening of the threshold but a recognition that the same underlying mood disturbance surfaces differently earlier in life, so that a young person who is persistently cranky, touchy and easily provoked may be depressed even though they never say they are sad. The clinical instruction that follows is a discrimination, not a relabelling: an irritable or cranky mood may develop in place of a sad or dejected one, and it must be differentiated from the ordinary irritability a child shows when frustrated. What converts irritability into a depressive sign is its pervasiveness and persistence, its company with anhedonia, sleep and appetite change and a drop in functioning, and its independence from a specific frustrating trigger. The irritability of the depressed child is a background weather, not a reaction to being told no.

RULE **In a child, an irritable mood can meet the depressed-mood criterion in place of sadness.** The examiner is testing whether the candidate knows that a persistently irritable young person can be experiencing a major depressive episode, and can separate that sustained, pervasive irritability from the situational crossness of a frustrated child. Miss the developmental note and the whole diagnosis is missed with it.

18.3 The age-graded shift in symptoms

The same disorder wears different clothes at different ages. Beyond the mood itself, the neurovegetative criteria are read differently in a growing child. Where an adult is expected to lose weight, the criteria direct that in a child one should instead consider a failure to make the expected weight gain: a still-growing child who is depressed may not lose weight but may fall off their expected growth trajectory, so the clinician reads the growth chart rather than the scale reading alone. More broadly, the presenting complaint tends to track development. Younger children more often present with somatic distress, clinginess, irritability and a flattening of play; adolescents move closer to the adult picture of low mood, hopelessness, guilt and articulated suicidal thinking, but with a heavier loading of irritability, interpersonal sensitivity and behavioural change than an adult would show. The recurrent bodily complaints - the aching stomach, the headache with no organic cause - are a genuine and frequent vehicle for depressive and anxious distress in the young, but that somatic presentation is developed in its own right elsewhere in this chapter and is only named here as one of the forms the disorder can take.

FEATURE	YOUNGER CHILD	ADOLESCENT
Predominant mood surface	Irritability, crankiness	Low mood and irritability, closer to the adult picture
Appetite and weight criterion	Failure to make expected weight gain	Weight loss or gain, as in adults
How distress is voiced	Somatic complaints, clinginess, loss of play	Hopelessness, guilt, articulated suicidal thoughts
Chief route to detection	Adult observation of change	Self-report becomes more available

The developmental presentation set out age against age; the load-bearing points are that irritability can be the mood and that a failure to gain expected weight can replace weight loss in the still-growing child.

Carry two developmental substitutions into the exam: in children and adolescents the depressed-mood criterion can be met by irritable mood, and the appetite criterion by a failure to make expected weight gain.

18.4 Masked depression: the concept and its careful reading

Masked depression is the older name for a depression whose mood is hidden behind a non-mood complaint. The term **masked depression** was coined for presentations in which the depressive affect is not the presenting feature at all, and the disorder instead shows itself through what were called **depressive equivalents**: recurrent somatic complaints, school refusal, a fall in academic performance, aggression, or in older children substance use and antisocial behaviour. In a child, for the developmental reasons already given, the depression is more often masked than in an adult, and the idea earns its keep as a reminder to hold depression in mind when the complaint on the referral letter is behavioural or physical. It should nonetheless be read critically. The concept is imprecise and historically over-extended, and the safer modern position is not that any behavioural disturbance is a hidden depression, but that a depressive disorder must be actively excluded when a child presents with an unexplained change in conduct, function or bodily state. The mask is a prompt to look underneath, not a licence to diagnose depression behind every difficult behaviour.

PITFALL

Diagnosing a child from the child's self-report alone. Depressed children, especially younger ones, under-report their own symptoms and may deny sadness outright, while a parent or teacher sees the irritability, withdrawal and functional decline plainly. A single-informant assessment built on the child's account can therefore return a falsely reassuring picture; the diagnosis in this age group is a multi-informant one, and the account of the adults who see the child across settings is not optional.

18.5 Detecting and tracking it: the rating scales

Because the disorder is inferred rather than declared, a structured instrument earns its place. Rating scales do not make the diagnosis, which remains clinical, but they standardise detection, quantify severity and let the clinician track change over treatment. The best known in this age group is the **Children's Depression Inventory**, a **27-item** self-report scale built as a downward extension of the adult Beck inventory for younger respondents. The original version resolved into five subscales - dysphoric mood, acting out, loss of personal and social interest, self-deprecation and vegetative symptoms - though the total score is what is generally used, and it is brief enough to complete in a single short sitting. Its structure tells you what a good paediatric depression scale must do: sample mood, the behavioural surface, anhedonia, self-worth and the neurovegetative signs together, because any one of these alone can be the way the disorder shows.

The current second edition is designed as a **multi-informant** instrument, pairing the child's self-report with parent and teacher forms, which is exactly the correction the single-informant pitfall

demands. It remains modelled on the Beck Depression Inventory, is a widely used self-report measure that includes an item on suicidality and is sensitive to treatment effects, so it doubles as an outcome measure across a course of care; its limitation is that results are mixed for distinguishing depression from other disorders, so it screens and monitors but does not adjudicate the diagnosis. It is applicable across the school-age and adolescent years, in the region of 7 to 17 years depending on the source consulted, and being a proprietary instrument it must be purchased for use.

GOOD TO REMEMBER

Choose the scale for the job. A self-report inventory with a suicidality item and known sensitivity to change is well suited to tracking a known case across treatment and to prompting a suicide conversation, but a scale that does not reliably separate depression from other disorders cannot be used to confirm the diagnosis. Score it, act on the suicidality item at once, and re-score it to judge response - but let the clinical formulation, not the number, make the diagnosis.

18.6 Management: severity decides the first move

Management is stepped to severity, and the first line for a mild presentation is deliberately not a drug. The organising principle is that treatment intensity should match illness severity, and the sharpest examinable edge is at the mild end. For **mild depression the first-line approach is supportive care or a psychological intervention, and an antidepressant should not be prescribed**. The reasoning is twofold: mild childhood depression has a substantial rate of spontaneous improvement and response to non-drug support, so a period of active watchful support with a psychological therapy captures many recoveries without exposing a child to medication; and the risk-benefit balance of antidepressants in the young is only favourable once the illness is severe enough to warrant it. As the picture moves to moderate or severe, a structured psychological therapy is offered either alone or in combination with an antidepressant. Involving the family and the school is not an optional extra but part of the treatment itself, because the child's environment carries much of the maintenance and much of the recovery.

- mild depression: supportive care and a psychological therapy first-line, with medication actively withheld;
- moderate to severe depression: a psychological therapy alone or combined with an antidepressant;
- family and school engaged throughout as part of the intervention, not as an afterthought;
- severity, suicide risk and comorbidity reviewed at the outset and again as treatment proceeds.

18.7 Pharmacotherapy: which drug, what dose, what evidence

When an antidepressant is indicated, the choice, the dose and the evidence behind it are all examinable. The first-line agent is **fluoxetine**, and the reason it holds that place is specific rather than generic: it is the one antidepressant for which the trial evidence shows benefits outweighing risks in this age group, and it is approved for use from **age 8**, with escitalopram approved from age 12 as the other option. Dosing is cautious and stepwise: start at 10 mg daily, increase after

one week to 20 mg daily, with a usual range of 20 to 60 mg daily, the lower end being the minimum therapeutic dose and the higher end reserved for the older or higher-weight child or where an early response is a priority. The general pharmacology of the antidepressants belongs to the Mood disorders: pharmacotherapy notes and is not reopened here; what is childhood-specific is that fluoxetine is chosen first on evidence, and that the youth-specific antidepressant-suicidality question and its monitoring schedule are developed in their own right elsewhere in this chapter.

The evidence that anchors the choice is worth knowing as a figure. The Treatment of Adolescents with Depression Study, which compared fluoxetine, cognitive behavioural therapy, their combination and placebo in adolescents with major depression, found over its acute phase a response of **61 percent** to fluoxetine against 35 percent to placebo, a number needed to treat of 4. That separation is the quantitative backbone of the recommendation, and the trial is also the source most often cited for the combined-treatment finding that adding a psychological therapy to medication does best of all, though the combination figures are not reproduced here.

IN INDIA

Fluoxetine is inexpensive, off-patent and among the most widely available antidepressants in the country, which makes the evidence-based first choice also the pragmatic one in a resource-limited service. The greater Indian constraint is not the drug but access to the psychological therapies that should precede and accompany it: specialist child mental health services are concentrated in a few centres, so much childhood depression is first seen in general adult psychiatry, paediatrics or primary care. The stepped principle still holds - a mild presentation is managed with support and psychological input, not a prescription - but its delivery leans heavily on family work and the school, and on task-shared counselling under the national mental health programme, where a full child psychiatry team is out of reach.

18.8 The safety floor every case carries

Whatever the severity, childhood depression management carries a minimum safety duty. Depression is the commonest diagnosis behind youth suicide, so every assessment of a depressed child carries a baseline screen for suicidality and early, close monitoring, and when an antidepressant is started the risk of harm must be carefully monitored in the early weeks. This is the shared minimal duty owned by the management of the disorder itself. It deliberately stops short of the detailed content that sits elsewhere: the full risk-assessment and safety-planning tools are developed in their own right elsewhere in this chapter, as is the paediatric antidepressant-suicidality warning with its absolute-risk figures and its structured early-monitoring schedule. The point to hold here is the floor, not the detail - that a suicide screen and early monitoring are not add-ons to be remembered for severe cases but the baseline of every depressed child's care, and that starting a drug raises rather than removes the monitoring obligation.

18.9 Course, comorbidity and the differential that catches candidates

Getting the diagnosis right means getting the boundaries right. Childhood depression is recurrent, carries forward into adult mood disorder in a substantial proportion, and rarely travels alone: anxiety disorders, the disruptive behaviour disorders and attention problems are frequent companions, and comorbidity both worsens the outlook and complicates treatment. The general neurobiology and life-course of mood disorder belong to the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; what is examinable here is the local differential. The chronic, non-episodic irritability that in a child can look like depression is also the territory of a separate chronic-irritability diagnosis, disruptive mood dysregulation disorder, which is developed in its own right elsewhere in this chapter and must be distinguished from a discrete depressive episode with an irritable mood. The behavioural surface of a masked depression can equally be read as a purely disruptive disorder. The discipline in every case is the same: establish whether a genuine, pervasive and persistent change in mood and functioning underlies the presenting behaviour, because that change is what separates a mood disorder from the conduct and irritability syndromes it can imitate.

TRAP **Reading an irritable, declining child as "just behavioural" and stopping there.** The classic error is to take the irritability, the falling grades or the acting-out at face value as an oppositional or conduct problem and never screen for the depression underneath. The developmental note that irritability can be the depressed mood exists precisely to stop this: a persistent, pervasive irritability with anhedonia and functional decline is a reason to assess for a depressive disorder, not a reason to close the file on a behavioural label.

10 mg	**20-60 mg**	**61% vs 35%**	**4**
FLUOXETINE STARTING DOSE, DAILY	USUAL DAILY RANGE	TADS RESPONSE, FLUOXETINE VS PLACEBO	NUMBER NEEDED TO TREAT

19 · Somatic symptom, conversion and pain disorders in children

A child in distress often has no words for it, so the distress finds a body to speak through. The recurring tummy ache before school, the morning headache that lifts by lunchtime, the exhaustion no blood test explains: these are among the commonest ways emotional trouble reaches a paediatrician, and they are examinable because a candidate must do two quite different things well. The first is to recognise ordinary somatisation, the healthy child whose anxiety or low mood is being felt in the gut rather than named as a feeling. The second is to recognise the two formal disorders that sit beyond it, somatic symptom disorder and conversion disorder, and to grade and reason about them without falling into the traps that have been deliberately built into the modern criteria. The general adult forms of these conditions, their full nosology and their treatment, are developed in the Somatic-symptom, sexual, impulse-control disorders and suicide notes; this topic owns the child-specific presentation and teaches the two disorders in their own right.

The order to hold runs from the softest to the hardest edge: first the somatisation of distress, a presentation and not yet a disorder; then somatic symptom disorder, defined by how the child and family respond to symptoms rather than by whether those symptoms are explained; then

conversion disorder, which turns on a positive neurological sign and is not a diagnosis reached by exclusion.

19.1 The body as the voice of distress

Before any disorder is diagnosed, know how ordinary childhood distress somatises. When children are anxious or depressed, the complaint that reaches the clinic is frequently physical. The characteristic symptoms of childhood **somatisation** are recurrent abdominal pain, headache, fatigue and nausea, and a useful developmental point is that a single prominent symptom is commoner in a child than the elaborate multi-system picture more typical of an adult. This is the presentation that separation anxiety, school refusal and childhood depression each borrow when they surface in the body rather than in mood or thought; those conditions are taught in full in their own places in this chapter, and here the point is only that the abdominal pain or the headache is the surface, and the affect underneath it is what the interview must reach.

19.2 Recurrent abdominal pain as the paradigm

One symptom carries most of the marks here. Recurrent abdominal pain is one of the most common medical complaints of childhood, and in the majority of children who present with it no organic pathology is ever identified. That single fact does most of the clinical work. It tells you that a careful history and examination, rather than a widening battery of tests, is the right first move, and that in a child with recurrent pain, normal growth and no alarm features, an emotional contributor is statistically the likeliest story. The pain is real, felt exactly where the child points; it is the meaning, not the sensation, that is misattributed. The discipline is to hold two truths together: most such pain is functional, and yet the minority with organic disease must not be missed, which is why red-flag features such as weight loss, nocturnal waking, bleeding or a localising sign always earn investigation regardless of how anxious the child seems.

> **PITFALL**
>
> The repeated normal investigation can itself become the disease. Each fresh scan that comes back clear is meant to reassure, but for an anxious child and a frightened family it often does the opposite: it confirms that something serious is being hunted for and has not yet been found. The result is escalating investigation, school absence and an entrenched sick role. Deciding early that the picture is functional, and saying so with conviction while keeping a light safety net, protects the child better than another test.

19.3 Somatic symptom disorder: the DSM-5 entity

Somatic symptom disorder is a formal diagnosis, distinct from the presentation above. Its architecture is worth carrying exactly. **Somatic symptom disorder** requires one or more somatic symptoms that are distressing or that cause significant disruption of daily life. The defining requirement, however, is not the symptom but the reaction to it: there must be **excessive thoughts, feelings or behaviours** about the symptoms, demonstrated by at least **one** of three features.

- disproportionate and persistent thoughts about the seriousness of the symptoms;

- a persistently high level of anxiety about health or about the symptoms;
- excessive time and energy devoted to the symptoms or to health concerns.

The symptomatic state must be persistent, typically lasting **more than six months**, although any one symptom need not be continuously present across that time. Severity is graded by how many of the three response features are met, which keeps the grading anchored to the psychological burden rather than to the count of bodily complaints.

MNEMONIC

Symptom, Story, Staying-power

A distressing bodily **symptom** (Criterion A); the excessive **story** the child and family build around it, in thought, health anxiety or time consumed (Criterion B); and the **staying-power** of a persistent symptomatic state (Criterion C). The middle term is the diagnosis.

19.4 The pivot away from medically unexplained symptoms

This is the single most tested conceptual point. The older somatoform category was built on the idea that the symptoms were medically unexplained. DSM-5 abandoned that foundation. Having **medically unexplained symptoms** is no longer sufficient to make the diagnosis; the diagnosis rests on the excessive thoughts, feelings and behaviours, and the somatic symptoms may or may not accompany a diagnosable medical condition. A child with genuine asthma or migraine can also have somatic symptom disorder if the response to those real symptoms is disproportionate and disabling. This reframing stops the diagnosis from being a label of last resort applied when the tests come back normal, and it protects the truth that the child's suffering is authentic whether or not medicine can explain the sensation.

RULE **Somatic symptom disorder is diagnosed by the response, not by the absence of a cause.** Ask not whether the symptom is explained but whether the thoughts, anxiety and behaviour around it are out of proportion and are consuming the child's life. A real diagnosis can sit alongside a real disease.

19.5 Applying the criteria to children

The adult criteria transfer, but the assessment does not. The somatic symptom disorder criteria appear suitable for use in children and adolescents, though the entity is far less studied in the young than in adults. The practical consequence is that a child cannot be assessed alone. Distress in childhood is co-authored: how a parent interprets a stomach ache, whether it is met with alarm or with calm, shapes how the child comes to experience and report it, and the same symptom can be trivial in one household and disabling in another. Assessment therefore gathers the child's account, the family's, and the school's, because the pattern of missed lessons and the child's function among peers reveals disability the consulting room cannot. The full child psychiatric assessment battery and its services are set out in the Child psychiatry: assessment, treatment, services and protection notes.

19.6 Conversion disorder and the incompatibility criterion

Conversion disorder is a different animal, defined by a positive examination. In **functional neurological symptom disorder**, also called conversion disorder, the presenting problem is altered voluntary motor or sensory function: a weak or paralysed limb, an abnormal gait, non-epileptic attacks that resemble seizures, or a sensory loss. The pivotal requirement is that the clinical findings provide evidence of incompatibility between the symptom and recognised neurological or medical conditions. Incompatibility is a specific and technical idea: it means the signs are internally inconsistent, behaving in a way no organic lesion could produce, rather than simply being unexplained or unusual. That internal inconsistency, demonstrated at the bedside, is what carries the diagnosis, and it is why the neurological examination in a suspected conversion disorder is an active search for positive signs rather than a screen for disease to rule out.

19.7 A rule-in, not a rule-out, diagnosis

Conversion disorder is not made because the scans are clean. This deserves its own place because getting it wrong is the classic failure. The diagnosis is not one of exclusion; it is made on positive clinical signs of incompatibility. The examples worth knowing are **Hoover's sign**, in which apparent leg weakness normalises when the opposite hip is flexed against resistance, and the tremor entrainment test, in which a functional tremor takes on the rhythm the examiner asks the other hand to tap. Because these signs demonstrate the symptom directly, conversion disorder can coexist with genuine neurological disease, and it must never be diagnosed merely because investigations are normal or because a symptom looks bizarre. A symptom that is odd is not thereby functional, and a normal scan proves the absence of the lesions it images, not the presence of conversion.

TRAP **Treating conversion disorder as a diagnosis of exclusion.** The tempting shortcut is to order the investigations, watch them return normal, and conclude by elimination that the paralysis or the non-epileptic attack must be psychological. That reasoning is unsafe in both directions: it misses the organic disease the tests did not cover, and it fails to demonstrate the positive incompatibility the diagnosis actually requires. Show the sign, do not merely fail to find a lesion.

19.8 Telling the three apart

The examinable discrimination is between three near neighbours. The somatisation of distress, somatic symptom disorder and conversion disorder are related but distinct, and a candidate is expected to separate them cleanly. The sharpest line runs between the two formal disorders: in **functional neurological symptom disorder the presenting problem is a loss of function**, of a limb or a sense, whereas in somatic symptom disorder the focus is the distress that the symptoms cause and the disproportionate response to them. The somatisation presentation sits before both, describing how childhood anxiety and depression speak through the body without necessarily reaching the threshold of either disorder. Persistent pain, once carved out as a separate pain disorder, is now captured within somatic symptom disorder when the response to it is excessive. For the general adult versions of all of these, and their management, the general somatic-symptom and conversion disorders are taught in the Somatic-symptom, sexual, impulse-control disorders and suicide notes.

FEATURE	SOMATISATION OF DISTRESS	SOMATIC SYMPTOM DISORDER	CONVERSION DISORDER
Status	A presentation, not a disorder	DSM-5 disorder	DSM-5 disorder
Core focus	Anxiety or low mood felt in the body	The distress and excessive response to the symptoms	A loss of voluntary motor or sensory function
Typical symptom	Recurrent abdominal pain, headache	Any distressing bodily symptom, explained or not	Weakness, abnormal gait, non-epileptic attacks
What secures it	Reaching the affect beneath the complaint	The excessive thoughts, feelings and behaviours	A positive sign of clinical incompatibility
Cardinal trap	Dismissing real suffering as fabrication	Reading it as merely "medically unexplained"	Diagnosing by exclusion

The three run from a presentation towards two disorders defined on opposite grounds: somatic symptom disorder by the response to a symptom, conversion disorder by a positive sign that the symptom is incompatible with disease.

GOOD TO REMEMBER

What changes practice on Monday is the stance, not a new test. Validate the symptom as genuinely felt, name the emotional contributor without implying the child is inventing anything, set a firm ceiling on investigation, and enlist the family and school in a graded return to normal function. For a suspected conversion presentation, gently demonstrate the incompatibility sign to the family, because a diagnosis they can see explained is one they can work with.

IN INDIA

Somatic presentation is arguably the default idiom of distress across much of Indian practice, where emotional vocabulary is often somatic and the first contact is a physician or paediatrician rather than a mental health clinician. Conversion presentations, including non-epileptic attacks framed by families in idioms of possession or spiritual affliction, are seen frequently in Indian child and adolescent services. Two habits follow. Screen for anxiety and depression behind the persistent tummy ache or headache rather than reaching only for another investigation, and, in a country where organic infective and nutritional disease is common, keep the safety net for red-flag features firmly in place even once a functional cause looks likely.

Somatic symptom disorder is diagnosed by the excessive response to symptoms, not by their being unexplained; conversion disorder is ruled in by a positive sign of incompatibility, never ruled in by exclusion.

1 or more DISTRESSING SOMATIC SYMPTOMS, CRITERION A	**at least 1 of 3** EXCESSIVE-RESPONSE FEATURES, CRITERION B	**over 6 months** PERSISTENT SYMPTOMATIC STATE, CRITERION C	**the majority** CHILDHOOD RECURRENT ABDOMINAL PAIN WITH NO ORGANIC CAUSE

20 · Disruptive mood dysregulation disorder

The diagnosis was built to solve a problem the field had created for itself. **Disruptive mood dysregulation disorder** names the child who is chronically, grindingly irritable and who erupts into severe temper outbursts many times a week, every week, for a year or more. It exists because a generation of chronically irritable children had been swept into a diagnosis of paediatric bipolar disorder on the strength of their rages, treated with mood stabilisers and antipsychotics, and followed into an adulthood that turned out to hold not mania but depression and anxiety. The construct was introduced to give that presentation a home of its own, in the depressive rather than the bipolar family, and to stop the reflex that read every explosive child as bipolar. For the examination the marks sit almost entirely on two things: the exact criteria, which are long and easily half-remembered, and the reasoning that separates this chronic, non-episodic irritability from the episodic mood elevation of bipolar disorder.

This topic owns the diagnostic architecture of the disorder and the operational rule that distinguishes it from paediatric bipolar disorder: the temper-outburst criterion, the frequency and inter-outburst mood requirements, the duration, pervasiveness, age-window and onset rules, the manic-exclusion clause, its placement among the depressive disorders, its differential against oppositional defiant disorder, its prevalence, its position in the two major classifications, and its indication-scoped pharmacotherapy. The historical over-diagnosis debate itself, the narrow-phenotype argument and the reasons the category was contested are developed in their own right elsewhere in this chapter; here they are named as the backdrop, not reopened.

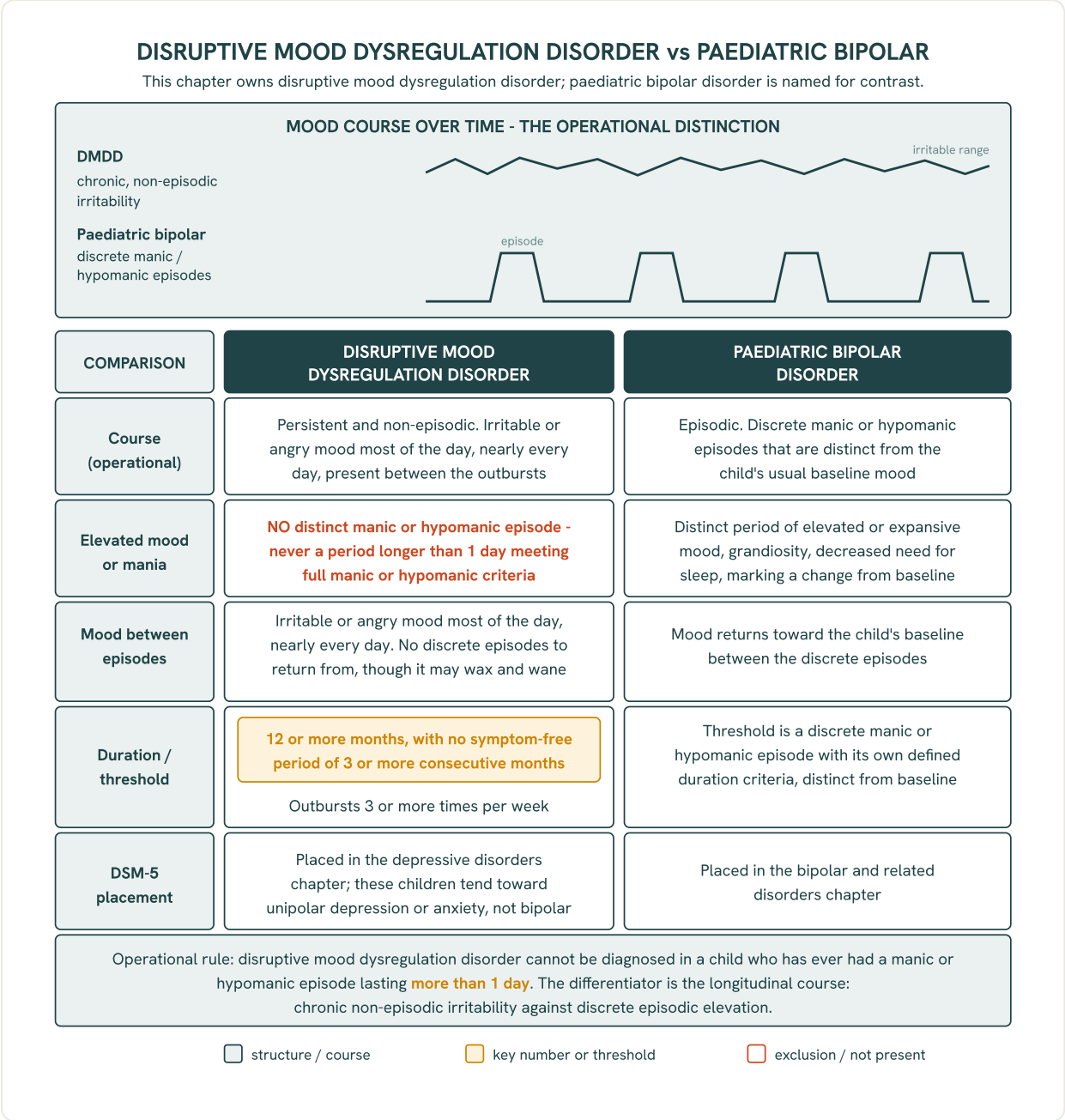

Fig 23.13. Disruptive mood dysregulation disorder against paediatric bipolar disorder: a mood-course timeline and a paired criteria grid showing chronic non-episodic irritability against discrete episodic mania, the 12-month duration floor, the manic-episode exclusion boundary of more than 1 day, and the depressive-chapter placement that carries the operational distinction.

20.1 What the disorder is, and why it sits among the depressive disorders

The placement is the argument in miniature. The classification files this disorder among the **depressive disorders**, and that decision carries the whole rationale for the category. Longitudinal work found that children with chronic, non-episodic irritability do not, as a rule, grow into adults with bipolar disorder; they grow into adults with unipolar depressive and anxiety disorders. The irritability is therefore read as a depressive-spectrum phenomenon expressed in a developmentally immature way, not as a prodrome of mania, and the disorder is housed accordingly. This is worth holding onto because it explains everything downstream: why the manic exclusion is written into the criteria, why lithium is not the answer, and why the differential that matters is not against conduct problems but against bipolar disorder. The name

itself is a compromise, splitting the presentation into two visible components - the **persistently irritable or angry mood** that forms the baseline, and the severe outbursts that punctuate it.

20.2 Criterion A: the temper outburst as the core symptom

The disorder is anchored on outbursts that are grossly disproportionate to their trigger. The core criterion is **severe recurrent temper outbursts**, which may be verbal, behavioural, or both, and which are grossly out of proportion in intensity or duration to the situation or provocation that set them off. In practice this is the verbal rage - screaming, shouting - and the physical aggression toward people or property that families describe as coming out of nowhere and lasting far longer than the small frustration that triggered it. Two qualities do the diagnostic work. The first is disproportion: the reaction is wildly larger than the provocation, so a minor "no" produces a twenty-minute destructive storm. The second is that the outbursts are inconsistent with developmental level; the tantrum of a two-year-old denied a sweet is expected, the same storm in a school-aged child is not. It is the pairing of severity, disproportion and developmental inappropriateness, not the mere fact of a tantrum, that makes an outburst count.

MNEMONIC

Storms on a stormy sky

The whole picture is two components at once: the **storms** are the severe temper outbursts, and the **stormy sky** is the persistently irritable, angry mood that persists between them. A child who has the storms but a calm sky between them does not have this disorder, and neither does the child who is merely irritable without the outbursts. Requiring both is the single feature that keeps the category tight.

20.3 Frequency and the mood between outbursts: the two thresholds that define the syndrome

An outburst threshold and a baseline-mood threshold must both be crossed. The frequency rule requires the outbursts to occur, on average, at least **three** times a week. This is a demanding bar and it is deliberate: it filters out the child who erupts occasionally under stress and captures only the pattern of repeated weekly explosiveness. The second threshold is the mood between the storms. The criteria require that, in the intervals between outbursts, the mood is persistently irritable or angry, present most of the day, nearly every day, and - crucially - observable by others such as parents, teachers and peers. This inter-outburst irritability is what separates the disorder from a simple explosive-behaviour problem. The child is not calm and pleasant between rages; the baseline itself is angry. Requiring the irritability to be observable by others, rather than merely reported, guards against a diagnosis built on a single exhausted parent's account, and it is a frequently examined clause.

RULE **Both the outbursts and the mood between them must be abnormal.** The defining structure of this disorder is a persistently irritable or angry baseline punctuated by severe, frequent temper outbursts. The outbursts alone are not enough, and the irritable baseline alone is not enough; the diagnosis lives precisely in the requirement that both be present, together, over a long stretch of time. This is what makes the irritability chronic rather than episodic, and chronicity is the whole point.

20.4 Duration, pervasiveness and the age rules

Four gating rules turn a cross-sectional picture into a diagnosis. The criteria then wrap the two core thresholds in a set of qualifying rules whose job is to enforce chronicity and to keep the diagnosis inside a defined developmental band. The symptoms must have been present for at least **twelve** months, with no symptom-free stretch of three or more consecutive months, so a transient irritable phase cannot qualify. They must be present in at least two of three settings - home, school and with peers - and be severe in at least one, which enforces the pervasiveness that separates a disorder from a reaction to one difficult environment. The diagnosis must not be made for the first time before age 6 or after age 18, because the outburst-plus-irritability picture is neither reliable nor distinctive at the extremes of that range. And by history or observation the onset of the symptoms must be before **age 10**. Together these rules are what make this a disorder of chronic, pervasive, early-onset irritability rather than a label for any child who is having a bad month.

REQUIREMENT	THE RULE	WHAT IT GUARDS AGAINST
Outbursts	Severe, disproportionate, at least three per week on average	The occasional stress-driven eruption
Baseline mood	Persistently irritable or angry between outbursts, observable by others	Explosive behaviour without a mood disturbance
Duration	Present at least twelve months, no gap of three or more months	A transient irritable phase
Pervasiveness	In at least two of three settings, severe in one	A reaction confined to one setting
Age window	Not diagnosed before age 6 or after age 18	Unreliable diagnosis at the developmental extremes
Onset	Symptoms begin before age 10	An adolescent-onset mood or behavioural disorder in disguise

The gating rules read against the failure mode each one is designed to exclude; the accompanying figure lays the same structure out as the two core thresholds wrapped in the chronicity and age constraints.

20.5 The manic exclusion and the chronic-versus-episodic distinction

This is the reasoning the whole category was built to force. The criteria carry an explicit exclusion: a child who has ever had a manic or hypomanic episode - a distinct period meeting the full symptom criteria, except duration - is, by definition, on the bipolar spectrum and cannot receive this diagnosis. The load-bearing distinction is longitudinal and it is the single most examined point in the topic: the irritability of this disorder is **persistent and non-episodic**, a chronic background state that may wax and wane without ever resolving into episodes, whereas bipolar disorder is **episodic**, defined by discrete manic or hypomanic episodes that stand out clearly from the child's usual baseline. You are not comparing two cross-sectional pictures; you are comparing a flat, chronically irritable course against a course marked by episodes with a beginning and an end. The bipolar controversy in children - the historical over-diagnosis, the

narrow-phenotype debate - is taken up in its own right elsewhere in this chapter; the operational discriminators the clinician actually applies are these:

- a chronic, non-episodic irritable baseline, present most of the day and nearly every day though it may wax and wane, favours this disorder; irritability that arrives and departs in discrete spells favours bipolar disorder;
- any distinct period, longer than one day, that met full manic or hypomanic criteria bars this diagnosis outright;
- a developmentally appropriate surge of high spirits at a genuinely exciting event is not mania and does not count toward it.

TRAP **Chronic severe irritability is not paediatric mania.** The classic error, and the very error the diagnosis was created to correct, is to see a child with frequent explosive rages and a short fuse and reach for bipolar disorder. Irritability is not episode-defining; it is the non-specific affect this disorder makes chronic. The question is never how angry the child is but whether the anger comes in discrete episodes distinct from baseline. If it does not, this is chronic irritability, and the manic-exclusion clause bars the bipolar label outright.

20.6 The differential against oppositional defiant disorder

The two overlap heavily, and the diagnostic-hierarchy rule resolves it. The other disorder this presentation is constantly confused with is **oppositional defiant disorder**, whose diagnostic architecture is developed in its own right elsewhere in this chapter. The overlap is substantial: roughly **15%** of children who meet criteria for oppositional defiant disorder also meet criteria for this disorder. The two share the angry, irritable, easily-provoked child, but this disorder demands more: severe, recurrent temper outbursts and a persistently disturbed inter-outburst mood, at a severity and chronicity that the oppositional diagnosis does not require. Because the presentations nest, the classification imposes a hierarchy: when a child meets criteria for both, only this disorder is diagnosed. That rule is the reverse of the usual instinct to give the "milder" oppositional label, and it reflects a judgement that the chronic mood disturbance is the more clinically significant fact about the child. The practical upshot is that a chronically irritable, explosive child who could be called oppositional is called by this disorder's name instead, once the outburst and mood thresholds are genuinely met.

PITFALL

Clinicians who anchor on the tantrums alone slide toward one of two wrong homes for this child: oppositional defiant disorder, if they read the outbursts as defiance, or bipolar disorder, if they read them as mania. Both misreadings share a single cause - attending to the explosive behaviour and neglecting the shape of the mood over time. The corrective is to characterise the baseline between outbursts and the longitudinal course before choosing a label, because it is the persistent, non-episodic irritability, not the outbursts, that actually discriminates this disorder from its neighbours.

20.7 Epidemiology

The prevalence figures carry a warning about the diagnosis itself. Community estimates vary substantially with the age of the sample and the instrument used, which is itself the

epidemiological headline. A population-based cohort of Brazilian eleven-year-olds, assessed with a dedicated diagnostic module, put the prevalence at about 2.5%, while a United States community sample of six-year-olds returned a figure closer to **8.2%**. Reading the two together, the diagnosis appears commoner in younger children, consistent with the general truth that irritability and tantrums are more prevalent, and less specific, earlier in childhood - which is part of why the criteria refuse the diagnosis below the lower age bound. Unlike several externalising disorders of childhood, a consistent sex difference has not been reported in population samples.

20.8 The two classifications part company

One major system created the disorder; the other declined to. The two international classifications disagree at the level of whether this entity exists at all. One houses it as a standalone diagnosis within the depressive disorders. ICD-11 **has no equivalent category**; the corresponding presentation is coded instead as oppositional defiant disorder with chronic irritability-anger, under the code 6C90.0. In other words, where one manual carves the chronically irritable child out as a separate depressive-spectrum disorder, the other keeps the child inside the oppositional category and attaches a specifier to mark the irritability. That system also holds that irritability-anger on its own is neither necessary nor sufficient for the oppositional diagnosis. This is a genuine structural disagreement, not a difference of wording, and it decides how the same child is recorded depending on which manual a service follows.

IN INDIA

Because Indian services classify under ICD, there is no disruptive mood dysregulation disorder to code in routine case files, referrals or official reports. The chronically irritable, explosive child is recorded as oppositional defiant disorder with the chronic irritability-anger specifier under its ICD code. A clinician trained on the other manual's categories should not go hunting for a separate mood-dysregulation diagnosis in an ICD-coded record: the same presentation lives here inside the oppositional diagnosis, and treating the two systems as describing different children, rather than the same child filed differently, is a recurring source of confusion in cross-referenced notes and in examination answers.

20.9 Management: what is established, and what is not

The honest teaching point is the absence of a specific evidence base. There is, as yet, **no established treatment** for this disorder as such. Two negatives and two provisional positives should be carried. First, lithium is ineffective here - a direct consequence of the disorder not being bipolar, and a useful confirmation that the depressive-spectrum placement is more than a filing decision. Second, SSRIs together with psychological and parenting interventions may be considered, though the supporting data specific to this indication are thin. Beyond that, treatment is genuinely investigational: trials of stimulant and adjunctive SSRI treatment for these children are in progress, and adjunctive SSRIs are in practice used for the irritable child with comorbid attention-deficit/hyperactivity disorder without a firm evidence base behind that use. The comorbid disorder itself, its stimulant and non-stimulant treatment, belongs to the ADHD and specific learning/communication disorders notes; here it is named only as the common travelling companion whose treatment sometimes carries the irritability with it. Benzodiazepines

are disfavoured, because they can increase disinhibition and aggression in this population. The examinable posture is therefore one of therapeutic humility, kept carefully distinct from the pharmacotherapy of aggression in conduct disorder, which is a separate indication with its own evidence and is developed elsewhere in this chapter.

The one line to carry: this is chronic, non-episodic irritability with severe frequent temper outbursts on a persistently angry baseline, placed among the depressive disorders precisely because it is not bipolar.

GOOD TO REMEMBER

Before this label changes anything you prescribe, characterise the course. If the irritability is chronic and non-episodic and the outbursts frequent on an angry baseline, you are in this disorder's territory and lithium and antipsychotics are not the reflex; if the irritability comes in discrete episodes distinct from baseline, you are not, and the assessment turns toward bipolar disorder. The clinical move that protects the child is to map the mood over months, not to weigh the size of the last tantrum.

3+ / week	**12 months**	**before 10**	**~15%**
TEMPER OUTBURSTS, ON AVERAGE	MINIMUM SYMPTOM DURATION	AGE AT SYMPTOM ONSET	OF ODD ALSO MEET CRITERIA

21 · Childhood bipolar disorder controversy

Few debates in child psychiatry have caused as much diagnostic harm as the question of whether young children can have bipolar disorder. The topic earns its marks not because paediatric bipolar disorder is common, but because a generation of children were labelled with it who did not have it, and a candidate is expected to understand exactly how that happened, why a new diagnostic category was created to stop it, and where the honest line now lies. The general bipolar syndrome, its full episode criteria and its neurobiology, belongs to the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; what this topic owns is the controversy itself, the reasoning behind it, and the discipline it demands at the bedside. The single most examinable idea is that a diagnosis with sharp, episodic boundaries was quietly stretched to cover children whose problem was not episodic at all.

The structure to carry into the exam is a short causal chain. A diagnostic upsurge occurred; it was driven by a specific conceptual move; that move collided with the defining requirement of the disorder; and the classification responded by carving out a separate home for the children who had been swept in. Each link is teachable, and each is where the marks sit.

21.1 The shape of the controversy

A rare disorder became, briefly, a common diagnosis. Over a relatively short span, bipolar disorder came to be applied to large numbers of children, first and most dramatically in the United States and afterwards, more cautiously, in other countries. The concern that grew out of this was not academic hair-splitting: it was that the disorder was being **over-diagnosed and**

over-treated in children, with young patients placed on mood stabilisers and antipsychotics on the strength of a label many of them did not warrant. That worry became formal enough that a new category, **disruptive mood dysregulation disorder**, was written into the classification for the express purpose of giving these children a more accurate diagnostic home. The whole controversy, in other words, is documented inside the classification that resolved it, which is why examiners treat it as settled teaching rather than open opinion.

21.2 How the upsurge happened: two presentations merged into one

The rise did not come from better detection of true mania; it came from a conceptual merger. The mechanism of the upsurge was that clinicians combined **two** genuinely distinct clinical pictures into a single category. On one side sat **episodic mania**, the classic, discrete elevation or expansion of mood that defines the adult disorder. On the other sat **non-episodic severe irritability**, a chronic, grinding crossness punctuated by explosive outbursts, with no discrete episode at all. Once these two were treated as versions of the same illness, the pool of eligible children expanded enormously, because chronic irritability is common and non-specific in the clinic while true episodic mania is not. The classification's answer was to draw the boundary sharply: in **DSM-5** the term bipolar disorder is explicitly reserved for the episodic presentation, so that the mere presence of a chronically irritable, explosive child can no longer be read as a mood-cycling illness.

RULE **Bipolar disorder is defined by episodes, not by irritability.** Irritability is a symptom that appears in mania, in depression, in anxiety and in ordinary childhood distress. What makes a presentation bipolar is a discrete change from baseline with an onset and an offset, not the intensity of a child's temper. Strip the episode away and you have not made a milder bipolar disorder; you have a different condition.

21.3 The crux: is chronic irritability a form of mania?

This is the question the whole debate reduces to. The crux is whether severe, **non-episodic irritability** should count as a paediatric form of mania. The answer written into the diagnostic rules is no, and the reasoning is structural rather than a matter of taste. Both DSM-IV and DSM-5 require distinct **a distinct manic episode** for a diagnosis of bipolar I disorder. A child whose irritability is present most of the day, most days, without ever resolving into and out of a bounded episode does not meet that requirement, however severe the presentation looks. This is why the debate was often framed as a contest between a narrow and a broad reading of the disorder. The **narrow phenotype** holds the episode requirement intact and keeps the diagnosis close to its adult form; the **broad phenotype** loosened it to include persistent irritability, and it was the broad reading that generated the over-diagnosis. The operational criteria for the disruptive mood dysregulation disorder that now houses these children are developed in their own right elsewhere in this chapter; here the point is only that the episode requirement is the fault line the two phenotypes fall on.

AXIS OF CONTRAST	NARROW PHENOTYPE (TRUE BIPOLAR)	BROAD PHENOTYPE (CHRONIC IRRITABILITY)
Mood pattern	Distinct manic episodes with a clear onset and offset	Persistent, non-episodic irritability with no discrete episode
Core disturbance	Episodic elevation or expansion of mood and energy	Chronic crossness with reactive, explosive outbursts
Diagnostic home	Bipolar I and the bipolar spectrum	Disruptive mood dysregulation disorder, placed among the depressive disorders
Longitudinal course	Continuity with adult bipolar disorder	Very low conversion to bipolar; risk of unipolar depression and anxiety

The two columns are not two grades of one illness; they are different conditions that were briefly filed together, and the episode requirement is what separates them.

21.4 Why a new category was created, and where it sits

The classification did not simply forbid the diagnosis; it built somewhere for the children to go. Removing the broad phenotype from bipolar disorder would have left a large group of genuinely impaired, chronically irritable children with no diagnostic label at all, and no clinician will leave such a child unnamed. The purpose of the new category was therefore twofold: to protect the bipolar diagnosis from erosion, and to give chronic paediatric irritability its own honest home. Crucially, that home was placed among the **depressive disorders** rather than in the bipolar section. That placement is not arbitrary filing. It is a claim about the underlying nature of the condition, and it is defended by the longitudinal evidence taken up next: these children do not travel toward bipolar disorder, so housing them beside it would repeat the very error the category was built to correct.

- it preserves the episodic meaning of the bipolar label;
- it gives severe non-episodic irritability a specific diagnosis of its own;
- it signals, by its placement, a depressive rather than a bipolar trajectory;
- it discourages the reflex prescription of mood stabilisers to irritable children.

21.5 What the follow-up data actually show

The longitudinal evidence is the strongest single argument against the broad phenotype. If chronic non-episodic irritability were really an early form of bipolar disorder, then children who show it should convert to bipolar disorder as they grow. They largely do not. The **rates of conversion** from severe non-episodic irritability to bipolar disorder are very low. What these children are at increased risk of, instead, is unipolar depressive and anxiety disorders in adulthood. This is a decisive discrimination for the exam, because it converts a question of definition into a question of prognosis: the two phenotypes do not share an outcome, and an illness is known partly by where it leads. The trajectory data are precisely what justify filing chronic irritability with the depressive disorders and keeping it out of the bipolar section.

GOOD TO REMEMBER

When a chronically irritable, explosive child is in front of you, the useful question is not "how severe is the temper" but "is there a discrete change from baseline". Hunt for a bounded period of elevated, expansive or unusually energised mood, distinct from the child's usual self, with a beginning and an end. If you cannot find one, you are not looking at bipolar disorder, and the treatment that follows a bipolar label does not follow.

21.6 The traps, and the position to defend

The examiner is testing discipline, not knowledge of a rare disorder. The safe position to write is the narrow one: bipolar disorder in children exists but is uncommon, it requires the same discrete episodes demanded in adults, and chronic irritability is not a substitute for those episodes. The candidate who can state that cleanly, name the category built to house the displaced children, and cite the low conversion rate as the reason for its placement has shown the whole reasoning chain the marks reward.

TRAP **Reading chronic irritability, or "very frequent mood swings", as paediatric rapid-cycling bipolar disorder.** The seductive move is to hear that a child is irritable "all the time" and explosive "several times a day" and translate that into ultra-rapid cycling. Frequency within a day is not cycling; cycling means discrete episodes. Label the presentation as chronic irritability, look for a true episode before you invoke bipolar disorder, and do not let severity stand in for episodicity.

PITFALL

The clinical harm of the broad phenotype was pharmacological. A bipolar label on a chronically irritable child leads almost automatically to a mood stabiliser or an antipsychotic, carrying metabolic and neurological burden, to treat a condition the child does not have. Naming the presentation accurately is not a semantic nicety; it is what keeps a child off years of unnecessary medication.

Episodic mania is bipolar disorder; chronic non-episodic irritability is not, and it was the conflation of the two that drove the over-diagnosis a separate diagnostic category was created to end.

IN INDIA

Indian child psychiatry classifies predominantly under ICD, which never carried the broad paediatric-bipolar phenotype the way American practice briefly did, so the over-diagnosis surge was largely a Western phenomenon that Indian trainees read about rather than lived through. The discipline it teaches still applies here. A chronically irritable, explosive child brought to an Indian clinic is common, and the temptation to reach for a bipolar label and a mood stabiliser is real; the correct response is to reserve the bipolar diagnosis for genuine episodic illness and to resist importing the broad reading that the international debate has already discredited.

two	very low	episodic
PRESENTATIONS WRONGLY MERGED INTO ONE	CONVERSION TO BIPOLAR DISORDER	WHAT THE BIPOLAR LABEL REQUIRES

22 · Early-onset schizophrenia and childhood psychosis

Psychosis in a child is not a smaller version of an adult illness; it is the same illness arriving early, on a brain that was already developing atypically. This topic is about schizophrenia and related psychotic states that declare themselves before adulthood, and it carries marks out of all proportion to how often the condition is seen, because it forces a candidate to do three things at once: recognise that true psychosis can begin in childhood, place it correctly on the neurodevelopmental continuum with the adult disorder, and separate it from the far commoner non-psychotic phenomena that mimic it. Get any one of those wrong and a well child is labelled schizophrenic, or a genuinely psychotic child is dismissed as imaginative, and both errors are examined.

The essential message is one of continuity with severity. Early-onset schizophrenia is uncommon, but where it occurs it is a harsher, more neurodevelopmentally saturated expression of the same process that produces adult schizophrenia. This topic owns that account in full: the definitions and the onset-age spectrum, the premorbid picture, the phenomenology, the neurocognition, the course and outcome, and the differential from the psychosis-mimics of childhood. The general neurobiology and pharmacology of schizophrenia belong to the adult chapters and are named here only where the reasoning demands them.

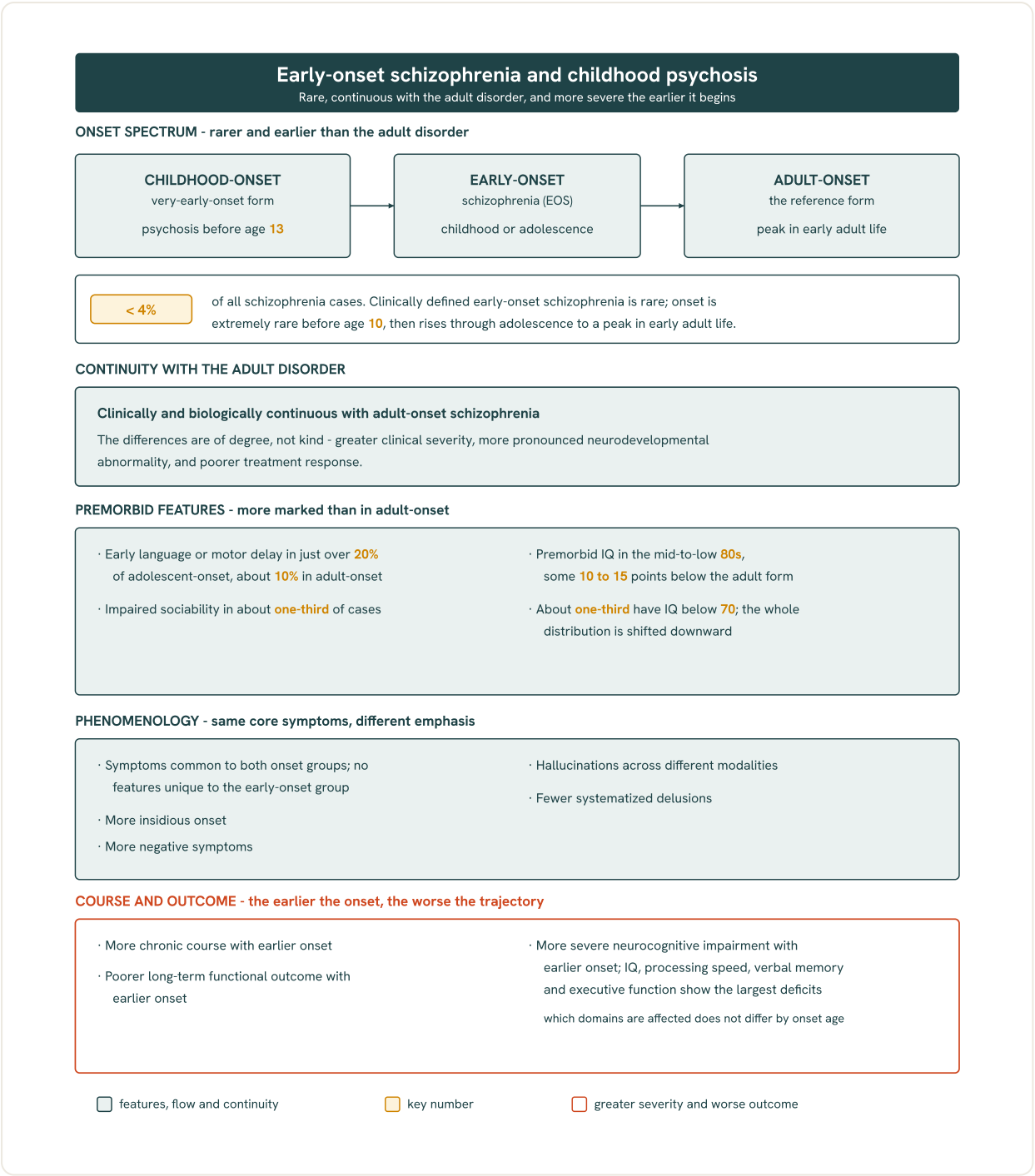

Fig 23.14. Early-onset schizophrenia and childhood psychosis: a rare disorder (under 4% of all cases, extremely rare before age 10) that is continuous with the adult form but more severe - childhood-onset psychosis begins before age 13, premorbid delay and low IQ are more common, phenomenology is broadly shared, and earlier onset predicts a more chronic course, poorer functional outcome and more severe neurocognitive impairment.

22.1 Definitions and the onset-age spectrum

Onset age is not a footnote; it defines the entities. The same diagnostic criteria used for adults are applied, unchanged, at every age, so what distinguishes these conditions is purely the age at which the full syndrome first appears. Two thresholds are worth holding. **Early-onset schizophrenia** refers to onset before adulthood, in the adolescent years, while the rarer **childhood-onset** or very-early-onset form is conventionally defined by onset of psychosis before the age of **13**, the boundary used by the National Institute of Mental Health childhood-onset cohort, which additionally required a near-normal premorbid intelligence and the absence of

significant neurological disease so that a primary psychosis was being studied rather than a secondary one.

Rarity is itself examinable. Clinically defined early-onset schizophrenia is genuinely rare, accounting for fewer than **4%** of all schizophrenia, even though self-reported psychotic-like experiences are far commoner in the general youth population and must not be equated with the disorder. Rarity increases sharply the younger the child: schizophrenia is extremely uncommon before the age of 10, and its incidence then rises steadily through adolescence towards a peak in early adult life. That gradient is why any convincing psychosis in a young child should prompt a careful search for a secondary cause before schizophrenia is accepted.

22.2 Continuity with the adult disorder

The default assumption is one illness across the lifespan, not two diseases. A large body of evidence indicates that early-onset schizophrenia is clinically and biologically **continuous** with the adult-onset disorder: the same diagnostic construct, broadly the same genetic and neuroimaging findings, the same treatment logic. What changes with earlier onset is quantitative, not categorical. Three differences stand out and are the ones to reproduce in an answer: earlier onset carries greater clinical severity, more pronounced neurodevelopmental abnormality, and a poorer response to treatment. The developmental framing follows directly. If schizophrenia is in part a disorder of aberrant brain maturation, then a case that surfaces during childhood or adolescence is one in which that aberration has been more marked, or has expressed itself sooner, and the child accordingly carries more of its developmental fingerprints.

RULE **Earlier onset is a more neurodevelopmental, more severe form of the same disorder, not a different one.** Do not present early-onset schizophrenia as a distinct childhood disease. Present it as adult schizophrenia declaring itself early, on a more compromised developmental substrate, which is precisely why its premorbid impairment is heavier, its symptoms more insidious, its cognition more affected and its outcome worse.

22.3 Premorbid functioning: the developmental fingerprint

The illness is usually visible, in retrospect, long before the first psychotic symptom. This is the single richest examination seam in the topic, because the premorbid abnormalities are more frequent and more severe than in adult-onset disease and they map cleanly onto the neurodevelopmental account. Careful histories find early **premorbid** difficulties across several domains, and they are worth carrying as a set because it is the pattern across all four, rather than any one of them, that points to a neurodevelopmental illness.

- **Language or motor delay.** Significant early delay is present in just over **20%** of adolescent-onset cases, markedly more than in adult-onset schizophrenia.
- **Impaired sociability.** The child who was always solitary, odd or poorly connected to peers: about a third of cases.
- **Premorbid IQ.** The group sits lower than the adult-onset group from the outset, averaging in the **mid-to-low 80s**, some 10 to 15 points below the adult-onset average.

- **Intellectual disability range.** Around a third of early-onset patients have a premorbid IQ below 70, with the whole distribution shifted downward rather than a small tail pulling the mean.

The clinical use of this is diagnostic, not merely descriptive. A convincing premorbid history of language delay, motor clumsiness, social isolation and academic struggle, followed by an insidious decline into psychosis, is far more consistent with a neurodevelopmental early-onset schizophrenia than with an acute reactive or drug-induced state, and it should raise rather than lower the index of suspicion.

> **GOOD TO REMEMBER**
>
> Always reconstruct the premorbid trajectory before you commit to a diagnosis. Ask the parents, and the school, about early speech and motor milestones, friendships, and the timing of any change. A lifelong drift of subtle developmental impairment that then steepens into positive symptoms supports a primary neurodevelopmental psychosis; a previously well child who becomes acutely psychotic over days should push you harder towards an organic, substance-related or mood-driven cause first.

22.4 Clinical phenomenology

The symptoms are the adult symptoms, with a shift in emphasis, not a new set. There are no phenomena unique to the early-onset group; the clinical symptoms are common to both. What differs is the balance and the presentation. Onset tends to be more **insidious** than in adults, creeping in over months of withdrawal, deteriorating function and blunting rather than announcing itself in a sudden florid break. The burden of **negative symptoms**, the avolition, flattening and social retreat, is heavier. Hallucinations tend to occur across different sensory modalities rather than being confined to the auditory, and delusions are typically less **systematized**, more fragmentary and less elaborately constructed than the organised delusional architecture of the older patient, in keeping with the cognitive immaturity and heavier cognitive impairment of the child. That combination, an insidious slide dominated by negative symptoms and disorganisation, is exactly what makes the diagnosis easy to miss early, when the picture can look like depression, oppositionality or simple failure to thrive at school.

22.5 The differential diagnosis of childhood psychosis

Far more children report psychotic-like phenomena than are psychotic, and this is the crux of the topic. The examiner is testing whether a candidate will resist the pull to over-diagnose. The first task is to establish that the phenomena are truly psychotic at all: developmentally normal imaginary companions, vivid fantasy, hypnagogic experiences and the misreported perceptions of a frightened or tired child are not psychosis, and isolated self-reported hallucinatory experiences in youth are common and usually transient. The second task is to exclude secondary and non-schizophrenic causes: delirium, epilepsy, central nervous system infection, substance intoxication, and the psychotic phenomena that can accompany severe mood disorder or trauma. The chronic irritability of disruptive mood dysregulation disorder, the mood-congruent phenomena and episodicity of the childhood bipolar disorder question, and the re-

experiencing and dissociation of childhood post-traumatic stress disorder are each developed elsewhere in this chapter and must be set against a psychotic presentation before schizophrenia is accepted. Distinguishing schizophrenia from autism spectrum disorder rests on the presence of genuine positive psychotic symptoms and a clear deterioration from a previously attained baseline, whereas the social-communication difficulties of autism are lifelong and non-psychotic; the disorder itself is taught in the Intellectual disability and autism spectrum disorder notes and is only named here.

TRAP **Reading normal or non-psychotic childhood experience as schizophrenia.** The classic error is to take a child's imaginary companion, vivid fantasy, or an isolated report of hearing a voice as evidence of psychosis and reach for schizophrenia. Such experiences are common, usually developmentally normal or transient, and clinically defined schizophrenia in childhood is rare. True psychosis requires sustained positive symptoms, disorganisation and functional deterioration, not a single frightening perception in an otherwise well child.

AXIS	EARLY-ONSET SCHIZOPHRENIA	ADULT-ONSET SCHIZOPHRENIA
Frequency	Rare, under 4% of all cases; increasingly rare the younger the child	The great majority of cases; peak incidence in early adult life
Premorbid development	Language and motor delay, impaired sociability and lower IQ far commoner	Premorbid impairment present but less frequent and milder
Onset and symptoms	More insidious onset, more negative symptoms, fewer systematized delusions	Often more acute; more organised, systematized delusions
Neurocognition	Same domains affected, more severe impairment	Same domains affected, less severe on average
Course and outcome	More chronic course, poorer functional outcome	Comparatively better course and outcome

The comparison is one of degree along shared axes rather than of kind; earlier onset shifts every axis in the direction of greater severity, which is the unifying idea of the whole topic.

22.6 Neurocognition

The cognitive deficit is qualitatively the same as in adults but quantitatively worse. Neurocognitive impairment is a core, not incidental, feature, and it is more severe the earlier the onset. Importantly, which domains are affected does not change between the groups: the largest deficits fall on **neurocognitive** measures of general intelligence, processing speed, verbal memory and executive function in both early- and adult-onset disease. The message for an answer is precise. Early onset does not produce a different cognitive profile, it produces a deeper one, and because it strikes during the years of active learning and skill acquisition, that deeper deficit compounds over development into a heavier functional handicap than the same absolute impairment would cause in a fully matured adult.

22.7 Course, outcome and principles of management

Age of onset is one of the more reliable prognostic markers available. The relationship is consistent and directional: the earlier the onset, the more **chronic** the course tends to be, and the poorer the long-term functional **outcome**. Early-onset schizophrenia thus sits at the more severe end of the schizophrenia spectrum on almost every measure that matters, which is why the poorer treatment response noted earlier is so consequential. Management follows the same principles as in adult schizophrenia, combining antipsychotic pharmacotherapy with family work, educational support and psychosocial rehabilitation, but with heightened attention to developmental needs, schooling and the greater sensitivity of the young to adverse effects; the specific agents, doses and monitoring lie outside what this topic establishes and should be taken from a pharmacology source and the antipsychotics chapter. What the topic does establish is the stance: treat early and adequately, support development actively, and hold realistic expectations shaped by the worse prognosis that early onset confers.

PITFALL

Because the onset is insidious and negative-symptom-dominated, the commonest clinical failure is not over-diagnosis but delay. The gradual withdrawal, falling grades and blunted affect are attributed to depression, laziness or oppositionality, and months pass before the psychosis is recognised and treated. In a young person with an unexplained, sustained decline in function and social contact, actively ask about attenuated psychotic phenomena rather than waiting for a florid presentation to force the diagnosis.

IN INDIA

Most services here classify under ICD, and the same criteria are applied irrespective of age, so the entity is recognised even where a dedicated child psychiatry service is not available. Two realities shape practice. First, specialist child and adolescent psychiatry capacity is thin outside the larger centres, so a psychotic child often reaches an adult general psychiatry clinic, where the developmental history is easy to under-take. Second, secondary and organic psychoses, including those of neuroinfection, epilepsy and metabolic disease, carry a meaningful load in Indian children and must be excluded with particular care before a primary early-onset schizophrenia is diagnosed. The practical rule is to invest in the premorbid and organic history, which is exactly the part that a rushed clinic is tempted to skip.

Carry this into the exam: early-onset schizophrenia is the same disorder as the adult form, arriving before adulthood on a more neurodevelopmentally impaired brain, and it is more premorbidly abnormal, more insidious, more cognitively affected, more chronic and poorer in outcome the earlier it begins.

under 4%	**before 13**	**just over 20%**	**about a third**
OF ALL SCHIZOPHRENIA IS EARLY-ONSET	ONSET DEFINING THE CHILDHOOD-ONSET FORM	WITH EARLY LANGUAGE OR MOTOR DELAY	WITH PREMORBID IQ BELOW 70

23 · Suicide and self-harm in children and adolescents

The act is the alarm, not the intent behind it. The single most useful move a clinician makes when a young person presents after hurting themselves is to stop trying to read their mind about whether they "really meant it" and to treat the act itself as the signal. **Self-harm** in children and adolescents is deliberately defined without reference to motive: it is any nonfatal act of self-poisoning or self-injury, whatever the degree of wish to die, and it is the doorway through which almost every completed youth suicide has first passed. This section owns the youth-specific tools for that presentation - what the act is, how common it is, how young people do it, what raises the danger, how the risk is assessed, and how it is managed once identified.

Marks accrue here because paediatric suicidality is where a candidate's judgement is tested rather than their recall. There is no criteria set to recite; there is a way of thinking. The examiner wants to see that you can define self-harm without collapsing it into "suicide attempt", that you know the risk factors and the structure of a risk assessment, and that you can turn that assessment into a concrete plan rather than a reassuring note. The wider adult framework and the pharmacovigilance question of antidepressants in the young are developed in their own right elsewhere; this topic teaches the child and adolescent detail in full.

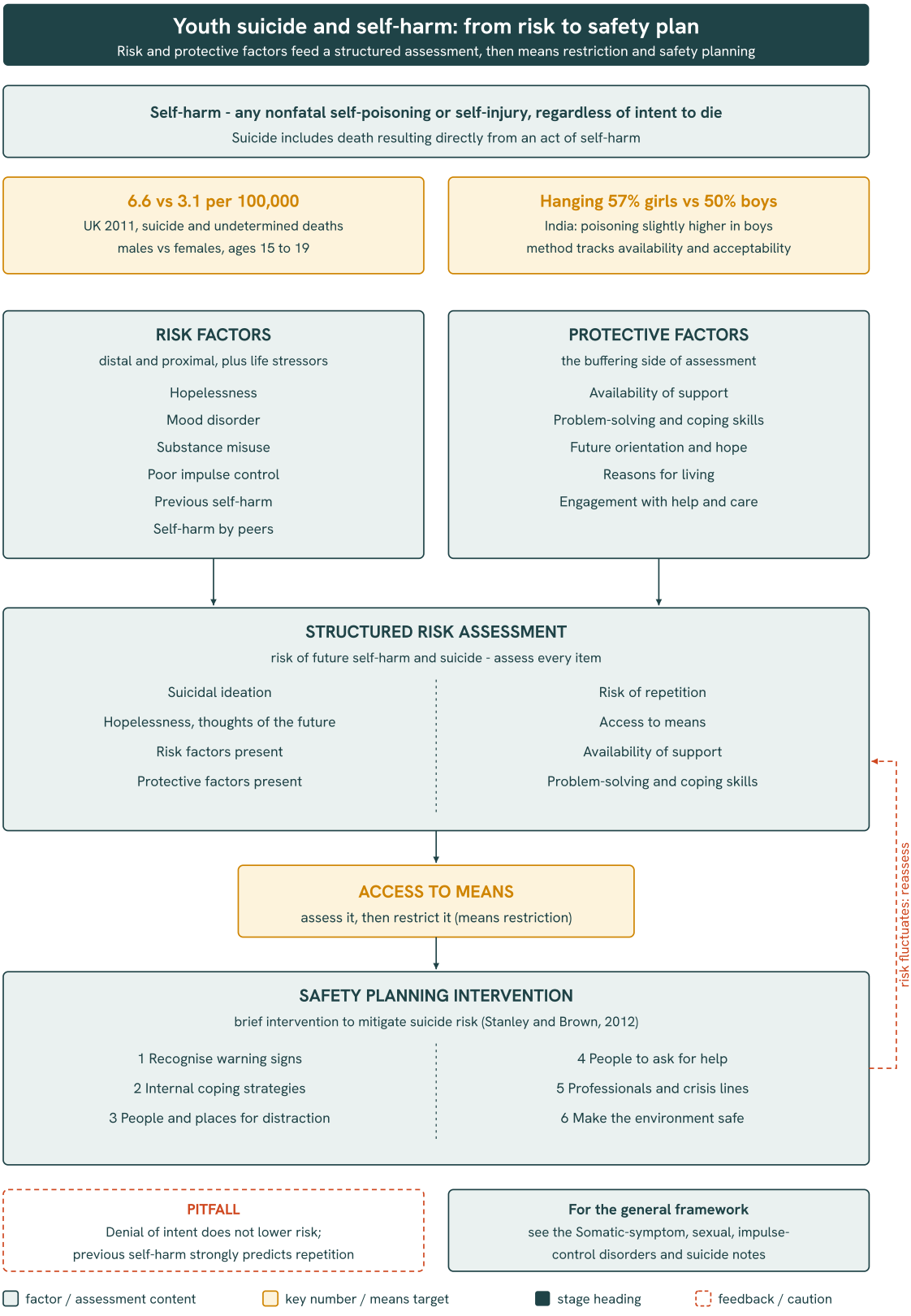

Fig 23.15. Youth suicide and self-harm pathway: risk and protective factors feed a structured risk assessment, then means restriction and a safety plan (Stanley and Brown), with reassessment as risk fluctuates.

23.1 What counts as self-harm, and how it relates to suicide

Self-harm is defined by the act, deliberately divorced from intent. The operational definition is broad on purpose. It takes in any form of nonfatal **self-poisoning or self-injury**, and it does so regardless of the motivation or the degree of intent to die that lay behind it. The cut made in private with no thought of dying, the impulsive overdose taken in front of a parent, and the near-lethal act interrupted by chance all fall under the one term, because the alternative - grading acts by inferred intent at the point of contact - is exactly where clinical assessment fails. Intent in an adolescent is fluid, poorly recalled and easily minimised, and an act that carried little wish to die today is still the strongest single marker of the act that may kill next year. Suicide sits at the fatal end of the same continuum: it is best understood as death resulting directly from an act of self-harm, not as a categorically different behaviour. Holding the definition this wide is what stops a clinician discharging a "minor" cut as unrelated to suicide risk.

- self-cutting and other self-injury;
- self-poisoning and overdose;
- hanging and self-strangulation;
- other dangerous acts such as running into traffic.

RULE **Assess the act, not the story about the act.** Because self-harm is defined without reference to intent, the clinician's job is never to decide whether the young person "meant it" and then act on that verdict. Every nonfatal act of self-poisoning or self-injury is taken as a marker of risk and assessed as such, whatever motive the young person or the family offers for it.

23.2 Epidemiology and the developmental gradient

Rare before puberty, then a sharp adolescent rise, with sexes that diverge. Completed suicide is uncommon before puberty and climbs steeply through adolescence, which is why the condition is a problem of the teenage years rather than of childhood proper. The pattern by sex is the examinable point and it reverses depending on which end of the continuum you count. For completed suicide the male rate runs higher: national figures for the United Kingdom put the rate of suicide and undetermined deaths in those aged 15 to 19 at **6.6 per 100,000** in males against **3.1 per 100,000** in females. Non-fatal self-harm shows the opposite skew, being commoner in girls, so that the same population produces more female acts but more male deaths - a reversal that turns on method and impulsivity rather than on distress. A methodological caution belongs here too: undetermined verdicts are not applied to those aged 10 to 14, so recorded rates in the youngest band understate the true burden and cross-country comparison must allow for how each coroner system counts.

23.3 Methods, and why the method is a clinical fact

Method is not incidental; it is where prevention gets its purchase. How a young person harms themselves is driven by what is culturally acceptable and what is physically available, and it varies by sex and by country in ways that matter at the bedside. The Indian pattern is a standing examination contrast. Where in many countries hanging predominates among males, Indian data show hanging to be somewhat more frequent in girls than in boys, and self-poisoning slightly

commoner in boys, so the Western default cannot simply be transposed. The clinical significance is that method choice is modifiable at the population level: restrict access to the lethal means that are locally common and you reduce deaths, because much youth self-harm is impulsive and a barrier at the moment of crisis buys the time in which the impulse passes.

METHOD	PATTERN BY SEX IN INDIAN YOUTH
Hanging	More frequent in girls (**57%**) than in boys (50%)
Self-poisoning	Slightly commoner in boys (**50%**) than in girls

Method distribution by sex in Indian youth; the contrast with the male-hanging predominance reported in many other countries is itself the teaching point, and it exists because acceptability and availability, not biology, set the method.

IN INDIA

Two locally common methods carry most of the mortality of youth self-harm: hanging and the poisons that sit within reach in Indian homes, above all agricultural pesticides and household chemicals. That makes means restriction a concrete, culturally specific task rather than an abstraction - securing or removing pesticides and medicines, and counselling families on storage, is often the highest-yield intervention available and one that does not depend on a scarce specialist. It also reframes the assessment: asking what is physically accessible to this particular young person in this particular home is not a formality but the part of the interview that most directly changes the odds.

23.4 Risk factors: a distal-proximal pathway

Suicidality is the endpoint of accumulating risk, not a single cause. No one factor explains a suicidal act; the useful model is a pathway in which longstanding vulnerabilities and immediate precipitants combine against a background stressor to push a young person over threshold. The core risk factors an examiner expects, read as one set, are **hopelessness**, an underlying mood disorder, substance misuse, poor impulse control, a history of previous self-harm, and self-harm among the young person's peers. They separate naturally along a time axis. The more distal contributors are the standing conditions - a depressive illness, a substance problem, a temperamentally impulsive style - that raise baseline vulnerability over months and years. The more proximal are the near-term markers, above all a recent act of self-harm or exposure to it in a close friend, and these fire in the setting of an acute life stressor such as a humiliation, a loss, a relationship rupture or a disciplinary crisis. Previous self-harm deserves particular weight as the strongest behavioural predictor of a further act, which is why a history of it reframes the whole assessment. The depressive illness that so often underlies the picture is developed as childhood depression in its own right elsewhere in this chapter, and the separate question of whether starting an antidepressant itself alters suicidality in the young is taken up as a distinct topic; here the mood disorder is named as a risk domain, not re-taught.

23.5 Assessing the risk of future self-harm

Structured content beats prediction. No instrument predicts which individual will die, and a candidate who promises the examiner a risk score has missed the point. What a good assessment does is systematically cover the domains that inform judgement and disposition, so that nothing

decisive is left unasked. The content the young person's assessment must reach is definable and this topic owns it: current suicidal ideation and its intensity; hopelessness and how the young person pictures their future; the risk factors present; the protective factors that pull the other way; the **risk of repetition**; **access to means**; the availability of support around them; and their problem-solving and coping capacity. Two of these do double duty as intervention targets rather than mere descriptors - access to means, because it can be restricted, and support, because it can be mobilised - which is what makes the assessment continuous with the plan rather than a separate exercise that precedes it.

DOMAIN OF THE ASSESSMENT	WHAT IT ESTABLISHES
Suicidal ideation	Whether thoughts are present, and their frequency, intensity and specificity
Hopelessness and view of the future	The affective marker most tied to intent; can the young person imagine things improving
Risk factors	The distal and proximal contributors and the precipitating stressor
Protective factors	Reasons for living, attachments and responsibilities that pull against the act
Risk of repetition	Prior self-harm and the features that predict a further act
Access to means	What lethal means are physically available; an explicit target for restriction
Availability of support	Who can watch, contain and respond; can it be mobilised now
Problem-solving and coping	The young person's capacity to tolerate and resolve the crisis

The content a youth suicide-risk assessment must cover; it is a structure for gathering the information that informs disposition and a plan, not a scale that outputs a probability of death.

TRAP **Reading a denial of ideation as an absence of risk.** The classic error is to accept "I don't want to die" at face value and stand down. A young person may minimise or genuinely fluctuate, and low intent at interview coexists with high danger when the means are lethal and to hand. Risk is judged from the whole assessment - prior acts, hopelessness, access to means and support - never from the single question about current wish to die.

PITFALL

Labelling self-harm of apparently low intent as "attention-seeking" and treating it as a behaviour to be ignored is a recurrent and dangerous clinical failure. The framing licenses the clinician to skip the assessment and the means-restriction conversation, yet low-intent acts are precisely the population from which impulsive deaths emerge. Communicative or interpersonal function and lethal risk are not alternatives; an act can be both a signal to others and a genuine hazard, and it is assessed on its danger regardless of what it also communicates.

23.6 Managing the risk: safety planning and means restriction

The plan is an intervention, not a promise extracted from the patient. Management of the identified risk turns on a small number of high-yield actions taken with the young person and their family. Chief among them is a **safety planning intervention** - the brief, structured safety plan described by **Stanley and Brown** - which works with the young person to recognise their own warning signs, to name internal coping strategies and people and settings that provide distraction, to identify who to contact in a crisis, and to reduce the danger of the immediate environment. It is deliberately distinct from the discredited "no-suicide contract", which asks for a promise and changes nothing. Coupled to it is **means restriction**: because access to means is both an assessment item and a modifiable target, removing or securing the lethal means available to this young person is a direct reduction in risk and a routine, non-negotiable component of the plan. Around these sit the treatment of the underlying mood, substance or impulse-control disorder, the mobilisation of family supervision and support, and clear, explicit arrangements for follow-up and re-assessment, since risk is not a fixed quantity settled at one interview but a state that shifts with circumstance and must be reviewed.

The general suicide-risk framework that applies across the lifespan - the wider theory of assessment and the adult evidence base - is developed in the Somatic-symptom, sexual, impulse-control disorders and suicide notes; what this section adds is the child and adolescent application of it, where developmental fluidity of intent, dependence on the family, and culturally specific means make the youth version its own competence.

GOOD TO REMEMBER

Every young person who has harmed themselves leaves the consultation with two things done, not one: a completed risk assessment and a concrete plan that includes means restriction. The assessment without the plan is an observation; the plan without honest means restriction is a document. Doing both, and arranging who reviews the young person and when, is the deliverable the examiner and the parent are both looking for.

Self-harm is defined by the act and not the intent behind it; take every nonfatal self-poisoning or self-injury as a risk marker, assess it across its full content, and convert that assessment into a safety plan with means restriction.

6.6 per 100,000 SUICIDE AND UNDETERMINED DEATHS, ADOLESCENT MALES (UK)	**3.1 per 100,000** SUICIDE AND UNDETERMINED DEATHS, ADOLESCENT FEMALES (UK)	**57%** HANGING AS A METHOD IN GIRLS (INDIA)	**50%** SELF-POISONING AS A METHOD IN BOYS (INDIA)

24 · Antidepressant use and suicidality warning in youth

This is the most politically charged prescribing decision in child psychiatry, and the examiner knows it. A young person with moderate to severe depression stands to gain from an antidepressant, yet the same drug carries a regulatory warning that it may increase suicidal

thinking in exactly this age group. Marks accrue here because the topic tests something harder than a fact: it tests whether a candidate can hold two true statements at once, that the risk signal is real and that the treatment is still, for the right patient, correct. The general pharmacology of antidepressants, their classes and mechanisms, belongs to the Mood disorders: pharmacotherapy notes; the assessment of suicide risk itself, its structured tools and safety planning, is developed elsewhere in this chapter. What this topic owns is the pharmacovigilance story: where the warning came from, how large the risk actually is, the adverse effects unique to starting these drugs in the young, and how the child must be monitored once the prescription is written.

The reasoning to carry into the exam runs in a fixed order. First the signal and the trials that produced it. Then the single most important move in the whole topic, converting an alarming relative risk into a small absolute one. Then the regulatory response, the youth-specific adverse effects, and the monitoring that the warning demands.

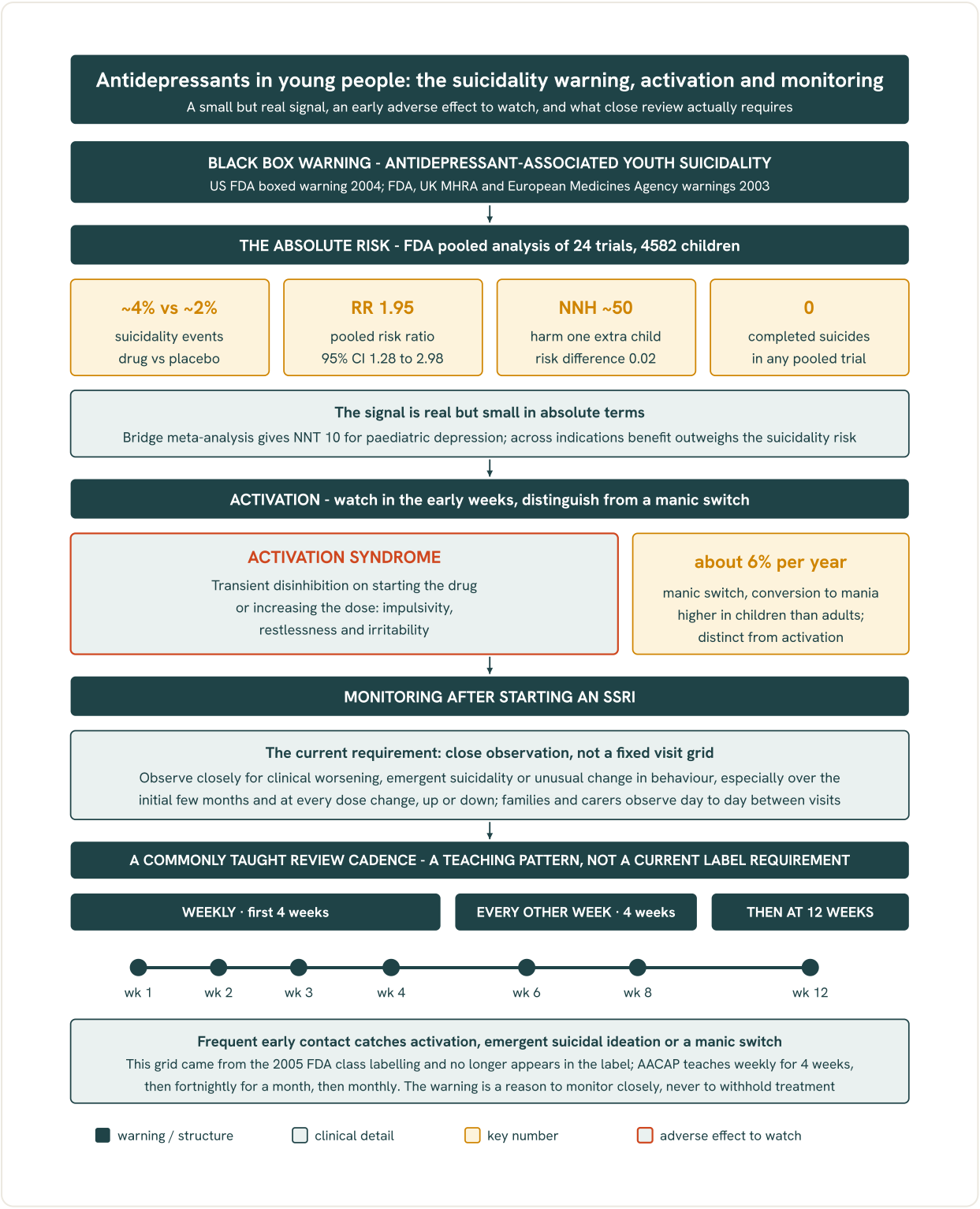

Fig 23.16. Antidepressants in young people: the paediatric suicidality warning quantified (absolute risk, risk ratio and number needed to harm), activation as the early adverse effect to distinguish from a manic switch, and what the label requires after starting an SSRI: close observation through the initial few months and at every dose change, with daily family observation between visits, rather than a fixed visit grid.

24.1 The signal and where it came from

The warning rests on a pooled analysis, not on a stream of deaths. When regulators grew concerned, the United States regulator assembled the paediatric placebo-controlled antidepressant trials and pooled their **suicidality** events, meaning suicidal ideation and suicidal behaviour recorded as adverse events, not completed suicide. Across 24 trials enrolling some 4582 children and adolescents, the pooled risk of a suicidality event on active drug against

placebo gave a risk ratio of **1.95** across all drugs and all indications. Restricted to the selective serotonin reuptake inhibitors used in depression trials, the risk ratio was 1.66. Those numbers are the origin of the entire controversy, and two features of them must be reproduced. They describe emergent suicidal ideation and behaviour, and the crucial fact that anchors every sensible reading of the data is that there were no completed suicides in any of the pooled trials.

24.2 Relative against absolute: the number that actually matters

A doubled relative risk sounds catastrophic until you see the base rate it doubles. A risk ratio near two means the event became about twice as likely, but on a rare event twice a small number is still a small number. The quantity that a clinician and a family can actually use is the **absolute risk difference**, and here it was 0.02, roughly two percentage points. In the regulator's cell rates that appears as approximately **4 per cent on antidepressant versus 2 per cent on placebo**. Inverting that risk difference gives a **number needed to harm** of approximately 50, meaning that for every fifty young people treated, one additional patient reports emergent suicidal thinking or behaviour who would not have on placebo. The teaching point is that the alarming headline and the modest reality are the same finding stated two ways.

The estimate is a range, not a point. Different pooled datasets used slightly different denominators and produced different absolute figures, and the exam rewards a candidate who can bound the signal rather than parrot one number. A meta-analysis cited in the standard formulary gives a lower estimate, roughly 2 per cent versus 1 per cent, with a number needed to harm of 112. A separate meta-analysis of 27 trials found a pooled risk difference of 0.7 per cent and a number needed to harm of 143, with the within-indication differences not reaching statistical significance and, again, no completed suicides. These are not competing truths to be adjudicated; they are the lower and upper bounds of a single small signal.

SOURCE	RELATIVE MEASURE	ABSOLUTE MEASURE	NUMBER NEEDED TO HARM
Regulator's pooled analysis	risk ratio 1.95, all drugs and indications	risk difference 0.02; about 4 per cent versus 2 per cent	approximately 50, derived
Formulary meta-analysis	lower estimate	2 per cent versus 1 per cent	112
27-trial meta-analysis	within-indication differences not significant	risk difference 0.7 per cent	143

The same suicidality signal, expressed by three groups. The relative risk is what alarmed regulators; the absolute risk difference and the number needed to harm are what a clinician weighs against benefit at the bedside.

RULE **Never quote the relative risk without the absolute risk beside it.** A risk ratio near two, stated alone, implies a doubling of deaths and is the single commonest misstatement of this evidence. Stated with its absolute risk difference of two percentage points and its origin in ideation rather than completed suicide, the same finding becomes a manageable, monitorable adverse effect.

24.3 The regulatory response: the boxed warnings

The warning is a class-wide regulatory act, and its dates and wording are examinable. The concern was taken up by regulators on both sides of the Atlantic: warnings on antidepressant-associated youth suicidality were issued by the American regulator, the United Kingdom medicines regulator and the European Medicines Agency in 2003. The American regulator then formalised the strongest label available to it, a **boxed (black box) warning**, in 2004, flagging worsening depression, agitation and suicidal ideation linked to these drugs and drawn from the review of adolescent depression studies. What the warning did NOT say is as important as what it said: it did not withdraw the drugs, did not forbid their use, and did not claim that they cause completed suicide. It mandated informed prescribing and close monitoring, which is the spirit a candidate should convey. Whether the warning was subsequently extended upward to young adults is a separate regulatory question that the sources used here do not settle, and a candidate should not assert an upper age boundary they cannot source.

> **GOOD TO REMEMBER**
>
> The warning changed the conversation with families more than it changed the pharmacology. Before starting an antidepressant in a young person, the modern standard is an explicit, documented discussion: that emergent suicidal thoughts are an uncommon early adverse effect, that they are usually transient, that the family should watch for them in the first weeks and report them immediately, and that this is precisely why review is scheduled early and often. A prescription written without that conversation is the one that gets challenged.

24.4 Why benefit still outweighs risk

The warning is a reason to prescribe carefully, not a reason to withhold. The same body of trials that produced the suicidality signal also demonstrated efficacy: the 27-trial analysis found a **number needed to treat** for paediatric major depressive disorder of **10**, and the authors judged that the benefits outweighed the suicidality risk across indications. Set the two frequencies side by side and the logic is stark: roughly one young person benefits for every ten treated, while the additional suicidality events run at one in fifty to one in a hundred and forty, and none of those events was a death. Against this sits the risk that the warning itself can do harm, because untreated adolescent depression is itself a leading driver of suicidal behaviour, and a chilling effect that reduces prescribing without reducing illness can raise the very outcome everyone fears. The examinable synthesis is therefore not "avoid antidepressants in the young" but "treat the depression that needs treating, and build the monitoring in".

TRAP **Reading the signal as an increase in completed suicide.** Candidates hear "antidepressants increase suicide risk in youth" and picture deaths. The pooled trials recorded emergent suicidal ideation and behaviour as adverse events and contained no completed suicides at all. Stating that antidepressants were shown to increase completed suicide in these trials is factually wrong and marks the answer as unsafe.

24.5 Activation, and its separation from a manic switch

Two early adverse phenomena are constantly confused, and distinguishing them is a favourite station. The first is **activation**, a transient disinhibitory response to starting an antidepressant or increasing its dose, characterised by impulsivity, restlessness and irritability. It is dose-related, it appears early, and it typically settles when the dose is reduced or the drug is withdrawn. Its clinical danger is that in an impulsive, disinhibited adolescent it can be the substrate on which emergent suicidal behaviour is enacted, which is one plausible route from the drug to the suicidality signal above. The second phenomenon is the **manic switch**, a conversion to a manic or hypomanic state, which is reported at approximately **6 per cent per year** in young people taking antidepressants and appears to be commoner in children than in adults, although there is no clear evidence that the antidepressant actually causes the switch rather than unmasking a latent bipolar diathesis. Activation must be differentiated from a manic switch, because the two demand different responses.

FEATURE	ACTIVATION	MANIC SWITCH
Nature	transient disinhibition with impulsivity, restlessness and irritability	sustained elevated or expansive mood, reduced need for sleep, grandiosity, pressured behaviour
Timing and course	early, dose-related, settles on dose reduction or withdrawal	may persist and may herald an underlying bipolar disorder
Frequency	a recognised early effect of initiation and titration	about 6 per cent per year, apparently higher in children
Response	reduce or stop the drug; the phenomenon reverses	reassess the diagnosis; a mood stabiliser may be needed

Activation is a reversible, dose-linked disinhibition; a manic switch is a syndromic mood change that questions the diagnosis. Confusing the two leads either to needless drug withdrawal or to a missed emerging bipolar disorder.

PITFALL

The frequent clinical error is to read early activation as the depression worsening and respond by pushing the dose higher, which amplifies the very disinhibition driving the problem. The mirror error is to see any early agitation as an emerging bipolar switch and stop the antidepressant altogether, abandoning a treatment the child needed. The safe move is to recognise activation as its own transient, dose-related entity, hold or reduce the dose, and reassess, reserving a diagnostic rethink for genuinely sustained, syndromic mood elevation.

24.6 The monitoring the warning demands

The warning converts into a concrete schedule of contact. Because the suicidality signal and activation both cluster in the early weeks, the regulator paired the label with a monitoring schedule following initiation of a serotonergic antidepressant in a child, front-loaded to catch trouble when it is most likely:

- weekly review over the first four weeks;
- then a review every other week for a further four weeks;

- then a review at week twelve.

The academic child psychiatry literature concurs with a schedule of the same shape, weekly for the first month, then fortnightly for a month, then monthly thereafter. Two honesties should accompany this in an exam answer. The schedule is expert consensus rather than a proven intervention, and there is no firm evidence that adhering to this exact calendar changes outcome; and "monitoring" means active enquiry into mood, suicidal ideation, activation and any switch to elevated mood, not a passive repeat prescription. Reproducing the schedule earns the mark; adding that it is consensus and defining what is actually monitored earns the rest.

The youth antidepressant signal is a doubling of emergent suicidal ideation, not of completed suicide, over a base rate of a few per cent; the response is careful prescribing, early front-loaded monitoring, and a family who know what to watch for, not withholding an effective treatment.

IN INDIA

The warning translates awkwardly into Indian practice. Antidepressants in children are largely prescribed off-label, most often fluoxetine, and frequently by non-specialists, because child psychiatry services are scarce and concentrated in a few tertiary centres. The front-loaded monitoring schedule, which assumes weekly face-to-face review through the first month, is difficult to deliver when families travel long distances and follow-up is irregular. The practical adaptation is to make the family the monitor: to explain activation and emergent suicidal thinking in plain language, to give an explicit instruction to return or telephone at once if either appears, and to use telephone or teleconsultation contact to approximate the weekly review the label intends. The signal itself is not Indian or Western; the ability to monitor for it is where the local reality bites.

1.95	~4% vs 2%	10	0
POOLED SUICIDALITY RISK RATIO	ABSOLUTE EVENT RATES, DRUG VS PLACEBO	NUMBER NEEDED TO TREAT, PAEDIATRIC MDD	COMPLETED SUICIDES IN THE TRIALS

Attachment disorders of childhood

25 · Reactive attachment disorder (criteria, etiology)

This is the one childhood diagnosis whose very definition points a finger at the caregiving environment rather than at the child. **Reactive attachment disorder** is the syndrome in which a young child, having been denied the ordinary experience of a responsive caregiver, fails to develop the normal behavioural signs of a **selective attachment**: when frightened, hurt or ill, the child neither turns to an adult for comfort nor takes much comfort when it is offered. What makes the disorder examinable is not the symptom picture of a flat, withdrawn, joyless child, but the demanding structure of its criteria - a specific behavioural pattern, a required history of grossly inadequate care, an explicit causal link between the two, a set of exclusions, and two age gates - all of which must hold together before the label can be used.

Its placement in the classification carries the whole argument. Rather than sitting with the neurodevelopmental disorders, where a naive reader might expect a disorder of early social behaviour to live, it is classified among the **Trauma- and Stressor-Related Disorders**, because the disturbance is understood as a response to an identifiable adverse experience. This topic owns the diagnostic criteria of reactive attachment disorder, its specifiers, its epidemiology, the etiological role of neglect, its associated features and course, and its within-section differentials. Its mirror-image sibling, **disinhibited social engagement disorder**, in which the same deprivation produces an indiscriminately over-familiar rather than a withdrawn child, is taught in its own account here and is named only as the contrasting pattern. The attachment-side comparison with autism, and the effects of institutional care with the management of these disorders, each belong to their own accounts elsewhere in this chapter.

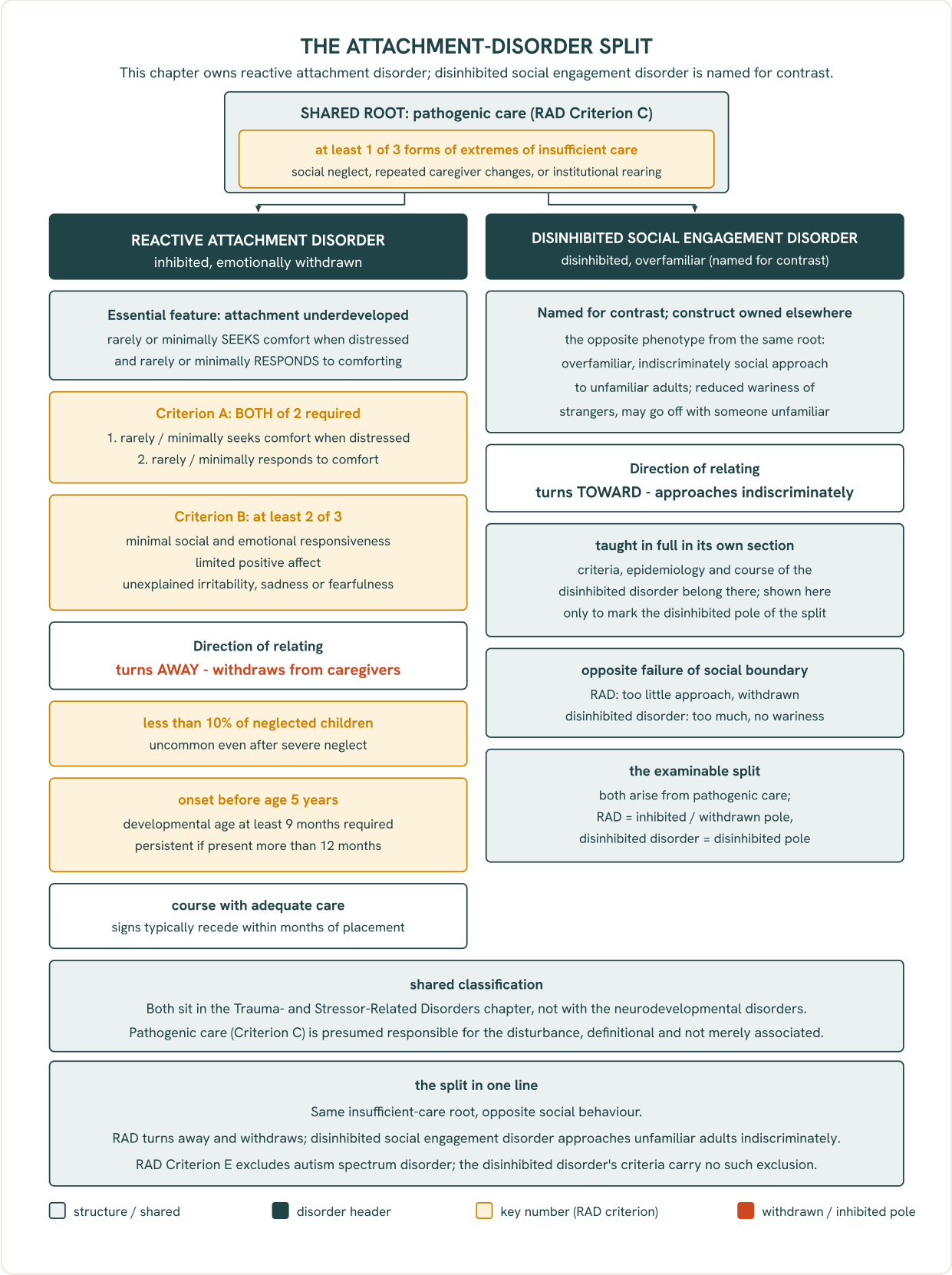

Fig 23.17. The attachment-disorder split: reactive attachment disorder (inhibited, withdrawn) against disinhibited social engagement disorder (disinhibited, overfamiliar, named for contrast), both arising from the same pathogenic care, with RAD turning away from caregivers and the disinhibited disorder turning toward unfamiliar adults.

25.1 The essential feature: an attachment that never took hold

The core of the disorder is an absent or grossly underdeveloped attachment to caregiving adults. A typically developing infant, by the second half of the first year, begins to show a

preferred attachment figure - seeking that person out when distressed and using the caregiver as a secure base. In reactive attachment disorder these behaviours are missing. The prevailing understanding is that these children retain the underlying **capacity to form selective attachments** but, deprived of the early relational opportunity to do so, do not show the behavioural manifestations of one; when distressed they rarely or only minimally seek comfort, and rarely or only minimally respond to comfort when it is given. The clinical impression is of a child who has learned that adults do not reliably answer need, and who has stopped asking. This separates the disorder at its root from a primary disorder of social capacity, and is why better caregiving can change the picture at all.

25.2 Criterion A: the inhibited, emotionally withdrawn pattern

Criterion A defines the behavioural signature, and it is a conjunction, not a menu. The child must show a consistent pattern of inhibited, emotionally withdrawn behaviour toward adult caregivers, evidenced by both of two features: the child rarely or minimally seeks comfort when distressed, and rarely or minimally responds to comfort when distressed. The word "both" is load-bearing. A child who seeks no comfort but is soothable once held, or who cannot be soothed but still turns to the caregiver, does not satisfy Criterion A. The pattern captured is a two-sided failure of the comfort transaction - the child neither initiates the bid for care nor completes it by being reassured - and it is this doubled withdrawal, present consistently rather than in a single frightened moment, that marks the inhibited attachment presentation the disorder is named for.

25.3 Criterion B: the persistent social and emotional disturbance

Criterion B broadens the picture from the attachment relationship to the child's general emotional life. Alongside the withdrawn attachment behaviour there must be a persistent social and emotional disturbance shown by at least two of three features: minimal social and emotional responsiveness to others; limited positive affect; and episodes of unexplained irritability, sadness or fearfulness that are apparent even during non-threatening interactions with caregivers. The third feature is the most clinically telling and the most easily overlooked, because it locates distress where there should be none - the flashes of misery or fear surface in ordinary, safe exchanges, not only under threat, which is what separates this from a child who is simply frightened by a specific stressor. Where Criterion A describes what the child fails to do within the attachment bond, Criterion B describes the pervading flatness and dysregulation that colour every relationship.

25.4 Criteria C and D: the pathogenic care and the causal link

This pair of criteria is what makes reactive attachment disorder unlike almost any other diagnosis in the manual. Criterion C requires that the child has experienced a pattern of extremes of insufficient care, evidenced by at least one of three recognised forms of deprivation:

- social neglect or deprivation, a persistent lack of having basic emotional needs for comfort, stimulation and affection met by caregiving adults;
- repeated changes of primary caregiver that limit the child's opportunity to form a stable attachment, as with recurrent moves through foster placements;

- rearing in unusual settings that severely limit the chance to form selective attachments, such as institutions with high child-to-caregiver ratios.

Criterion D then does the work that no associated risk factor could: it requires that the inadequate care in Criterion C is **presumed responsible** for the disturbed behaviour in Criterion A, as when the disturbance is seen to have followed the onset of neglect. This is what elevates **pathogenic care** from a mere correlate to a definitional requirement: the diagnosis is not "a withdrawn child who happens to have been neglected" but "a withdrawn child whose withdrawal is judged to arise from that neglect".

MNEMONIC

The three N's of Criterion C

The pathogenic care takes three recognised forms, each beginning with an **N**: social **N**eglect of the child's emotional needs, repeatedly **N**ew caregivers whose turnover prevents a stable bond, and iN**N**stitutional or otherwise unusual rearing that starves the child of any selective figure. Only one of the three need be present, but at least one must, and Criterion D then requires that it be judged the cause.

RULE **Pathogenic care is definitional to reactive attachment disorder, not merely associated with it.** A child cannot receive this diagnosis without a documented history of grossly insufficient care, and the criteria go further in demanding that the care be presumed to have caused the disturbance. Remove the neglect and the diagnosis dissolves, which is exactly why the disorder is filed with the trauma- and stressor-related conditions rather than with disorders of innate social development.

25.5 Criteria E to G: the autism exclusion and the two age gates

The final three criteria are exclusions and thresholds that keep the diagnosis honest.

Criterion E is an explicit exclusion built into the disorder's own criteria set: the presentation must not meet the criteria for autism spectrum disorder. It is worth noting that this exclusion sits inside reactive attachment disorder but has no counterpart in the criteria of its disinhibited sibling. The fuller attachment-side reasoning for telling these disorders apart from autism, and autism itself, are taught elsewhere; here the point is only that the exclusion exists and must be cleared. Criterion F sets the upper age gate: the disturbance must be evident **before age five years**. Criterion G sets the lower, developmental gate: the child must have a developmental age of **at least nine months**. The rationale for that floor is precise and examinable - around this developmental level the capacity to form a selective attachment normally emerges, so a child who has not reached it cannot be said to have failed at a task they were not yet equipped to attempt. The gate is developmental, not chronological, which matters in a population where developmental delay is common.

GOOD TO REMEMBER

Before diagnosing reactive attachment disorder in a slow-to-develop or neglected infant, establish the child's developmental age, not just the age in months. A child functioning below the nine-month developmental level has not yet reached the stage at which a selective attachment would normally form, so the absence of comfort-seeking cannot be read as the disorder. Screening developmental level first prevents the diagnosis being pinned on a child who is simply not developmentally ready to attach.

25.6 The specifiers: persistence and severity

Two specifiers refine the diagnosis once it is made. The condition is specified as **persistent** when it has been present for **more than twelve months**, marking a presentation that has not resolved with time or with any change of circumstance and that therefore carries a heavier prognostic weight. Current severity is specified as severe when the child exhibits all of the symptoms of the disorder, with each manifesting at relatively high levels. Both specifiers matter because the disorder is, in favourable circumstances, recoverable; a presentation persisting beyond a year despite the chance of better care, or severe across every symptom, marks a child in whom the deprivation has bitten deep and for whom a change of environment alone may not suffice.

25.7 Epidemiology and etiology: neglect necessary, but far from sufficient

The single most important etiological fact is a paradox. Serious social neglect is at once a diagnostic requirement and the only established risk factor for the disorder, and yet the great majority of severely neglected children never develop it. The disorder is seen relatively rarely even in clinical settings; its general-population prevalence is unknown, and even among neglected children it is uncommon, arising in **fewer than one in ten** of them even where the neglect has been severe. That combination - neglect is necessary but not sufficient - is the whole of the etiological teaching a candidate can state with confidence. Pathogenic care sets the stage, but something further, some pattern of individual vulnerability or some specific quality of the neglectful environment, decides which deprived child develops the disorder and which does not, and those vulnerability factors and the critical ingredients of the neglect remain incompletely defined, with no confident biological mechanism assertable beyond the requirement of neglect itself.

25.8 Associated features, comorbidity and course

What travels with the disorder travels with it because of the shared root in neglect. The associated problems are largely those of a deprived early environment: **developmental delays**, particularly in cognition and language, together with stereotypies and the physical stigmata of severe neglect such as malnutrition. Internalising symptoms may co-occur; a relationship with externalising problems or with attention-deficit/hyperactivity disorder has been suggested but is not clearly established, and the reader should hold it loosely. The course is the most hopeful part of the picture and the part most dependent on what happens next. Signs of the disorder typically recede within months once the child is placed in an adequate caregiving environment; in the absence of that enhanced caregiving the signs may persist for several years. Prognosis, in short,

tracks the quality of the post-neglect caregiving far more than any property of the child. The full evidence on institutional deprivation and its remediation, and the interventions themselves, are the province of the attachment-disorder management account elsewhere in this chapter.

PITFALL

When a child's withdrawn signs persist after placement in what looks like a good home, the reflex is to call the treatment a failure or the child untreatable. The more useful reading is that a withdrawn, unrewarding child is genuinely hard to warm to, and can quietly elicit less engaged caregiving even from willing carers - a continuing de facto neglect that keeps the disorder alive. The clinical task then is not to abandon the placement but to support the caregiver's sensitivity, because persistence is often a sign that the child is still not, in practice, receiving the responsive care the diagnosis presumes was missing.

25.9 ICD-11, and telling the disorder apart from its mimics

The two classifications converge, and the differentials are where marks are won or lost. The ICD-11 clinical descriptions mirror the manual approach with closely similar requirements - a history of grossly insufficient care, a persistent and pervasive pattern of inhibited, emotionally withdrawn behaviour toward caregivers with both the minimal comfort-seeking and the minimal response to comfort, and the insufficient care presumed responsible - coding the disorder as **6B44**, its ICD-10 predecessor having been the reactive attachment disorder of childhood, F94.1. Where the neglect has taken the form of repetitive maltreatment such as chronic physical or sexual abuse, the child is additionally at risk of co-occurring post-traumatic stress disorder or complex post-traumatic stress disorder, so the same pathogenic care can generate a frank trauma disorder alongside the attachment disturbance. Two differentials recur in examinations:

CONDITION	ATTACHMENT BEHAVIOUR	THE DISCRIMINATING POINT
Reactive attachment disorder	Comfort neither sought nor accepted	Requires a history of pathogenic care and a developmentally excessive failure of selective attachment
Childhood depression	Comfort still sought and accepted	Reduced positive affect and withdrawal overlap, but the depressed child's attachment is intact; consider this disorder only with significant social neglect and absent comfort-seeking
Intellectual developmental disorder	Selective attachment forms with development	The child with intellectual disability forms selective attachments once a developmental age of about seven to nine months is reached, with social-emotional skills matching cognitive level

The two examination differentials of reactive attachment disorder; in both the pivot is whether attachment behaviour - the seeking and accepting of comfort - is preserved.

The depression differential is genuine because both conditions show blunted positive affect and withdrawal, so the discriminator must be the attachment behaviour itself: the depressed child still turns to a caregiver and is soothed, and the neurovegetative signs and excessive guilt of depression are not features of the attachment disorder. The intellectual disability differential matters because developmental delay so often accompanies neglect; the resolution is that a child whose social-emotional functioning tracks their cognitive level has delay rather than an attachment disorder, and this disorder is diagnosed only when the failure to attach is out of step with the child's developmental level.

Carry into the exam: reactive attachment disorder is the inhibited, withdrawn child who neither seeks nor accepts comfort, arising from a documented history of pathogenic care presumed to be its cause, evident before age five in a child of at least a nine-month developmental age, with autism excluded.

TRAP **The two attachment disorders are mirror images, and candidates reliably swap them.** Reactive attachment disorder is the internalising, inhibited, emotionally withdrawn pattern - the child who will not turn to an adult. Its disinhibited sibling, taught separately here, is the externalising, indiscriminately sociable pattern - the child who wanders off with any stranger. Both spring from the same pathogenic care, but a vignette describing an over-familiar, boundary-less child is not this disorder, and answering "reactive attachment disorder" to it is the classic error.

IN INDIA

Because Indian services classify under ICD, the operative label in case files and referrals is the ICD entity coded 6B44, and the clinician works from its clinical descriptions rather than a separate criteria manual. The pathogenic-care contexts the criteria describe are far from rare here: children reared in institutions with high child-to-caregiver ratios, and children moved repeatedly between placements before adoption, are exactly the settings Criterion C names, so the caregiving history must be documented carefully, because the diagnosis stands or falls on it. The deprivation evidence and the interventions themselves belong to the attachment-disorder management account elsewhere in this chapter.

<10%	Before 5	9 months	>12 months
OF NEGLECTED CHILDREN, EVEN WHEN NEGLECT IS SEVERE	YEARS: THE ONSET GATE	MINIMUM DEVELOPMENTAL AGE TO DIAGNOSE	DEFINES THE PERSISTENT SPECIFIER

26 · Disinhibited social engagement disorder

A child who never learned that a stranger is a stranger. **Disinhibited social engagement disorder** turns on one arresting behaviour: the child walks up to adults they have never met, engages them with warmth and physical closeness, and will wander off with them showing none of the wariness a securely reared child of the same age keeps in reserve for the unfamiliar. Its essential feature is a manner of **culturally inappropriate, overly familiar** conduct with relative strangers that violates the social boundaries the child's own culture sets for that age. It is the

outward, sociable, boundary-crossing face of early adversity, and it is the mirror image of the withdrawn, comfort-refusing picture of the other attachment disorder of childhood, **reactive attachment disorder**, which is developed in its own right elsewhere in this chapter.

The examiner's interest here is discriminative and conceptual. This is a disorder that looks, to an untrained eye, like nothing worse than a friendly temperament, and the marks reward the candidate who can explain why an approach that charms an adult is in fact a marker of disturbed early development, who can hold the picture apart from the general impulsivity of attention-deficit/hyperactivity disorder, and who can name the specific ways it parts company from its sibling attachment disorder. This topic teaches disinhibited social engagement disorder in full: its phenomenology, its diagnostic criteria, the caregiving history it requires, its epidemiology, its comorbidity and differential, its course and its place in the two classifications. Reactive attachment disorder itself, the comparison of both attachment disorders with autism spectrum disorder, and the management of attachment disorders and the effects of institutional care are each owned elsewhere and are named here only where the reasoning needs them.

26.1 The essential feature: indiscriminate sociability that crosses boundaries

The abnormality is not that the child is friendly, but that the friendliness has no discrimination in it. The healthy toddler is sociable too, but their sociability is calibrated: warmth for the familiar, a checking wariness for the stranger, and a caregiver used as a secure base from which to venture and to which to return. In this disorder that calibration is absent. The child treats the unfamiliar adult as though they were a trusted figure, with a lack of the reticence that age and culture would ordinarily impose. Crucially, and unlike its withdrawn sibling, this pattern does not require or imply a failure of attachment. It can appear in children whose attachments range from clearly disturbed to apparently secure, which is the single most counter-intuitive fact about the condition and the one an examiner most likes to test. The behaviour is a marker of a particular kind of early experience rather than a direct readout of the child's bond with any one caregiver, and the sign frequently persists even after other, more obvious markers of neglect have resolved.

GOOD TO REMEMBER

Because the disinhibited approach can coexist with a formed and even secure attachment, the fact that a fostered or adopted child is visibly bonded to the new caregiver does not exclude the diagnosis. Clinicians who reason "but the child is attached to the parents, so this cannot be an attachment disorder" miss it routinely. Watch how the child behaves toward the unfamiliar adult in the room, not only toward the parent.

PITFALL

The most consequential clinical error is to read the overfamiliarity as endearing charm and to reassure the family that the child is simply outgoing. The willingness to go off with an unknown adult is a genuine safeguarding concern, not a personality quirk, and framing it as sociability both misses the disorder and leaves a vulnerable child unprotected.

26.2 Criterion A: the behavioural signature

The diagnosis is built on a pattern of actively approaching and engaging unfamiliar adults. The child must show at least **two** of four characteristic behaviours, so that no single incident carries the diagnosis and the requirement is for a genuine pattern. The four are drawn from the same underlying failure of stranger-wariness expressed at different moments of an encounter.

- Reduced or absent reticence in approaching and interacting with unfamiliar adults;
- Overly familiar verbal or physical behaviour, out of keeping with culturally sanctioned, age-appropriate social boundaries;
- Diminished or absent checking back with the caregiver after venturing away, even in unfamiliar settings;
- Willingness to go off with an unfamiliar adult with minimal or no hesitation.

MNEMONIC

Approaches, over-Familiar, no Checking-back, goes Off willingly

The four behaviours track a single encounter with a stranger from start to finish: the child moves in without hesitation, is too familiar too fast, does not glance back to the caregiver for reassurance, and will leave with the stranger. Any two of the four, sustained as a pattern, satisfy this criterion.

26.3 Criterion B: the disinhibition is social, not merely impulsive

This criterion is what keeps the diagnosis from collapsing into general impulsivity. The behaviours must reflect **socially disinhibited** conduct and not be limited to the impulsivity seen in attention-deficit/hyperactivity disorder. A child who blurts, interrupts and acts without forethought across every domain is showing a broad behavioural impulsivity; the child captured by this disorder shows a specific loss of the social brake that governs approach to unfamiliar people. The distinction is drawn inside the criteria set itself precisely because the two can look alike at a glance and because they genuinely co-occur. Attention-deficit/hyperactivity disorder is developed as its own entity in the ADHD and specific learning/communication disorders notes; the point of Criterion B is only to insist that the disinhibition on display is of the social, stranger-directed kind rather than a general failure of impulse control.

26.4 Criteria C and D: the required history of pathogenic care

No amount of overfamiliar behaviour makes the diagnosis without the caregiving history behind it. The child must have experienced a pattern of **extremes of insufficient care**, evidenced by at least one of three circumstances: social neglect or deprivation, in the form of a persistent lack of having basic emotional needs met by caregiving adults; repeated changes of primary caregiver that prevent stable attachments from forming, as with successive foster placements; or rearing in unusual settings that severely limit the opportunity to form selective attachments, the institutional nursery being the archetype. The accompanying criterion requires that this care is presumed responsible for the disturbance, so that the abnormal behaviour is tied causally to the deprivation and not merely coincident with it. This pathogenic-care requirement is shared, word for word, with the sibling attachment disorder, and the effects of deprivation and

institutional rearing are themselves the subject of a separate topic in this chapter; here the history is named as the necessary etiological gate through which the diagnosis must pass.

26.5 Criterion E, and the two things this disorder does not require that its sibling does

Here the two attachment disorders separate on the page of criteria, not just in clinical feel. The developmental requirement is modest: the child must have a **developmental age** of at least **9 months**, which simply ensures the child is developmentally capable of the selective attachment whose failure the disorder describes. What is far more testable is what the criteria do NOT contain. This disorder carries no criterion demanding onset before a particular age, and it carries no exclusion for autism spectrum disorder, whereas the sibling attachment disorder's criteria include both an onset requirement and an autism exclusion. A candidate who imports those two conditions into this diagnosis from the neighbouring one is making the error the examiners write the question to catch.

TRAP **Importing the sibling disorder's onset and autism rules.** Because the two attachment disorders share their pathogenic-care requirement, candidates assume the rest of the criteria match too, and wrongly demand an onset-before-age-five and rule the diagnosis out in a child with autism. Neither belongs here: this disorder has no onset-age criterion and no autism-spectrum exclusion. The attachment-versus-autism discrimination itself is owned by a separate topic in this chapter.

26.6 The specifiers: persistent and severe

Two specifiers record chronicity and intensity. The disorder is designated **persistent** when it has been present for more than **12 months**, marking the presentation as an entrenched rather than a transient one, which matters because a durable course carries different prognostic weight. It is designated severe when the child exhibits all of the criterion behaviours, each expressed at relatively high levels, rather than the minimum two that satisfy the diagnosis. The specifiers are not idle bookkeeping: an examiner may use them to check that a candidate grasps that this is a dimensional, persisting phenomenon and not a one-off observation, and the persistence specifier in particular foreshadows the disorder's tendency to endure.

26.7 Epidemiology

Rare in the population, and uncommon even among the deprived children in whom it is sought. The general-population prevalence is unknown, and the disorder appears genuinely rare; it develops in only a minority even of children who have suffered severe deprivation, which tells us that pathogenic care is necessary but far from sufficient and that other, still poorly understood factors decide which deprived child goes on to show the syndrome. Where a figure exists it comes from high-risk sampling: in low-income community populations in the United Kingdom the prevalence reaches **up to 2 per cent**. That single anchoring number should be read for what it is, an estimate from an enriched, deprived sample rather than a base rate for children at large, and its very scarcity is part of the teaching, because the clinician who expects to see the disorder often will over-diagnose ordinary sociability.

26.8 Comorbidity

The company it keeps points back to a shared failure of inhibition. In younger children and through middle childhood the disorder often co-occurs with attention-deficit/hyperactivity disorder and with the externalising disorders more broadly, a pattern that has been read as evidence of a shared deficit in **cognitive inhibitory control**: the same weak brake that fails to hold back the socially disinhibited approach may also underlie the broader disruptive picture. Autism spectrum disorder may also co-occur, which is one reason the criteria deliberately omit an autism exclusion. Alongside these, the conditions that travel with early neglect, cognitive and language delays and stereotypies, may be present as well, reflecting the deprivation the child has lived through rather than the attachment disorder as such. The co-occurring conditions are named here, not taught; each is owned by its own topic.

26.9 Differential diagnosis: distinguishing it from ADHD with social impulsivity

The local differential that the criteria themselves anticipate is with attention-deficit/hyperactivity disorder. A child with that disorder and prominent social impulsivity can approach strangers and act without the usual social caution, superficially resembling the disinhibited attachment picture. The separating principle is that this attachment disorder does not, of itself, involve difficulties with attention or hyperactivity: its abnormality is confined to the social, stranger-directed disinhibition, whereas the impulsive child's approach sits within a pervasive inattentive and overactive syndrome. When the full requirements for both are met, both may be diagnosed, and comorbidity is common rather than exceptional. The broader comparison of the attachment disorders with autism spectrum disorder is a distinct discrimination, owned by another topic in this chapter and cross-referring the Intellectual disability and autism spectrum disorder notes for autism itself.

> Carry this into the exam: a friendly, boundary-crossing child who will go off with a stranger, in the setting of a history of extremes of insufficient care, has the externalising attachment disorder, and unlike its withdrawn sibling it may persist even in a markedly improved home.

26.10 Course and prognosis: the persistence that defines the disorder

The prognostic signature is a stubborn independence from the quality of later care. Where the sibling attachment disorder tends to recede once a child is placed with an adequate, responsive caregiver, this disorder does not reliably follow the caregiving environment. Its outcome is only modestly associated with the quality of the post-neglect home, and in many cases the behaviour persists even after that environment has become markedly improved. A further dissociation sharpens the picture: the disorder has not been identified in children who first experience social neglect only after the age of 2 years, which points to a sensitive early window and suggests that the core deficit is not simply the current absence of an attachment relationship but something laid down early and carried forward. This is why the diagnosis is not merely a description of a child's present home but a statement about an enduring trait.

RULE **Good caregiving reliably rescues the withdrawn sibling; it does not reliably rescue this one.** The single most examined contrast between the two attachment disorders is prognostic. Reactive attachment disorder's signs typically fade with adequate care, whereas the disinhibited pattern frequently persists despite a markedly improved caregiving environment. Treat the persistence as the diagnostic and prognostic fingerprint of the disinhibited disorder.

AXIS OF CONTRAST	REACTIVE ATTACHMENT DISORDER (NAMED FOR COMPARISON)	DISINHIBITED SOCIAL ENGAGEMENT DISORDER (THIS TOPIC)
Core behaviour toward others	Emotionally withdrawn, rarely seeks or responds to comfort	Indiscriminately sociable, over-familiar with unfamiliar adults
Emotional direction	Internalising	Externalising, disinhibited
Relation to the attachment bond	Reflects an absent or grossly underdeveloped attachment	Can occur across attachments from disturbed to secure
Response to improved caregiving	Signs typically recede with adequate care	Often persists despite markedly improved care
Onset-age and autism-exclusion criteria	Both present in its criteria set	Neither present

The two attachment disorders share their pathogenic-care requirement but diverge on direction, on the role of the attachment bond, and above all on whether adequate care resolves them; the disinhibited disorder is named for comparison here and taught in full only in its own right.

26.11 Classification: ICD-11, and a material difference over onset

Both major systems recognise the disorder, but they do not define it identically. In ICD-11 it is coded **6B45** and requires, as its essential features, a history of grossly insufficient care together with a persistent, pervasive pattern of reduced or absent reticence in approaching unfamiliar adults. The examinable divergence lies in onset: the ICD-11 description states that the symptoms are evident before the age of 5 years, whereas the DSM criteria impose no onset-age requirement at all and set only the modest developmental-age floor discussed above. It is held apart from autism by the indiscriminately social, over-familiar approach, the opposite of autism's social withdrawal, and by the absence of autism's restricted and repetitive behaviours. The predecessor category in ICD-10 was Disinhibited Attachment Disorder of Childhood, coded F94.2, a label a candidate will still meet in older Indian records.

IN INDIA

Two things anchor the topic to Indian practice. First, most services still diagnose and code under ICD, so the operative labels are the current 6B45 and the legacy F94.2 that persists in older files, and the onset-before-five wording of the ICD description becomes clinically live for a child who presents late out of long institutional care. Second, the population in whom this disorder is realistically sought, children reared with severely limited opportunity to form selective attachments, maps directly onto childcare institutions and orphanage settings, which makes the pathogenic-care history the first thing to establish. The deprivation evidence and the management response, including the primacy of a stable and sensitive caregiver, are developed in the attachment-disorder management topic in this chapter and are not reproduced here.

9 months	up to 2%	12 months	6B45
MINIMUM DEVELOPMENTAL AGE	PREVALENCE, LOW-INCOME UK COMMUNITY SAMPLE	THRESHOLD FOR THE PERSISTENT SPECIFIER	ICD-11 CODE

27 · Differentiation of RAD and DSED from ASD

A severely neglected child and an autistic child can look strikingly alike across a clinic room, and telling them apart is the whole of these marks. A young child brought from an institution or a grossly depriving home may be socially odd, emotionally flat, poorly related and slow to speak, and the first diagnosis that comes to an inexperienced mind is autism. The task this topic sets is the disciplined separation of the two attachment disorders of childhood, **reactive attachment disorder** and **disinhibited social engagement disorder**, from **autism spectrum disorder**. The examiner is not testing whether you can define each condition; the criteria of the two attachment disorders are developed in their own right elsewhere in this chapter, and autism spectrum disorder is taught in full in the Intellectual disability and autism spectrum disorder notes. What is tested here is the reasoning that keeps a deprived child from being mislabelled autistic, and, just as importantly, that keeps a genuinely autistic child from having every deficit written off as the effect of a difficult early life.

The comparison runs along a small number of axes, and each of them cuts cleanly if it is asked properly: the history of care, the presence or near-absence of selective attachment, the quality of social communication, the presence or absence of a restricted and repetitive repertoire, and, decisively, what happens when the child is placed in a good environment. Hold those five questions in mind and the differential resolves; miss the history and it does not. This fragment teaches only the attachment-side view of the boundary. It names reactive attachment disorder and disinhibited social engagement disorder without re-teaching them, and it points to the Intellectual disability and autism spectrum disorder notes for autism spectrum disorder itself.

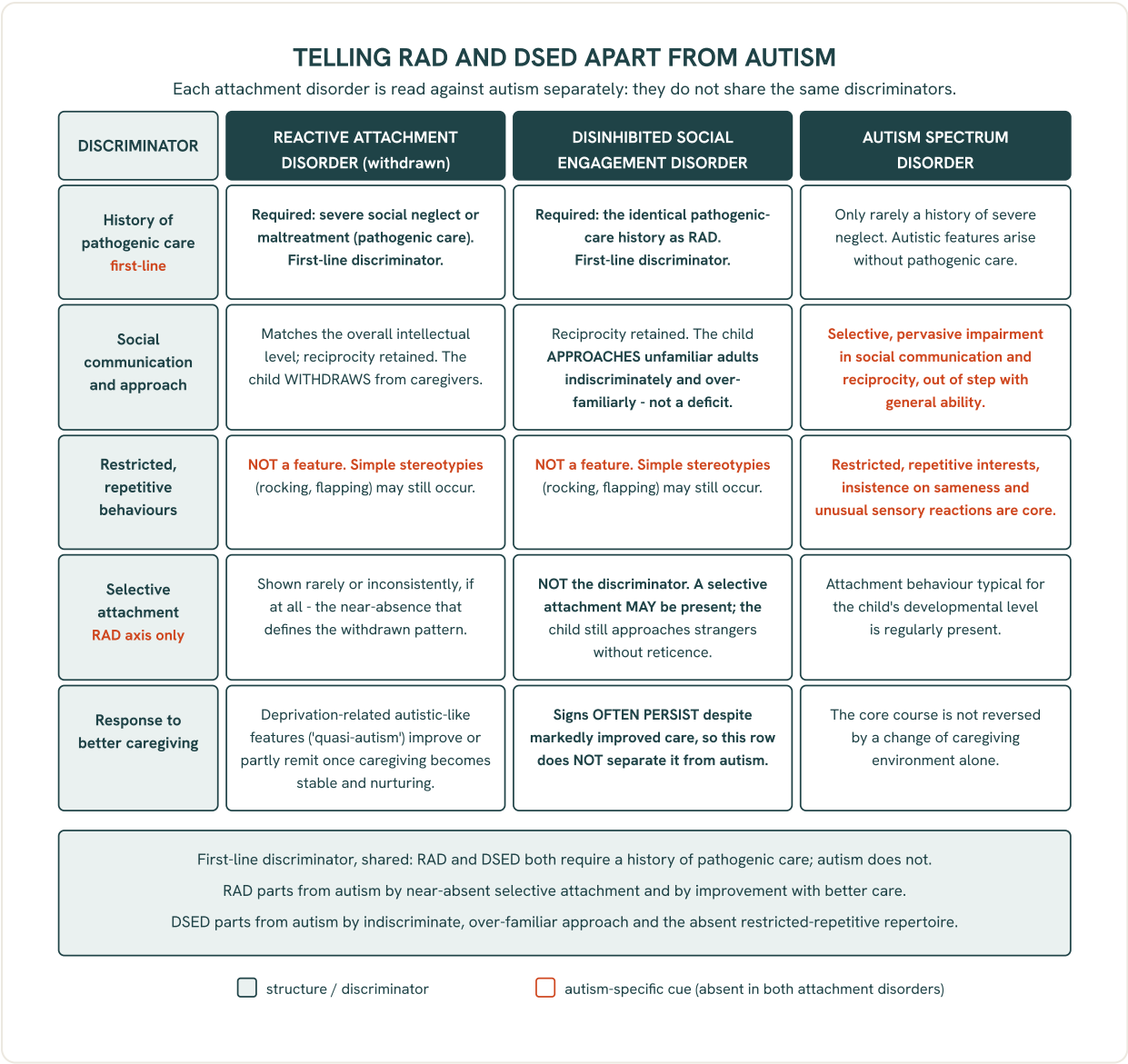

Fig 23.18. Telling reactive attachment disorder and disinhibited social engagement disorder apart from autism spectrum disorder, the attachment-side view, read separately for each disorder: the required history of pathogenic care is the first-line discriminator for both, while near-absent selective attachment and improvement of quasi-autistic features with better caregiving carry reactive attachment disorder, and indiscriminate, over-familiar social approach with preserved reciprocity carries the disinhibited disorder; the restricted-repetitive repertoire is absent in both; for autism spectrum disorder itself, see the Intellectual disability and autism spectrum disorder notes.

27.1 The required history of pathogenic care is the first-line discriminator

Begin with the history, because it is where the whole differential turns. The two attachment disorders are not free-standing behavioural pictures; each requires, as a diagnostic precondition, a documented **history of severe social neglect or maltreatment** - repeated changes of caregiver, institutional rearing, or a pattern of care that failed to meet the child's basic emotional needs. Autism spectrum disorder carries no such requirement and only rarely arrives with such a history. This asymmetry makes the care history the single most efficient opening question in the differential: an aberrant social presentation on a background of grossly inadequate care points first towards an attachment disorder, whereas the same presentation in a child whose early caregiving was adequate points away from it. The history does not, on its own, settle the diagnosis, because a maltreated child can also be autistic, but it sets the prior, and a candidate

who reaches for autism before taking the developmental and placement history has already made the classic mistake.

RULE **No pathogenic care, no attachment disorder; abundant pathogenic care, think of it first.** Reactive attachment disorder and disinhibited social engagement disorder both demand a history of grossly insufficient care as a precondition, while autism spectrum disorder does not. The care history is therefore the discriminator to establish before any other, and it reframes every social sign that follows.

27.2 Selective attachment behaviour: present in autism, scarce in reactive attachment disorder, beside the point in the disinhibited disorder

Autistic children do form attachments; the emotionally withdrawn attachment-disordered child largely does not. This is the point most candidates get backwards. Contrary to the old caricature of the autistic child as unattached, children with autism spectrum disorder regularly show **attachment behaviour typical for their developmental level** - they seek their parent when distressed, prefer familiar caregivers, and show the ordinary preferences of a bonded child, even if the social style around that bond is atypical. The child with reactive attachment disorder shows this selective, preferential attachment only rarely and inconsistently, if at all; the emotionally withdrawn pattern that defines that disorder is precisely a failure to turn to a discriminated attachment figure for comfort. Observing the child across separation and reunion, or simply watching whom the child goes to when hurt or frightened, is a practical way to elicit this contrast. The presence of developmentally expectable attachment is evidence for autism over reactive attachment disorder; its near-absence in a neglected child is evidence for the attachment disorder. Disinhibited social engagement disorder does not sit on this axis at all, and assuming that it does is the second reversal to avoid. The disinhibited child's difficulty is not a missing selective attachment but an indiscriminate, over-familiar approach to unfamiliar adults; such a child may hold an ordinary selective attachment to a caregiver and still go off with a stranger without reticence. Against autism, the disinhibited disorder is therefore separated by the direction and indiscriminateness of social approach, not by absent attachment.

27.3 Reciprocal social communication is selectively impaired only in autism

This is the axis that separates a delayed child from a deviant one. Autism is defined by a pervasive, selective deficit in **social-communicative behaviour** - impaired social reciprocity, deficits in non-verbal communication, and difficulty with relationships - that stands out against, and is disproportionate to, the child's general level of functioning. The child with reactive attachment disorder shows social communication that is broadly commensurate with their overall intellectual level: their language may be delayed by the deprivation itself, and their affect may be muted, but the capacity for reciprocal social interaction is retained rather than qualitatively deviant. In other words, the neglected child is often behind across the board, whereas the autistic child is specifically and disproportionately impaired in the social-communicative domain. The clinical move, therefore, is to ask whether the social deficit is out of step with the rest of development. If it is a selective peak of impairment, that favours autism; if the child's social relatedness tracks their general delay, that favours the attachment disorder. The disinhibited

disorder leaves reciprocal social communication intact in a way that is, if anything, more obvious: the disinhibited child approaches, talks to and engages unfamiliar adults readily and without reticence, which is the mirror image of the autistic child's reduced social approach and impaired reciprocity. Excessive, undiscriminating sociability is not a social-communication deficit, and it is the axis on which the disinhibited disorder parts from autism most cleanly.

27.4 The restricted, repetitive repertoire belongs to autism

The second pillar of autism is simply absent in the attachment disorders. Autism requires not only the social-communication deficit but a restricted, repetitive and inflexible repertoire. The **restricted interests and repetitive behaviours** that characterise autism are not a feature of reactive attachment disorder or disinhibited social engagement disorder. Concretely, what you look for and do not find in the pure attachment disorder is:

- excessive adherence to rituals and routines, with distress at small changes;
- restricted, fixated interests of abnormal intensity or focus;
- unusual sensory reactions - hyper- or hypo-reactivity to sounds, textures or lights.

A caveat prevents this axis from being used naively. Both attachment-disordered and institutionally reared children may show simple motor stereotypies such as body-rocking or hand-flapping, especially where stimulation has been sparse, and these can superficially mimic autistic mannerisms. A single stereotypy is not the restricted-repetitive-inflexible repertoire. What is specific to autism is the whole inflexible pattern - the insistence on sameness, the circumscribed interests and the sensory features together - not an isolated self-soothing movement that a barren environment has shaped.

27.5 Quasi-autism and the test of a nurturing environment

The clearest natural experiment in this whole differential is what a good home does to the picture. Some children reared under severe institutional deprivation develop a cluster of autistic-like features - difficulties in social reciprocity alongside restricted and repetitive behaviours - that is close enough to autism to be called **quasi-autism**. Here the static cross-sectional signs may genuinely overlap with autism, and the axes above can leave real doubt. The discriminator that does the work in these children is longitudinal, not cross-sectional: the deprivation-related picture shows significant improvement, or at least partial remission, when the child is moved into a stable, sensitive and nurturing environment, and that trajectory is what differentiates it from autism, in which the core features persist despite good caregiving. This is why placement in a good home is not only the treatment but part of the diagnostic test. A picture that lifts substantially with better care was never true autism; a picture that holds firm despite it is the real thing.

PITFALL

Overshadowing runs in both directions, and both are errors. The first is to see a socially odd, deprived child and diagnose autism at the first appointment, before the care history and before any period of stable placement has had a chance to reveal a reversible, quasi-autistic picture. The second, subtler, error is to assume that because a child was maltreated every deficit must be attachment-related, and so to miss a genuinely co-occurring autism that will not remit with a good home and needs autism-specific support in its own right. Neglect and autism can coexist; a maltreatment history does not exclude the neurodevelopmental diagnosis.

27.6 Bringing the axes together at the bedside

No single sign is pathognomonic; the diagnosis is the convergence of the axes plus the history plus the course. The efficient examination answer moves through the discriminators in order - the care history first, then attachment behaviour, then whether the social deficit is selective or part of a global delay, then the presence or absence of the restricted-repetitive repertoire, and finally the response to enhanced caregiving over time - and reads them together rather than trusting any one in isolation. Note which axes carry which disorder. The care history carries both attachment disorders. Absent selective attachment carries only reactive attachment disorder. The course axis carries reactive attachment disorder and quasi-autism, and it does not separate the disinhibited disorder from autism, because the signs of the disinhibited disorder often persist despite markedly improved care. What separates the disinhibited disorder from autism is the phenotype itself: an indiscriminate, over-familiar approach to strangers is the opposite of an autistic deficit in social approach and reciprocity, and the restricted, repetitive repertoire is absent. The accompanying figure lays out the same contrast so it can be recalled at a glance.

DISCRIMINATING AXIS	REACTIVE ATTACHMENT DISORDER	DISINHIBITED SOCIAL ENGAGEMENT DISORDER	AUTISM SPECTRUM DISORDER
History of care	Severe neglect or maltreatment required as a precondition	The same pathogenic-care history required	Only rarely a history of pathogenic care
Selective attachment	Rare, inconsistent or absent	Not the discriminator; a selective attachment may be present	Present and typical for developmental level
Social communication	Commensurate with overall intellectual level; capacity for reciprocity retained	Reciprocity retained; social approach is indiscriminate and overfamiliar rather than deficient	Selectively and disproportionately impaired
Restricted, repetitive repertoire	Not a feature; isolated stereotypies may occur	Not a feature; isolated stereotypies may occur	Present - rituals, fixated interests, sensory features
Course with a nurturing environment	Improves; deprivation-related features can remit	Signs often persist despite improved care; this axis does not discriminate it from autism	Core features persist despite good caregiving

The attachment-side comparison across the five axes that carry the differential, read separately for each attachment disorder; the care history discriminates both from autism, the attachment and course rows carry reactive attachment disorder, and the disinhibited disorder is separated from autism by indiscriminate social approach and the absent restricted-repetitive repertoire.

TRAP **Reversing the attachment axis.** The examination favourite is the belief that the autistic child is the one who cannot attach. It is the reactive attachment disorder child who fails to show selective attachment; the autistic child usually attaches to caregivers in a developmentally typical way. State the direction correctly and this half of the question is won.

Carry this into the exam: a required history of pathogenic care, plus improvement of autistic-like features once the child is in a stable and nurturing home, points to an attachment disorder or quasi-autism, not to autism spectrum disorder.

GOOD TO REMEMBER

Do not force the diagnosis at a single visit for the deprived, socially atypical young child. Establish the care history, observe selective attachment directly through separation and reunion, judge whether the social deficit is selective or part of a global delay, and then let time in a stable placement do its diagnostic work. If the picture lifts with good care, it was on the attachment-and-deprivation side; if it holds firm, take the autism diagnosis seriously and arrange autism-specific assessment and support.

IN INDIA

The differential is unusually live in Indian practice because institutional and multiply-disrupted care is not rare: children present from orphanages, children's homes and severely deprived family settings, and many are seen first around adoption or foster placement. Two practical points follow. Most services diagnose under ICD, which frames both the attachment disorders and the quasi-autism boundary, and this favours a longitudinal view - reassessing after the child has spent time in a settled home rather than fixing an autism label at intake. And because language delay in these children so often reflects deprivation and a multilingual environment rather than a communication disorder, the social-communication axis must be judged against the child's whole developmental level, not against a single clinic-room silence. The full assessment battery and the services and protection pathways for such children sit in the Child psychiatry: assessment, treatment, services and protection notes.

History	Attachment	Repertoire	Course
SEVERE NEGLECT REQUIRED IN THE ATTACHMENT DISORDERS, ONLY RARELY PRESENT IN AUTISM	DEVELOPMENTALLY TYPICAL IN AUTISM, SCARCE IN REACTIVE ATTACHMENT DISORDER	RESTRICTED AND REPETITIVE ONLY IN AUTISM, NOT IN THE ATTACHMENT DISORDERS	QUASI-AUTISM IMPROVES WITH A NURTURING HOME; AUTISM PERSISTS

28 · Management of attachment disorders and effects of institutional care

This is the one childhood disorder whose treatment is a person, not a prescription. The two attachment disorders of early childhood, the emotionally withdrawn reactive attachment disorder and the indiscriminately sociable disinhibited social engagement disorder, both arise from the same soil: grossly insufficient care in the years when a child should be forming a selective attachment to one or two committed adults. Those two syndromes are described in full in their own right elsewhere in this chapter; what this topic owns is the ground beneath them - the effects of institutional rearing and early deprivation - and the management that follows from it. The marks here are conceptual and safety-critical at once. An examiner is checking that you understand the intervention is psychosocial and relational rather than pharmacological, that the two disorders respond differently to a good caregiver, and that the coercive "attachment therapies" sometimes marketed to desperate families are not merely unproven but have killed children.

The etiological premise is a form of **pathogenic care**: care so deficient, or so repeatedly interrupted, that the child never has the stable, responsive adult a secure attachment requires. Institutional rearing is the purest natural model of this, and it is why the entire evidence base rests on children raised in institutions and then placed in families. The requirement that pathogenic care precede the diagnosis is the etiological context the two disorder sections depend on; here it is the starting point for everything that follows.

28.1 The rearing that produces the two patterns

Two divergent behaviour sets emerge from the same deprivation. The observational basis for the modern classification is old and instructive. In **Barbara Tizard**'s study of institution-reared children whose caregivers had been explicitly instructed not to form attachments with them, of

26 such children, 8 nonetheless formed secure attachments, 8 were emotionally withdrawn and unresponsive in the pattern we now call reactive attachment disorder, and 10 were indiscriminately social with unfamiliar adults in the pattern we now call disinhibited social engagement disorder. Earlier still, Rene Spitz's descriptions of anaclitic depression in institutionalised infants foreshadowed the withdrawn phenotype. The lesson embedded in those numbers is that deprivation is a probabilistic exposure, not a deterministic one: even under an instruction not to bond, roughly a third of the children found an attachment anyway, which is the first clue that resilience and the availability of even one responsive adult matter enormously.

28.2 Timing and dose: the developmental window

The longer and the earlier the deprivation, the greater the harm. Risk is graded by both the duration of deprivation and the age at which it occurs. Attachment disorders are very rare in children adopted **before 6 months** of age, and signs of disinhibited social engagement have not been reported in children whose deprivation began only after the age of two years, marking a sensitive early period during which the developing attachment system is most vulnerable. Brief institutional care leaves children resembling their never-institutionalised comparison groups on most measures, and the neurophysiological differences seen in deprived children are no longer detectable at age eight if the child is placed with a family before twenty-four months. Yet the potency of prolonged deprivation is sobering: only **about 15%** of children adopted after at least six months of institutional rearing in Romania were free of any diagnosable disorder at any point into their mid-twenties.

- duration of deprivation - the longer the child lives in it, the more likely the disorders;
- age at onset - the early months carry a sensitive window, and disinhibited signs are tied to deprivation that begins early;
- age at placement - earlier rescue is associated with greater reversibility of the measurable deficits.

28.3 The natural experiment: the two disorders diverge under a good caregiver

Foster care turned into a randomised trial is what proves the point. The strongest evidence comes from the **Bucharest Early Intervention Project**, a prospective randomised controlled trial of high-quality foster care as an alternative to continued institutional care. Children with reactive attachment disorder randomised to foster families showed a reduction in symptoms within months of placement, falling to the levels of a community sample of never-institutionalised children. Children with disinhibited social engagement disorder in the same trial continued to show behaviours consistent with the diagnosis after placement, typically improving but not fully resolving. The **English and Romanian Adoptees study** converges on the same split: Romanian children adopted into English families no longer manifest reactive attachment disorder but continue to show persistent signs of disinhibited social engagement. The interpretation is the heart of the topic. Reactive attachment disorder behaves like a condition of missing attachment, so it lifts once a real attachment forms; disinhibited social engagement

behaves like something more enduring, whose core deficit is not simply the absence of an attachment relationship.

AXIS	REACTIVE ATTACHMENT DISORDER	DISINHIBITED SOCIAL ENGAGEMENT DISORDER
Response to a stable, sensitive caregiver	Symptoms reduce toward those of never-institutionalised community children	Signs reduce but commonly persist rather than resolve
Persistence after adoption	Does not continue once an attachment forms	Can persist in a minority even after attachments to the new family form
What this implies about the deficit	Essentially a lack-of-attachment state, remediable by the relationship	Not reducible to a missing attachment; a more enduring social-behavioural disturbance

The two disorders share a cause but not a prognosis under treatment; the differential response to a good caregiver is itself diagnostic teaching.

TRAP **Assuming both disorders remit fully once the child is placed in a loving home.** They do not. Reactive attachment disorder largely resolves with a positive caregiving environment, but disinhibited social engagement signs frequently persist even after the child has clearly formed attachments to the new caregivers. A candidate who writes that placement cures both has missed the single most examined discrimination in this topic.

28.4 The long reach of early deprivation

The effects outlast childhood and broaden well beyond attachment. Follow-up of the English and Romanian adoptees into **early adulthood, at ages 22 to 25**, found that those with longer institutional care before adoption had lower educational achievement, higher unemployment, and greater use of mental-health services. Prolonged deprivation was also associated with high rates of attention-deficit and autistic-type features and of callous-unemotional traits, and self-reported emotional symptoms actually rose from adolescence into adulthood rather than fading. This tells the clinician that institutional deprivation is not a narrow attachment problem but a broad neurodevelopmental and psychiatric risk exposure, and that some of its consequences declare themselves only years after the child is safe and settled. Where a deprived child later carries attention or behavioural difficulties, those are treated on their own terms; the treatment of the general attention-deficit disorder and its medication is the ADHD and specific learning/communication disorders notes'.

PITFALL

The recurring clinical error is to attribute every later difficulty in a post-institutional child to "attachment", and so to miss a comorbid, treatable disorder. A previously deprived child with genuine attention-deficit hyperactivity or autistic features needs that condition assessed and managed directly, not reframed as an attachment wound. Over-psychologising the history leaves an eminently treatable comorbidity untreated.

28.5 Management, first principle: secure the child's safety

Nothing else begins until the child is safe. Because these disorders arise from maltreatment or grossly deficient care, the first step of management is to assess and secure the child's safety, which includes medical referral for the consequences of neglect, involvement of child protective services where warranted, and an assessment of parental fitness. This is not bureaucratic sequencing: child maltreatment carries significant morbidity and non-trivial mortality, particularly in the youngest children, so establishing safety is genuinely the intervention that comes before all others. Only once safety is established does full attention turn to the psychosocial work of building an attachment.

IN INDIA

Institutional rearing remains a live reality here, with large numbers of children in child-care institutions, and national policy increasingly favours family-based alternatives - kinship care, foster care and adoption - over long-term institutionalisation. The statutory machinery for protection and placement is not taught here; that framework belongs to the Child psychiatry: assessment, treatment, services and protection notes. Two cautions for the exam. First, no Indian general-population prevalence figure for either attachment disorder is established, so quote none: the evidence base is Western institutional and adoption cohorts. Second, the same policy shift the evidence supports, moving children out of institutions and into responsive families, is exactly what the Romanian and English data would predict helps most.

28.6 The core intervention: a stable, sensitive caregiver

Everything effective in this topic reduces to one relationship. The central, evidence-informed intervention for both disorders is to provide and support a primary attachment figure who is sensitively responsive, who values the child as an individual, and who places the child's needs ahead of their own. For reactive attachment disorder this is frequently sufficient in itself: the symptoms generally resolve once the child is in a family that offers a genuinely positive caregiving environment. For disinhibited social engagement disorder, clinical consensus adds a further, face-valid but formally unevaluated measure - limiting the child's unnecessary contact with unfamiliar adults for several months so that a selective attachment to the new caregivers has room to consolidate before the child's sociability is diffused across everyone they meet.

RULE **The treatment is a committed, sensitive caregiver, and the relationship itself is the active ingredient.** Interventions work to the extent that they install and support one responsive adult who consistently reads and meets the child's needs; techniques that do not serve that end do not treat the disorder. This is why placement quality, not any specific therapy, is the strongest lever available.

GOOD TO REMEMBER

Once the child is safe, the most powerful thing a clinician can arrange is stability of caregiving - one committed adult, protected from disruption and repeated moves, over a long horizon. Placement breakdown and a revolving door of carers reproduce the original injury. Supporting the caregiver so the placement holds is itself the treatment, not a preliminary to it.

28.7 Dyadic therapies, medication, and the interventions to refuse

Add structured dyadic work, keep medication in its lane, and never coerce. Where more than placement is needed, the attachment-specific treatments focus on the **adult-child dyad** and include the Circle of Security, Attachment and Biobehavioral Catch-Up, and child-parent psychotherapy. These combine developmental guidance with review of videotaped parent-child interaction and attend to instrumental problems such as housing and medical care; long-term intervention is often necessary. Pharmacotherapy has no role in the core attachment disorder itself: the standard account describes management as entirely psychosocial, and no medication is directed at reactive attachment disorder or disinhibited social engagement disorder as such, with any drug treatment reserved for a co-occurring condition rather than the attachment disorder. Finally, the discriminating clinician must actively refuse a category of harm: the **coercive "attachment therapies"** - holding and restraint procedures, "rebirthing" and the like - are discredited by the 2016 practice parameters of the American Academy of Child and Adolescent Psychiatry and have caused the deaths of children. Management is the sensitive-caregiver and dyadic model, never coercion.

MNEMONIC

Secure, Attach, Support, Refuse

Secure the child's safety first; build the primary **A**ttachment through one sensitive caregiver; **S**upport that dyad with manualised therapy and treat comorbidity only; **R**efuse coercive holding and restraint "therapies" outright.

The active treatment is a stable, sensitively responsive caregiver: reactive attachment disorder largely resolves with it, disinhibited social engagement disorder only partly, medication treats only comorbidity, and coercive holding therapies are never used and have killed children.

about 15%	before 6 months	22 to 25	26
OF LONG-INSTITUTIONALISED ROMANIAN ADOPTEES FREE OF ANY DISORDER INTO MID-TWENTIES	ADOPTION AGE BELOW WHICH ATTACHMENT DISORDERS ARE VERY RARE	AGE AT WHICH ADULT ADOPTEE OUTCOMES WERE ASSESSED	INSTITUTION-REARED CHILDREN IN THE FOUNDING BEHAVIOURAL OBSERVATION

Tic disorders and Tourette syndrome

29 · Classification of tic disorders (transient, chronic, Tourette)

Classification here is made almost entirely by the clock. A child brought in for repetitive blinking, sniffing or throat-clearing does not arrive with a diagnosis attached; what turns a description of movements into one of several named disorders is a small set of rules about how long the tics have lasted, whether they are motor, vocal or both, and whether an earlier tic diagnosis has already been made. Those rules are worth a disproportionate share of the marks,

because they are simple to state, easy to half-remember, and endlessly examinable: a single-word change, "and" to "or" or "less than" to "more than", moves the child from one diagnosis to another.

This section owns the classificatory scaffold: what counts as a tic at all, how simple tics are separated from complex ones, the full set of diagnostic categories, the duration and modality rules that divide them, the type specifier, and the hierarchy that governs which label a child may hold. The full diagnostic criteria and phenomenology of Tourette's disorder, the neurobiology, the comorbidity pattern with attention-deficit/hyperactivity disorder and obsessive-compulsive disorder, the management of tics, and the differentiation of tics from other abnormal movements are each developed in their own right elsewhere in this chapter; here they are named where the classification touches them, not reopened.

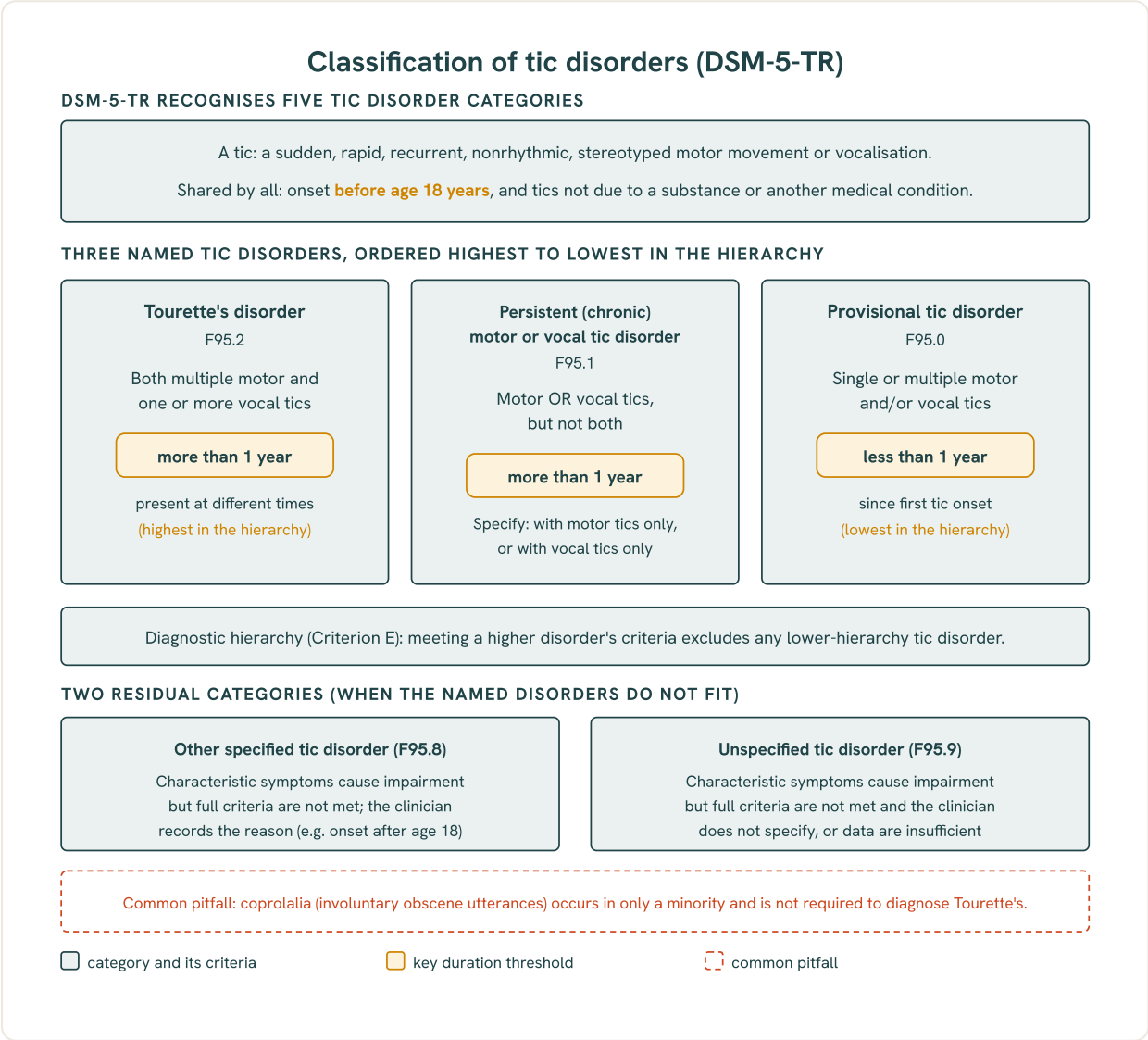

Fig 23.19. Classification of tic disorders: the three named DSM-5-TR disorders ordered by hierarchy - Tourette's disorder (both motor and vocal tics), persistent (chronic) motor or vocal tic disorder, and provisional tic disorder (less than 1 year) - together with the two residual categories and the coprolalia pitfall.

29.1 What counts as a tic

Everything downstream rests on one definition. A **tic** is a sudden, rapid, recurrent, nonrhythmic, stereotyped motor movement or vocalisation. Each word does work. *Sudden* and *rapid* mark the tic as fast and abrupt, unlike the slow writhing of a dystonic movement; *recurrent*

and *stereotyped* capture that the same movement repeats in a fixed form; *nonrhythmic* separates it from tremor, which is regular and oscillatory. The same definition covers both the motor and the vocal forms: a vocal tic is simply a tic whose effector is the respiratory and laryngeal apparatus, which is why a sniff, a grunt or a throat-clear is classified alongside a blink or a shrug rather than treated as something categorically different. Two further features, which sit in the phenomenology rather than the bare definition, make tics recognisable at the bedside and are developed with the Tourette phenomenology elsewhere in this chapter: tics are typically preceded by an uncomfortable premonitory urge and are, for a time, voluntarily suppressible. Naming them here is enough; the classification does not turn on them.

29.2 Simple and complex tics, and the taboo phenomena

Within both the motor and vocal channels, tics run from the fragmentary to the orchestrated. **Simple tics** are brief and meaningless: simple motor tics such as eye-blinks, facial grimaces and shoulder shrugs, and simple vocal tics such as throat-clearing, sniffing and grunting. **Complex tics** are longer, more elaborate and can look purposeful, which is exactly what makes them hard to read: complex motor tics include echopraxia (imitating another's movements) and copropraxia (obscene gestures), and complex vocal tics include palilalia, echolalia and coprolalia. The three "-lalia" terms are constantly confused and constantly examined, so fix them: **echolalia** is repeating the last word or sound one has just heard, **palilalia** is repeating one's own sounds or words, and **coprolalia** is the involuntary uttering of obscene or taboo words. Coprolalia is the feature the public most associates with Tourette's, and that association is the source of the single most common misconception in the topic, because it is present in only a minority and is required for no tic diagnosis at all.

TRAP **Coprolalia is neither necessary nor common.** The examination reflex, and the lay assumption, is that a diagnosis of Tourette's requires the patient to swear involuntarily. It does not. Coprolalia appears in only a minority of those affected and is a feature of no tic disorder's criteria. A candidate who makes coprolalia a requirement, or who withholds a tic diagnosis because it is absent, has mistaken a striking piece of phenomenology for the definition.

29.3 The diagnostic categories, and the logic that orders them

One organising idea makes the whole scheme memorable. The current DSM-5-TR classification recognises **five** tic-disorder categories: Tourette's disorder; persistent (chronic) motor or vocal tic disorder; provisional tic disorder; and the two residual categories, other specified and unspecified tic disorder. Which of the first three a child receives is decided by three things: the duration of the tics, their modality (motor, vocal or both), and whether an earlier tic diagnosis has already been made. Duration draws the line between the short-lived and the persistent; modality draws the line between the single-channel disorder and Tourette's; and the diagnostic history, through the hierarchy rule, prevents a child from sliding down into a lesser label. Hold that trio and the categories almost derive themselves; memorise the boxes without the logic and they blur together in exactly the way examiners rely on.

MNEMONIC

Time, then modality, then history

Read the three deciding questions in order. **Time:** have the tics lasted under a year or beyond it? Under a year and the label is provisional; beyond it and the disorder is one of the persistent forms. **Modality:** among the persistent forms, is only one channel involved, or are both? One channel is persistent (chronic) motor or vocal tic disorder; both is Tourette's. **History:** the hierarchy then forbids demoting a child who already qualifies for a higher category. Three questions, in that order, and every case lands in exactly one box.

29.4 Provisional tic disorder: the transient category

This is the diagnosis for tics that have not yet earned a chronic label. Provisional tic disorder is diagnosed when a child has single or multiple motor and/or vocal tics that have been present for **less than a year** since the first tic appeared, with onset in childhood, not attributable to a substance or another medical condition, and in a child who has never met criteria for Tourette's or a persistent tic disorder. The word "provisional" is deliberately provisional: at diagnosis one cannot know whether the tics will fade in a few months, as most childhood tics do, or persist past the year and convert the diagnosis into a chronic one. The older literature, and current Indian practice, call this same picture *transient tic disorder*, and the change of name matters: "transient" pre-judged an outcome knowable only in retrospect, whereas "provisional" states honestly that the diagnosis is held pending the passage of time. The motor-only or vocal-only specifier is not applied here; that distinction belongs only to the persistent disorder.

29.5 Persistent (chronic) motor or vocal tic disorder, and its specifier

The defining move here is the exclusive "or". Persistent (chronic) motor or vocal tic disorder is diagnosed when a child has had tics of one modality only, motor tics or vocal tics but not both, which may wax and wane in severity yet have persisted for **more than a year** since the first tic, again with childhood onset, not due to a substance or medical condition, and in a child who has never met criteria for Tourette's. The most consequential words in the whole classification are that "not both": the presence of even one vocal tic in a child who also has motor tics, once the year has passed, moves the diagnosis out of this category and into Tourette's. Because the disorder is restricted to one channel, it carries a required **type specifier**, either "with motor tics only" or "with vocal tics only", recording which channel is involved. That specifier exists only here; it is meaningless for Tourette's, where both channels are present by definition, and for the provisional disorder, where the picture is still unsettled.

29.6 Tourette's disorder at the apex

Tourette's is the category defined by the presence of both channels. Tourette's disorder sits at the top of the tic-disorder classification, and the feature that places it there is the requirement for both multiple motor tics and at least one vocal tic over the course of the illness, though the two need not be present at the same time. That both-modalities rule is the whole of what the classification needs from Tourette's; the full criteria, the characteristic waxing-and-waning course, the premonitory urge and the rest of the phenomenology are developed in their own right

elsewhere in this chapter and are not restated here. What matters for classification is the contrast with the category beneath it: where the persistent motor or vocal tic disorder is single-channel by rule, Tourette's is dual-channel by rule, and a child accrues the Tourette's label the moment both a motor and a vocal tic have been present over the required span of time. The coprolalia the public expects plays no part in this; a child with only simple motor and vocal tics and no obscene utterance whatsoever still has Tourette's once both channels and the duration are satisfied.

29.7 The diagnostic hierarchy and the one-way rule

The categories are not a menu; they are a ladder. The tic-disorder diagnoses are **hierarchical**, running from Tourette's at the top, through persistent (chronic) motor or vocal tic disorder, to provisional tic disorder, with the two residual categories beneath. The operative rule, written into each disorder's criteria as its exclusion clause, is that once a child has ever met criteria for a disorder higher in the hierarchy, a lower diagnosis can no longer be made. A child who has qualified for Tourette's cannot later be re-labelled with a persistent single-channel disorder simply because, at one review, only motor tics happen to be active; the higher diagnosis, once earned, holds. This is why the diagnostic history is the third deciding question and not an afterthought: the classification is cumulative and one-directional, and it deliberately resists the temptation to downgrade a child to a milder-sounding label during a quiet spell.

RULE **Duration and modality assign the category; the hierarchy protects it.** Under a year of tics is provisional; beyond a year it is one of the persistent forms; one channel among those is persistent motor or vocal tic disorder and both channels is Tourette's. The hierarchy then adds a one-way lock: a diagnosis once met at a higher level cannot be exchanged for a lower one. Every genuinely difficult classification question in this topic is testing one of those two mechanisms.

29.8 The residual categories, the whole scheme in one view, and the boundaries

The two residual categories catch what the specific ones cannot. **Other specified tic disorder** is used when a tic presentation causes clinically significant distress or impairment but does not meet the full criteria for any specific tic disorder, and the clinician chooses to record the particular reason (for example an onset after the childhood window). Unspecified tic disorder is used for the same sub-threshold situation when the clinician does not state the reason, or when there is insufficient information to do so. Both exist so that a real, impairing tic problem that sits outside the neat boxes still has a home in the record rather than being forced into a category it does not fit.

CATEGORY	TICS REQUIRED	DURATION	TYPE SPECIFIER
Provisional tic disorder	Motor and/or vocal	Less than a year	Not applied
Persistent (chronic) motor or vocal tic disorder	Motor *or* vocal, not both	More than a year	Motor only / vocal only
Tourette's disorder	Both motor and vocal	Long-standing	Not applied
Other specified / unspecified	Sub-threshold or atypical	Any	Not applied

The three specific categories read across duration and modality, with the residual pair holding the sub-threshold and atypical presentations; the accompanying figure lays the same decision out as its three ordered questions.

Two boundaries complete the picture without belonging to the classification proper. The first is with the other abnormal movements a tic must be told apart from before it even enters this scheme, because only a genuine tic is classified at all. The discriminating features are developed in their own right elsewhere in this chapter; here they are simply named:

- the stereotypies of stereotypic movement disorder and of autism spectrum disorder;
- chorea and the other hyperkinetic movement disorders;
- dystonia; and
- the compulsions of obsessive-compulsive disorder.

The second boundary is with normal childhood. Transient tics are common in school-age children and frequently resolve without ever crossing into a disorder, so the categories describe the impairing or persistent minority, not every child who blinks or clears the throat.

PITFALL

The commonest clinical error is to reach for a chronic-sounding label too early. A child seen a few weeks into a bout of blinking has, by definition, a provisional picture, and naming it Tourette's or a persistent disorder before the year has elapsed both overstates the prognosis to an anxious family and, because of the one-way hierarchy, is hard to walk back. The opposite error is to treat every fleeting tic as pathological, when brief tics are a common and usually self-limiting part of childhood. The clock, not the severity of a single clinic snapshot, decides the category.

IN INDIA

Routine Indian services classify under ICD rather than the manual these criteria are drawn from, and candidates should move comfortably between the two. The ICD scheme carries the same three-part structure under its own headings: a transient category for short-lived tics, a chronic motor or vocal category for the single-channel persistent form, and a combined category corresponding to Tourette's, so the logic of duration and modality transfers directly. The chief translation to remember is that the "provisional" label maps onto the older, and still widely used, term "transient tic disorder"; a child recorded as provisional under one system and transient under the other is the same child, filed by two manuals that agree on the substance.

GOOD TO REMEMBER

The category you assign changes the conversation you have. A provisional diagnosis is an occasion for measured reassurance, because most such tics remit; a persistent or Tourette's diagnosis reframes the problem as a longer-term condition whose burden usually comes less from the tics themselves than from the co-occurring attention and obsessional problems taken up elsewhere in this chapter. Assign the category by the rules, but let it, and not the alarming look of a bad spell of tics, set the tone of what you tell the family.

The one line to carry: duration sets provisional (under a year) against the persistent forms (over a year), modality sets single-channel persistent tic disorder against dual-channel Tourette's, and the hierarchy locks a higher diagnosis in place once it has been met.

Five	Under 1 year	Over 1 year	Both channels
TIC-DISORDER CATEGORIES	PROVISIONAL TIC DISORDER	PERSISTENT (CHRONIC) FORM	REQUIRED FOR TOURETTE'S

30 · Tourette syndrome diagnostic criteria and phenomenology

Tourette syndrome is the most recognisable and, in the public imagination, the most caricatured of the tic disorders, which is exactly why the exam tests whether a candidate can strip the caricature away and diagnose it on its actual rules. **Tourette's disorder** is a childhood-onset neurodevelopmental condition defined by the coexistence, at some point in the illness, of multiple motor tics and at least one vocal tic. A **tic** is a sudden, rapid, recurrent, non-rhythmic motor movement or vocalisation, and the whole diagnosis turns on recognising these movements for what they are and then applying four spare criteria that are almost entirely about pattern and time rather than about any single dramatic symptom. The marks here are not won by knowing that the condition exists; they are won by the discriminations - both tic types against one, the duration clock, the age ceiling - and by the phenomenology of the tic itself, which is what separates a tic disorder from every other movement disorder a child can present with.

This account owns the diagnostic criteria of Tourette's disorder and the phenomenology of tics: the criteria in full, the anatomy of simple and complex tics, the premonitory urge, the waxing-and-waning course, suppressibility and the factors that modify tics, the epidemiology across childhood, the trajectory into adult life and its suicide risk, and the standardised measurement of tic severity. The taxonomy of the tic disorders as a family - provisional, persistent and Tourette's - is set out in the classification account elsewhere in this chapter; the etiology and neurobiology, the pattern of comorbidity with attention-deficit/hyperactivity disorder and obsessive-compulsive disorder, the management of tics, and the formal differentiation of tics from stereotypies, chorea, dystonia and compulsions each belong to their own accounts here and are named only where the reasoning demands.

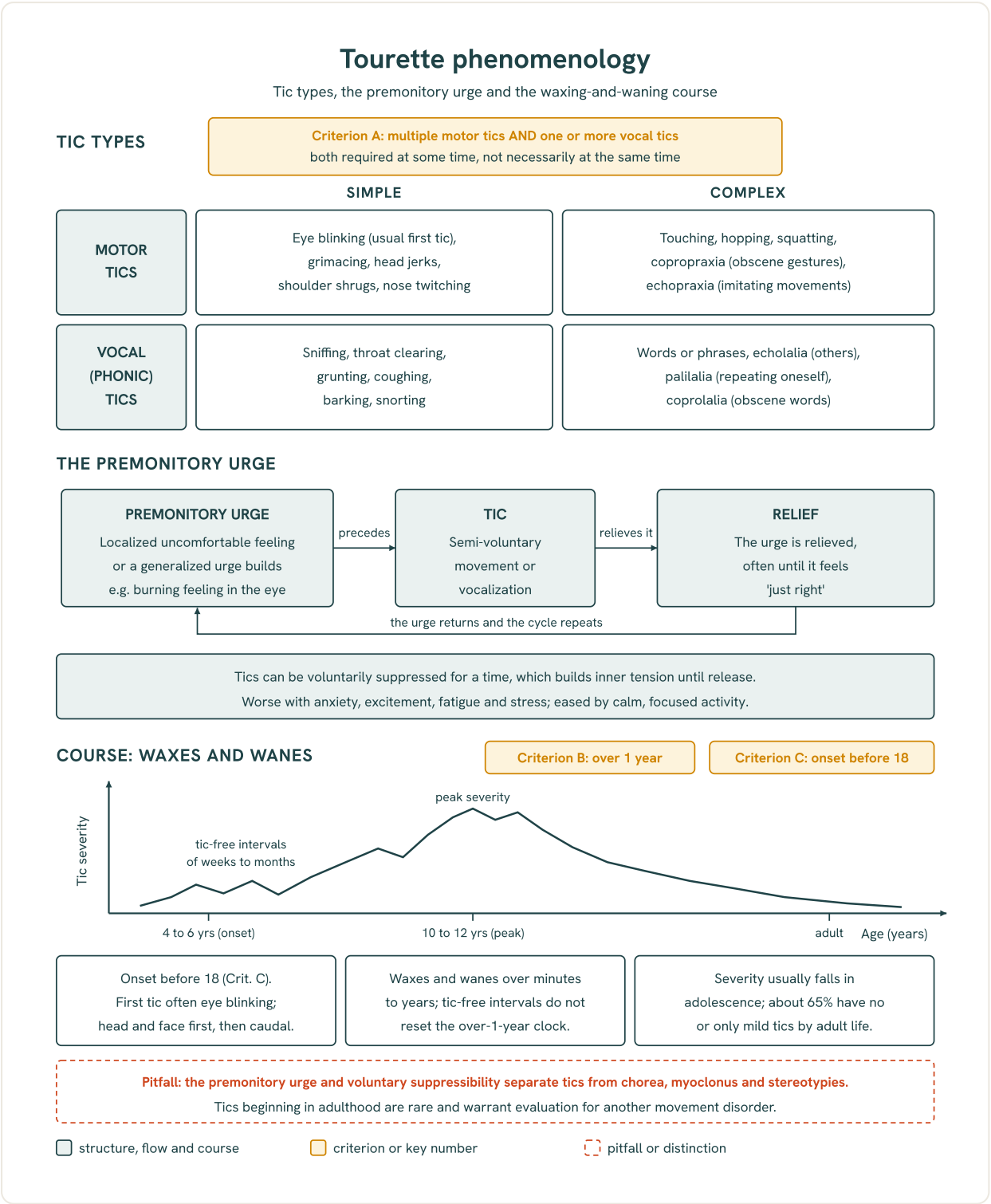

Fig 23.20. Tourette phenomenology: motor and vocal tics in simple and complex forms, the premonitory-urge-to-relief cycle, and the waxing-and-waning course from onset through peak severity to adult life.

30.1 Criterion A: both motor and vocal tics, and why that is the whole game

The single fact that makes a tic disorder Tourette's is the presence of both kinds of tic.

Criterion A requires multiple motor tics and one or more vocal tics, and the crucial rider is that they must have been present at some time during the illness, though not necessarily concurrently. This is the discriminator that separates Tourette's from persistent (chronic) tic disorder, in which a child has motor tics only or vocal tics only but never both. A child may have had years of motor tics and only later develop a single throat-clearing or sniffing vocal tic; the moment both types

have been present, even if they never appeared in the same week, the diagnostic threshold for Tourette's is crossed. The corollary matters clinically: a boy currently showing only motor tics may still warrant the Tourette's label if a vocal tic featured earlier in his history, which is why the criterion is written around the whole course of the illness and not the cross-section in front of you. It is also why a careful longitudinal history, not just the present examination, is diagnostic.

RULE **Tourette's disorder is the tic disorder with both motor and vocal tics; the two types need never have coincided in time.** Persistent tic disorder carries one type only. The examiner is testing whether you read Criterion A across the lifetime of the illness rather than across the day of assessment, so a history of an earlier vocal tic in a currently motor-only child still satisfies it.

30.2 Criteria B, C and D: the duration clock, the age ceiling and the exclusions

The remaining three criteria are about time and attribution, and each is a discrete examinable rule. Criterion B is the duration requirement: the tics must have persisted for **more than one year** since the first tic appeared. The subtlety that catches candidates is that this clock is measured from the onset of the first tic and is *not* reset by tic-free intervals; the tics may wax and wane, and may even disappear for weeks, yet the year is counted from that first onset. Criterion C sets the age ceiling: onset of tics must be **before age 18**. A genuinely new tic disorder beginning in adult life is exceedingly rare and should prompt a search for another movement disorder or a secondary cause rather than a first diagnosis of Tourette's. Criterion D is the exclusion: the disturbance must be not attributable to the physiological effects of a substance or another medical condition, the classic offenders being a stimulant such as cocaine, and medical conditions such as Huntington's disease or a post-viral encephalitis. Together the four criteria form a pattern-and-time definition in which no single symptom is pathognomonic.

CRITERION	REQUIREMENT
A	Multiple motor tics and one or more vocal tics, at some time in the illness, not necessarily together
B	Tics have persisted more than one year from first onset, though they may wax and wane
C	Onset before age 18
D	Not attributable to a substance or another medical condition

The four diagnostic criteria of Tourette's disorder; A is the discriminator from persistent tic disorder, and B, C and D are the time-and-attribution rules that surround it.

MNEMONIC

Two types, one year, under eighteen, nothing else

Two types of tic, motor and vocal, at some time in the illness (A); persisting more than **one year** from first onset (B); onset **under eighteen** (C); and **nothing else** - not a substance and not another medical condition - accounting for it (D). Four spare rules, no single dramatic symptom, is the whole definition.

Carry into the exam: both multiple motor and one or more vocal tics at some time in the illness, persisting more than one year from first onset, with onset before age 18, and not due to a substance or medical condition.

30.3 The phenomenology of tics: simple, complex, and the first tic

Before the criteria can be applied, the movements have to be correctly identified as tics, and that is a phenomenological skill. Tics are conventionally sorted along two axes: motor against vocal, and simple against complex. **Simple tics** are brief and meaningless - an eye blink, a facial grimace, a head jerk or a shoulder shrug on the motor side; a sniff, a throat-clear, a grunt or a cough on the vocal side. **Complex tics** are longer, appear more purposeful and can look almost voluntary: touching, tapping, jumping or a coordinated sequence of movements on the motor side, and syllables, words or phrases on the vocal side. The complex vocal tics include the phenomena the condition is unfairly famous for - the involuntary utterance of obscenities, and the repetition of others' or one's own words - and it is worth stating plainly that these dramatic symptoms are uncommon and are in no way required for the diagnosis. Most patients never show them. The typical developmental sequence is characteristic: simple motor tics are the most common initial presentation, and eye blinking is the most common initial tic, with the tics tending to begin in the head and face and to progress caudally down the body as the child grows, complexity generally increasing with age.

	MOTOR	VOCAL (PHONIC)
Simple	Eye blink, grimace, head jerk, shoulder shrug	Sniff, throat-clear, grunt, cough
Complex	Touching, tapping, jumping, co-ordinated sequences	Syllables, words, phrases; ob-scene utterances and word repe-tition (uncommon)

Tics classified on the two axes; note that the socially notorious complex vocal tics sit in one cell only, are uncommon and are never a requirement for diagnosis.

TRAP **The involuntary utterance of obscenities is neither necessary nor common in Tourette's disorder.** Candidates, primed by the public image, treat it as the defining or even a required feature. It is a complex vocal tic seen in only a minority; a patient can have unmistakable Tourette's without ever producing it, and its absence never excludes the diagnosis.

30.4 The premonitory urge and the semi-voluntary quality of tics

The feature that most reliably tells a tic apart from another movement disorder is the sensation that precedes it. Older children and adults commonly describe a **premonitory urge** - a localised uncomfortable sensation or a more generalised inner tension that builds before the tic and is momentarily relieved by performing it. A patient may report a burning feeling in the eye that eases only on blinking, or a tension in the neck that resolves on jerking the head. The tic is often repeated until the sensation resolves and it feels just right, an experience that overlaps phenomenologically with the "just right" perceptions of obsessive-compulsive disorder and helps explain why the two conditions travel together so often. This urge-relief cycle is what gives tics

their peculiar *semi-voluntary* quality: the movement is experienced as neither wholly involuntary, like a chorea, nor wholly deliberate, but as a response to an internal pressure the patient can feel and partly control. Because young children may not yet be able to articulate the urge, its absence in a small child does not count against the diagnosis, but its presence in an older patient is strong confirmatory evidence that the movements are tics.

PITFALL

The semi-voluntary quality is regularly misread. Because a child can suppress the tics briefly and describes an urge behind them, teachers, parents and even clinicians conclude the movements are "a habit", "for attention" or "put on", and the child is disciplined rather than assessed. The urge and the partial control are intrinsic features of a neurological disorder, not evidence of volition or manipulation; reframing them for the family is often the first therapeutic act.

30.5 Course over time: waxing, waning and suppressibility

Tics are fundamentally fluctuating, and their fluctuation is a diagnostic feature rather than noise. Over the natural history the tics **wax and wane** in frequency, anatomical location, number, complexity and severity, changing over intervals as short as minutes and as long as years, and some individuals experience tic-free intervals of weeks to months. This waxing and waning is so characteristic that a movement disorder with a rigidly fixed, unvarying pattern should raise doubt about the diagnosis. Superimposed on this fluctuation is **suppressibility**: tics can be voluntarily held back for varying periods, usually at the cost of mounting inner tension that is then discharged in a flurry once the effort relaxes, which is why a child may appear tic-free in the clinic and then tic heavily on reaching the car. Tics are also state-dependent, increased by anxiety, excitement, fatigue and stress and reduced during calm, absorbing, focused activity. The examinable consequence bears repeating from the duration criterion: because the year is counted from the first tic, these tic-free stretches do *not* reset the one-year clock, and a child with three months of tics, two tic-free months and then further tics can still meet Criterion B.

- Fluctuation over minutes to years, with possible remissions of weeks to months, is expected and supports the diagnosis;
- brief voluntary suppression at the price of rising tension distinguishes tics from chorea and myoclonus, which cannot be held back;
- anxiety, excitement, fatigue and stress worsen tics, while calm focused engagement lessens them.

30.6 Epidemiology: how common, in whom, and when it starts

The epidemiology answers the two questions the exam asks around every childhood disorder - how rare is it really, and who gets it. Isolated tics are extremely common in childhood while Tourette's itself is uncommon, and holding those two facts together is the point. Transient tics affect a large minority of children - of the order of 5 to 20 per cent - and are usually self-limiting, whereas Tourette syndrome is far rarer, at around 0.7 per cent of children and adolescents. A meta-analysis of studies of children put the pooled prevalence at about 0.77 per cent, and a national survey estimate gave 3 per 1,000 clinically identified cases, with a school-age estimate of

3 to 9 per 1,000 children. The concordance of these independent figures around roughly three to eight per thousand is reassuring, and the takeaway is a rare disorder sitting under a very common symptom. The condition shows a consistent male predominance, with a male-to-female ratio of **2:1 to 4:1**. As for timing, the average age at onset is **4 to 6 years**, with tic severity typically greatest at 10 to 12 years before the usual adolescent decline, so a child most often presents in early school years and is at his worst around the transition to secondary school.

IN INDIA

Indian services classify under the ICD rather than the DSM, so the operative label in case files and referrals is the ICD tic-disorder entity, though the diagnostic logic - both tic types, more than a year, onset in childhood - is the same. In practice Tourette's is under-recognised here: mild tics are frequently dismissed as a "nervous habit" or as naughtiness by families and schools, the flamboyant complex vocal tics that would trigger referral are the exception rather than the rule, and children commonly reach a psychiatrist late, often only when a comorbid attention or obsessive-compulsive problem forces the issue. A low index of suspicion for the quiet, motor-tic-predominant child is the main corrective.

30.7 Prognosis into adulthood and the suicide risk

The natural history is, for most, genuinely encouraging, and that reassurance is part of good management. Tic severity most often declines through adolescence, and by early adulthood the majority have shed the bulk of their tics: 20 per cent or fewer continue to have significant functional impairment by the age of twenty, and about 65 per cent have no or only very mild tics by adult life. This favourable trajectory justifies a conservative, watchful stance in many children and a frank, hopeful conversation with families who arrive fearing a lifelong sentence. That optimism, however, must be balanced against a real and under-appreciated risk at the severe end of the spectrum. Among young people with a persistent tic disorder, about one in ten has suicidal thoughts or behaviours, and the disorder carries an increased risk of both suicide attempt (odds ratio 3.86) and death by suicide (odds ratio 4.39). This elevation persists after adjusting for psychiatric comorbidity, and the strongest predictors are persistence of tics beyond young adulthood and a previous attempt, so the very patients whose tics do not remit are the ones who most need active screening for suicidality rather than reassurance alone.

GOOD TO REMEMBER

Hold both messages at once. The default forecast you give a family of a newly diagnosed child is optimistic, because most tics fade by adult life. But the minority whose tics persist into and beyond adolescence, and anyone with a prior attempt, sit in a higher-risk group for suicide and deserve explicit, repeated safety screening. Do not let the good general prognosis lull you into skipping the risk assessment in the severe, persistent case.

30.8 Measuring severity: the standardised rating of tics

Diagnosis is clinical, but quantifying severity and tracking change calls for a structured instrument. The best-established is the **Yale Global Tic Severity Scale**, a clinician-rated instrument that scores motor and vocal tics separately across five dimensions - number,

frequency, intensity, complexity and interference - and adds a global impairment rating. Rating the two tic types on the same five axes captures the fact that a child may have many mild motor tics but a single highly disabling vocal one, or the reverse, and the separate impairment score records how much the tics actually cost the child in daily life, which need not track their raw frequency. Such standardised scales improve the reliability of diagnosis and give quantifiable baseline data - frequency and intensity - against which the response to any later intervention can be judged. They are tools for measurement and monitoring, not substitutes for the criteria: a high severity score confirms how bad the tics are, but the diagnosis itself still rests on the four-criterion pattern applied to the history. Comorbidity, chiefly with attention-deficit/hyperactivity disorder and obsessive-compulsive disorder, is usually what drives impairment beyond the tics themselves and is developed as a pattern in its own right elsewhere in this chapter; a severity rating is therefore read alongside, not instead of, an assessment of the comorbid load.

>1 year	2:1 to 4:1	4 to 6 yr	~65%
TIC DURATION FROM FIRST ONSET (CRITERION B)	MALE-TO-FEMALE RATIO	AVERAGE AGE AT ONSET	NO OR MINIMAL TICS BY ADULT LIFE

31 · Etiology and neurobiology of Tourette syndrome

Tourette syndrome is one of the few child psychiatric disorders whose story can be told at the level of a circuit, a neurotransmitter and a gene, and that is exactly why it is examined. A tic is a sudden, recurrent, non-rhythmic movement or vocalisation, and the question this topic answers is where in the nervous system such a fragment of movement is generated, why it is felt as almost-but-not-quite voluntary, and why it clusters so tightly in families. The honest summary a candidate must be able to give is that the disorder is understood as a developmental disturbance of the motor control loops that link the cortex to the deep grey matter, running on an abnormal dopaminergic tone, on a genetic substrate that is among the strongest in child psychiatry.

What makes the neurobiology worth teaching, rather than reciting, is that each strand of evidence carries a clinical corollary. The circuit model explains why tics are patterned and suppressible; the dopamine model explains why one class of drug calms tics and another provokes them; the genetic model explains why tics, obsessive-compulsive symptoms and inattention travel together in the same families. This account owns the etiology and neurobiology. The classification of the tic disorders, the diagnostic criteria and phenomenology, the comorbidity pattern, and the management of tics are each taught in their own accounts within this chapter and are named here only where the mechanism points to them.

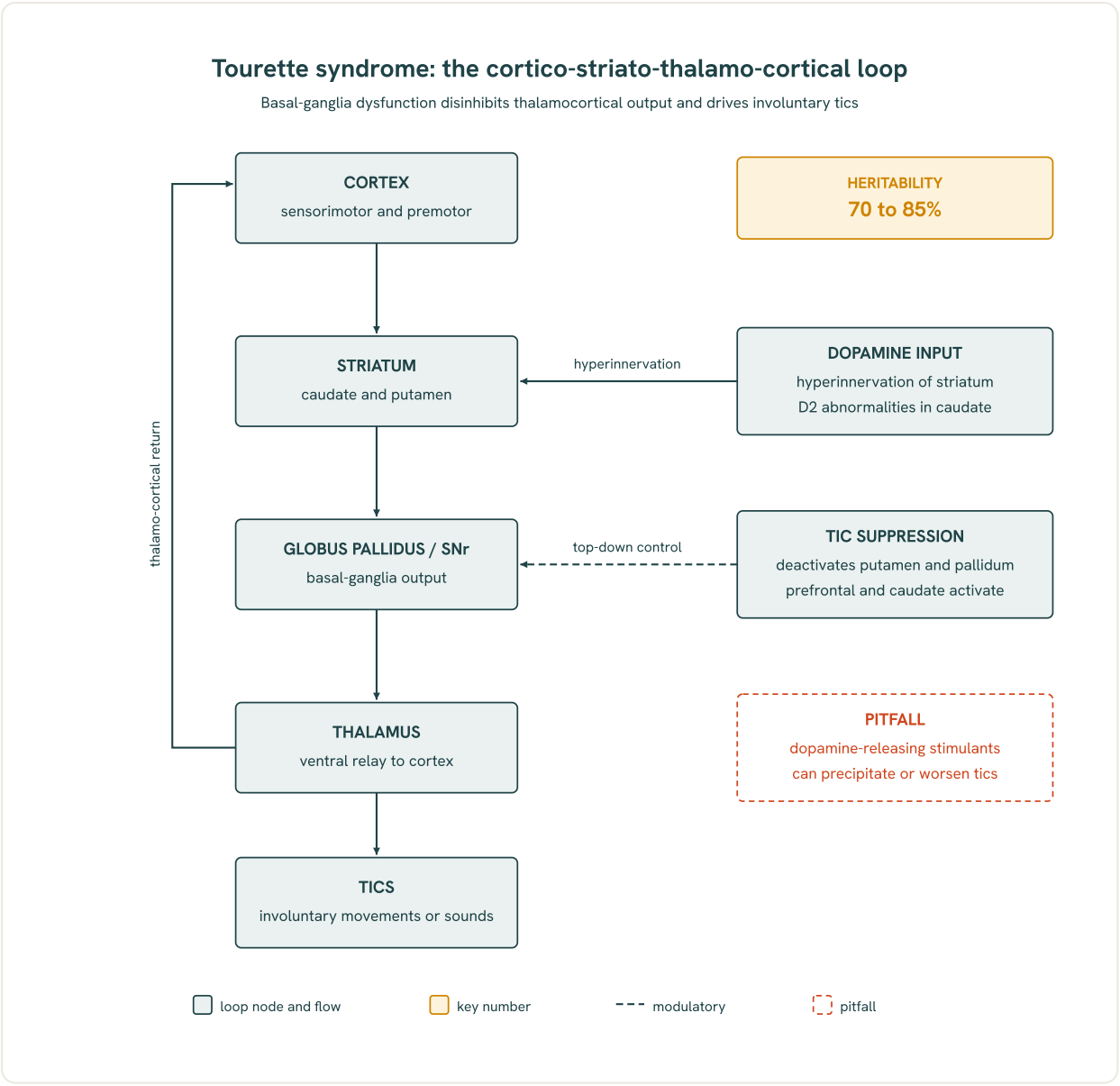

Fig 23.21. The cortico-striato-thalamo-cortical circuit in Tourette syndrome: dopaminergic hyperinnervation of the striatum dysregulates the basal-ganglia loop, disinhibiting thalamocortical output to produce tics.

31.1 A disorder of a motor circuit, not of a single brain region

The organising idea is that tics arise from dysfunction of the loops connecting cortex and basal ganglia, not from damage to one isolated structure. The prevailing model locates the disorder in the **cortico-striato-thalamo-cortical (CSTC) circuit**, the set of parallel loops through which the cortex projects to the striatum, the striatum to the pallidum and thalamus, and the thalamus back to the cortex, gating which motor programmes are released and which are held back. In Tourette syndrome this gating is faulty: fragments of movement that should be suppressed escape into action as tics. Volumetric and functional imaging support the model from several directions at once, implicating cortical thinning in the sensorimotor cortex, striatal structural anomalies and disruption of temporolimbic pathways. The last of these matters because it links the pure motor phenomenon to the affective and urge-laden quality that patients describe, and it is why the disorder sits with the neurodevelopmental conditions rather than with the movement disorders of later life.

MNEMONIC

Cortex - Striatum - Thalamus - Cortex

The name of the circuit is its own map, read in the direction the signal travels: the **C**ortex drives the **S**triatum, the striatum gates the pallidum and **T**halamus, and the thalamus closes the loop back to the **C**ortex. A tic is what happens when this loop fails to hold an unwanted motor programme in check, so every anatomical finding in this topic can be pinned to one station on the ring.

31.2 The basal ganglia as the hub of tic generation

Within that circuit, the weight of evidence falls on the basal ganglia. The **basal ganglia** - the striatum, comprising the caudate and putamen, together with the globus pallidus - are held to be central both to the generation of tics and to their suppression. Two findings anchor this. Structurally, a smaller **caudate** volume in childhood has been linked to worse tic severity in later life, which makes the caudate not merely a site of the lesion but a prognostic marker, one of the few in the disorder. Functionally, a greater capacity of the basal ganglia to suppress cortical activity is associated with lower tic severity, so the same structure that generates the intrusive movement also carries the machinery that damps it down. This is the anatomical reason the disorder is not static: the balance between generation and suppression can shift with maturation, which is part of why tics so often wax through childhood and settle in adolescence.

RULE **Tourette syndrome is a disorder of the cortico-striato-thalamo-cortical circuit with the basal ganglia at its hub, not a disorder of a damaged focal lesion.** The tic is a failure of the normal gating that decides which motor programmes reach the muscles. Every neurobiological finding worth memorising - the striatal anomalies, the caudate-severity link, the dopaminergic abnormality, even the response to medication - is a statement about this loop and its neurochemical tone, which is why the model unifies the imaging, the pharmacology and the natural history into one picture.

31.3 The neurology of voluntary tic suppression

The one feature that separates a tic from a chorea or a myoclonus is that a patient can hold it back, and the imaging of that effort is examinable. Tics are only partially involuntary: most patients can suppress them briefly, at the cost of a mounting inner tension that is relieved when the tic is finally released. When patients suppress tics voluntarily, imaging shows deactivation of the putamen and globus pallidus alongside activation of the prefrontal cortex and caudate. The reading is intuitive once the circuit is in mind: the prefrontal cortex and caudate exert top-down control that quietens the pallidal output driving the tic. This is not an academic detail. It is the neurobiological basis for the behavioural treatment of tics, in which patients are trained to deploy exactly this top-down control against the premonitory urge; that treatment is taught in the account of tic management in this chapter, and the mechanism here is what makes it rational rather than merely empirical.

31.4 The dopaminergic hypothesis

The best-supported neurochemical account is of an abnormal dopaminergic tone within these same striatal circuits. The **dopaminergic hypothesis** holds that tics arise from abnormal

dopamine neurotransmission, specifically an increased dopaminergic innervation, or hyperinnervation, of the striatum together with D2-receptor abnormalities in the caudate. Two independent lines of evidence converge on it:

- functional imaging with **SPECT** shows increased dopaminergic innervation of the striatum in affected individuals;
- studies of **monozygotic twins** discordant for severity show the D2-receptor abnormality to be greater in the more severely affected twin, tying the receptor finding directly to how ill the child is.

The twin observation is the more powerful of the two, because monozygotic co-twins share their entire genome: a difference between them in receptor binding that tracks their difference in severity points to something acting on the dopaminergic system beyond the inherited sequence, and it locates the abnormality in the caudate, the very structure the circuit model already implicated. Dopamine is the best-supported system but not the only one proposed; serotonergic, noradrenergic, glutamatergic and GABAergic contributions have all been suggested, though the evidence for them is weaker and none can be asserted here with the same confidence.

31.5 Pharmacological convergence: the drugs that prove the point

The strongest clinical argument for the dopamine model does not come from a scanner at all - it comes from watching what medicines do. The pharmacology converges on the hypothesis from both ends. **Dopamine-releasing drugs such as amphetamine precipitate or exacerbate tics, whereas dopamine D2-receptor-blocking neuroleptics suppress them**, and patients with the disorder release more dopamine in response to amphetamine than controls do. That double dissociation - worsen with a releaser, improve with a blocker - is difficult to explain on any model other than an overactive or oversensitive striatal dopamine system, and it is the reason the D2-blocking antipsychotics became the mainstay of pharmacological tic control, an application developed in the tic-management account of this chapter. The mechanism is owned here; the drugs, doses and titration belong there.

> **PITFALL**
>
> When a child with attention problems is started on a stimulant and tics appear or worsen, the reflex is to conclude the drug caused a new disorder. The mechanism argues otherwise: a dopamine-releasing agent unmasks or amplifies a tic diathesis that the CSTC and dopaminergic abnormality had already set up, rather than manufacturing Tourette syndrome from nothing. Reading the exacerbation as pharmacological proof of an underlying dopaminergic sensitivity, not as iatrogenic causation, keeps the clinician from both over-alarming the family and missing the co-occurring tic disorder; what to do about the stimulant itself is a decision for the comorbidity and management accounts.

31.6 A strongly heritable, polygenic disorder

Tourette syndrome and the tic disorders are among the most heritable conditions in child psychiatry, and the shape of that heritability is as examinable as its size. The **heritability** of tic disorders is estimated at roughly **70 to 85 per cent**, meaning that most of the variation in

whether and how severely tics appear is attributable to genetic factors. Two qualifications matter. First, there is no sex difference in the familial or heritable risk: although the disorder is expressed more often in boys, the underlying genetic liability is carried equally by both sexes, so a family history counts the same whether the affected relative is male or female. Second, the inheritance is **polygenic**: the liability is built from many genes of small effect rather than a single dominant locus, and the tic disorders most likely lie along a continuous developmental spectrum rather than forming a discrete category cut cleanly from the unaffected. This is why the search for a single Tourette gene failed and why the modern account is of accumulated common variation crossing a threshold.

31.7 A genetic architecture shared with OCD, ADHD and other neurodevelopmental disorders

The genetics do not stop at the tic - they reach across into the disorders that so reliably accompany it. Chronic tic disorders **share genetic variance** with obsessive-compulsive disorder, attention-deficit/hyperactivity disorder and other neurodevelopmental disorders including autism spectrum disorder. The common variants are shared in a graded fashion that correlates with severity, so the more heavily loaded a child is genetically, the broader and heavier the phenotype tends to be. This is the deep reason that tics rarely arrive alone: the same inherited liability that raises the risk of tics raises, in overlapping measure, the risk of obsessions, compulsions and inattention, which is why the clinically dominant problem is so often a comorbidity rather than the tic itself. The comorbidity pattern and the impairment it drives are taught in the dedicated account of Tourette comorbidities in this chapter; obsessive-compulsive disorder and attention-deficit/hyperactivity disorder as disorders in their own right belong elsewhere, the latter to the ADHD and specific learning/communication disorders notes. What this topic owns is the mechanism beneath the association: shared, graded, polygenic liability, not coincidence.

STRAND OF EVIDENCE	CORE FINDING	NATURE OF THE EVIDENCE	WHAT IT EXPLAINS
The circuit	CSTC dysfunction with sensorimotor cortical thinning and temporolimbic disruption	Volumetric and functional imaging	Why tics are patterned, gated movements with an urge quality
The hub	Striatum and pallidum central to tic generation and suppression; smaller childhood caudate tracks worse later severity	Structural and longitudinal imaging	The anatomical seat and a prognostic marker
The chemistry	Striatal dopaminergic hyperinnervation and caudate D2 abnormality	SPECT and monozygotic-twin studies	The neurochemical lesion and the drug response
The pharmacology	Releasers worsen tics, D2-blockers suppress them	Clinical and pharmacological convergence	Why antipsychotics calm tics and stimulants can provoke them
The genes	Strongly heritable and polygenic, with variance shared with OCD, ADHD and other neurodevelopmental disorders	Twin, family and molecular genetics	Familial clustering and the comorbidity pattern

The five convergent strands of the neurobiology of Tourette syndrome; each carries a clinical corollary, which is what makes the mechanism examinable rather than ornamental.

GOOD TO REMEMBER

Two things the neurobiology should change on Monday. Take a genuine family history in every child with tics, because the liability is strongly heritable and carried equally by both sexes, so an affected mother counts as much as an affected father. And when you counsel a family, frame the accompanying obsessive-compulsive or attentional symptoms not as separate misfortunes but as expressions of the same shared, graded genetic liability, which both normalises the pattern and prepares them for the comorbidity being the part that most needs treating.

31.8 Contested and environmental contributions

Genes set the liability, but they do not act in a vacuum, and the environmental side of the story is where confident teaching gives way to careful hedging. A polygenic liability expressed along a developmental spectrum implies that non-genetic factors help decide who crosses the threshold and how severely, and perinatal and other early influences have long been proposed as such modifiers; none, however, can be stated here as an established quantitative risk. The most examined and most contested environmental hypothesis is the post-infectious one, in which group A streptococcal infection is proposed to trigger tics through an autoimmune mechanism, described under the label PANDAS. That proposal is taught, with its evidence and its controversy, in the account of paediatric obsessive-compulsive disorder and PANDAS in this

chapter. It belongs in an etiology topic only as what it is: a proposed and genuinely contested infection-associated trigger, on limited evidence, and not an established cause of Tourette syndrome.

TRAP **Presenting PANDAS as a settled cause of Tourette syndrome is the classic examination error.** Candidates who have read about the autoimmune hypothesis reach for it as though a preceding sore throat closed the case, but the causal claim is contested and the evidence limited, and no examiner rewards stating it as established fact. The safe answer names PANDAS as a proposed, disputed infection-associated trigger and returns the weight of the etiology to where the evidence actually is: the heritable, polygenic disturbance of the cortico-striato-thalamo-cortical circuit and its dopaminergic tone.

IN INDIA

The neurobiology described here is built from imaging and molecular studies that a routine Indian clinic will never run: SPECT and volumetric MRI are research tools, not part of the diagnostic work-up, and the diagnosis of a tic disorder remains wholly clinical under the ICD framework used in Indian services. What the mechanism does change in practice is counselling. Because the disorder is strongly heritable and polygenic, a careful family history is the single most useful piece of the etiological assessment available at the bedside, and families - who often arrive fearing they caused the tics through parenting - can be told accurately that the liability is largely constitutional and inherited, not a product of upbringing or of any single event.

Carry into the exam: Tourette syndrome is a **70 to 85 per cent** heritable, polygenic neurodevelopmental disorder of the cortico-striato-thalamo-cortical circuit, with the basal ganglia at its hub and abnormal striatal dopaminergic tone, sharing its genetic liability with OCD and ADHD; dopamine releasers worsen tics and D2-blockers suppress them, and PANDAS is a contested trigger, never an established cause.

70-85% HERITABILITY OF TIC DISORDERS	**CSTC** THE IMPLICATED CORTICO-STRIATO-THALAMO-CORTICAL CIRCUIT	**Dopamine** STRIATAL HYPERINNERVATION AND CAUDATE D2 ABNORMALITY	**OCD, ADHD** DISORDERS SHARING TIC GENETIC VARIANCE

32 · Comorbidities (ADHD, OCD) in Tourette syndrome

Tourette's is almost never a disorder of tics alone. The great majority of people who reach a clinic with Tourette's carry at least one other psychiatric condition, and for most of them it is that accompanying condition, and not the movements, that drives them to seek help and that shapes the school years, the working life and the family's distress. Of all the conditions that keep Tourette's company, two stand out so consistently that they are best thought of as part of the syndrome's clinical identity rather than as chance neighbours. Attention-deficit/hyperactivity disorder and obsessive-compulsive disorder are the two **defining comorbidities** of Tourette's disorder, and their prominence holds in both clinical and community samples, which tells you that this is not simply a referral artefact of the most complicated patients reaching specialist care.

A candidate who pictures Tourette's as an isolated movement problem has the wrong mental model, and the exam is built to expose exactly that misconception.

This topic owns the comorbidity pattern in Tourette's: which conditions cluster with it, why that clustering matters more than the tics for outcome, and how that fact reorders the clinical priorities. It does not own, and must not re-teach, the disorders themselves. The full criteria, subtyping and treatment of **attention-deficit/hyperactivity disorder**, including its stimulant, non-stimulant and alpha-adrenergic agonist options, belong to the ADHD and specific learning/communication disorders notes; the criteria and management of **obsessive-compulsive disorder** belong to the Anxiety, OCD and trauma-related disorders notes. What is taught here is the overlap and its consequences, which is where the marks in this area actually sit.

32.1 Comorbidity is the rule, not the exception

The isolated tic patient is the unusual one. The single most important reframing in this topic is that a person presenting with Tourette's and nothing else is the minority case. Co-occurring psychiatric disorder is so common that a clinician who fails to look for it will miss the larger part of the patient's difficulty. That the same two conditions, ADHD and OCD, dominate the comorbidity profile in community samples as well as in clinics matters for a precise reason: if the association held only in specialist series it could be dismissed as referral bias, the sickest and most tangled patients being the ones who reach tertiary care. Because it holds in the community too, the link is a property of the syndrome itself and not of who happens to be referred. This is why the modern account of Tourette's treats it as a neurodevelopmental condition whose psychiatric comorbidity is intrinsic, and why screening for that comorbidity is a mandatory part of every assessment rather than an optional extra.

32.2 ADHD: the earliest and often the most disabling companion

The inattention and impulsivity usually arrive before the tics and outlast the clinic's attention to them. Of the two defining comorbidities, ADHD is typically the one that declares itself first, its inattention, restlessness and impulsivity commonly evident in the years before the tics emerge and are recognised. For the child, this ordering has a cruel consequence: the academic and social damage of untreated attention and impulse difficulties is often well established by the time anyone notices the tics and attaches the Tourette label. The impairment of comorbid ADHD in this population lands where impairment always does with ADHD - on learning, on the ability to hold a place in the classroom, on peer relationships and on family life - and it frequently exceeds anything the tics themselves are doing. The natural clinical error is to let the visible, dramatic movements monopolise the consultation while the quieter but more corrosive attentional problem goes unaddressed. The specific pharmacology of ADHD, and the long-standing question of using stimulants in a child who also tics, are matters for the ADHD and specific learning/communication disorders notes; the point that belongs here is simply that ADHD is usually present, usually early, and usually the heavier burden.

32.3 OCD: the obsessive-compulsive phenomena that shade into the tics

The obsessive-compulsive side of Tourette's has a texture of its own. Obsessive-compulsive disorder is the second defining comorbidity, and in the Tourette context it tends to wear a characteristic face. What many patients describe is a **tic-related OCD**: compulsions driven less by a fear of catastrophe and more by an intolerable sense that something is not yet complete or symmetrical, an urge to touch, tap, order, count or repeat until an internal feeling of "just right" is reached. This blurs, at its edges, into the tics themselves, because the more complex tics and the simpler compulsions can be hard to tell apart, both being preceded by an uncomfortable inner urge that the act discharges. That overlap is diagnostically important and is developed where the phenomenology of tics is taught in this chapter; here the essential message is that OCD is a defining companion of Tourette's and that its presentation in this group leans towards the symmetry-and-completion phenotype rather than the contamination-and-checking picture. The criteria, the full symptom dimensions and the treatment of OCD are the province of the Anxiety, OCD and trauma-related disorders notes, and nothing about them is re-taught here.

32.4 The overlap seen from the OCD side: tics within OCD and the triad

Look at the same relationship from the obsessive-compulsive end and it points straight back to tics. The comorbidity is genuinely bidirectional, and the exam likes to test it from the OCD direction because that is where a clean, quotable figure exists. Among people whose primary diagnosis is OCD, **up to 30%** carry a lifetime tic disorder, and this comorbid tic disorder is most common in men whose OCD began in **childhood-onset OCD**. In children in particular the three conditions cluster together often enough to be named as a unit: a **triad of OCD, tic disorder and ADHD** that can be seen presenting together. Recognising the triad matters clinically because encountering any one of its members should prompt a deliberate search for the other two; the child referred for compulsions may have unrecognised tics and untreated inattention sitting behind them, and the whole picture, not the presenting fragment, is what has to be managed. This is the overlap that the older literature gathered under the loose heading of a Tourette-associated or "Tourette-plus" pattern, and it is the reason the tic disorders and the obsessive-compulsive disorders are read together rather than in separate silos.

32.5 The Tourette-plus phenotype and where the impairment actually sits

Add the comorbidities to the tics and you have described most real patients. Pure Tourette's, tics and nothing more, is the textbook simplification; the common clinical reality is the **Tourette-plus** picture, in which the tics are accompanied by ADHD, or by OCD, or by both, frequently alongside further difficulties such as co-occurring mood and anxiety disturbance. The reason this composite label earns its keep is not tidiness but prognosis. In these patients the condition that most damages daily functioning is usually not the tics but the accompanying disorder: the inattention that sinks the school report, the compulsions that consume hours of the day, the anxiety or low mood that closes the world down. The tics may be what an observer notices first and what the family finds most socially exposing, yet when you total up the lost function, the comorbidity is generally the larger contributor. Holding that ranking in mind is

what separates a clinician who treats the noisiest symptom from one who treats the most disabling one.

RULE **In Tourette's, the comorbidity usually decides how disabled the patient is, not the tics.** Tic frequency and tic severity are a poor guide to overall functioning, because the co-occurring ADHD, OCD, anxiety or mood disorder typically carries more of the impairment. Assess the whole comorbidity load before you judge how ill the patient is, and never let the visibility of the movements stand in for a measure of the disability.

32.6 The clinical logic: treat the comorbidity, often before the tics

The order of treatment follows the order of impairment. If the comorbid condition is usually the greater source of disability, then the sequencing of management follows almost automatically: in most Tourette-plus patients the comorbidity is identified and treated first, and in a good number the tics need no direct drug treatment at all once the ADHD or the OCD is under control and the associated distress has eased. This is not a rule that the tics are ignored, but a rule of priorities - you address what is doing the most harm before you turn to what is most conspicuous. It also reframes the whole assessment: a proper Tourette's evaluation is as much an inventory of the accompanying conditions and their relative impact as it is a characterisation of the tics. The behavioural and pharmacological treatment of the tics themselves is developed elsewhere in this chapter; what this topic establishes is the reasoning that so often places that tic treatment second in the queue.

DEFINING COMORBIDITY	HOW IT RELATES TO THE TOURETTE PICTURE	WHERE ITS IMPAIRMENT PRINCIPALLY LANDS
ADHD	Usually the earliest problem, often evident before the tics are recognised; the commonest companion of Tourette's	Learning and academic attainment, classroom conduct, peer relationships and family strain
OCD	Frequently a symmetry-and-completion, tic-related pattern that blurs at its edge into the more complex tics	Time lost to compulsions, internal distress from the "not just right" urge, and rigidity of routine
Mood and anxiety disorders	Common further companions in the Tourette-plus picture rather than defining ones; named, not the focus here	Withdrawal, avoidance and lowered mood that constrict daily life beyond what the tics do

The comorbidity pattern of Tourette's read as a whole: the two defining companions and the further conditions that complete the Tourette-plus picture, set against the domain each one chiefly disables. The tics are frequently the least of it.

- ADHD and OCD are the two defining comorbidities, present in clinical and community samples alike;
- the overlap is bidirectional - a substantial share of OCD patients carry a lifetime tic disorder;
- in children the three may cluster as an OCD, tic disorder and ADHD triad;
- the comorbidity usually causes more functional impairment than the tics;

- so assessment inventories the comorbid load, and treatment commonly addresses it first.

32.7 The wider company Tourette's keeps

Beyond the defining pair, a broader psychiatric load is the norm. Naming ADHD and OCD as the defining comorbidities does not mean they are the only ones. Mood and anxiety disorders co-occur with Tourette's frequently enough to belong in every assessment, the anxiety spectrum running from separation anxiety in the younger child to generalised anxiety, both of which are taught in their own right elsewhere in this chapter and are only named here as members of the comorbid picture. The practical implication is cumulative: because several conditions may stack on top of the tics, the disability a Tourette's patient carries is often the sum of a number of partly independent problems, each needing its own recognition. A clinician who screens only for the two headline comorbidities and stops there will still under-count the burden. The discipline the topic asks for is a systematic sweep across attention, obsessive-compulsive phenomena, mood and anxiety in every patient who presents with tics, rather than a search confined to whichever symptom happens to be loudest on the day.

In Tourette's, screen every patient for ADHD and OCD, and remember the comorbid condition usually disables more than the tics do.

TRAP **Assuming that tic severity tracks overall impairment.** The classic examination error is to grade a Tourette's patient's illness by how frequent or forceful the tics are. Candidates make it because the tics are the visible, nameable feature and feel like the measure of the disorder. In fact the co-occurring ADHD, OCD, anxiety or mood disorder is usually the heavier burden, so a patient with modest tics and severe untreated ADHD may be far more disabled than one with florid tics and nothing else.

PITFALL

The commonest management failure is to pour the consultation into suppressing the tics while the comorbid ADHD or OCD goes untreated. The movements are what the family points to and what draws the eye, so effort and medication get spent on them, and meanwhile the child's schooling collapses under unmanaged inattention or their waking hours are eaten by compulsions. The corrective is to rank the sources of impairment before reaching for a tic drug, and to treat the condition that is doing the most harm first.

GOOD TO REMEMBER

What changes on Monday is the shape of the assessment. Every patient who presents with tics is screened deliberately for attention and impulse problems and for obsessive-compulsive phenomena, and the relative impairment of each is weighed before any treatment plan is drawn. If the comorbidity is the greater burden, it is named as the priority in the plan and addressed first; the tics can very often wait, and sometimes need no direct drug treatment once the comorbidity is controlled.

IN INDIA

Two realities of Indian practice sharpen this topic. First, tics are frequently dismissed by families and even by non-specialists as mere habits or naughtiness, so the child often reaches services not for the movements but for the school failure of comorbid ADHD or the puzzling rituals of comorbid OCD; the comorbidity is the presenting complaint far more often than the tics are. Second, specialist tic services are scarce and unevenly distributed, which makes the impairment-first logic doubly useful: a general psychiatrist or paediatrician who cannot offer sophisticated tic-directed therapy can still transform a child's life by recognising and treating the accompanying ADHD or OCD, which is usually where the real disability sits anyway.

up to 30%	ADHD + OCD	Tourette-plus
OF OCD PATIENTS HAVE A LIFETIME TIC DISORDER	THE TWO DEFINING COMORBIDITIES OF TOURETTE'S	COMORBIDITY USUALLY OUTWEIGHS THE TICS

33 · Management of tics (habit reversal, CBIT, pharmacotherapy)

Most people with tics never need a drug, and knowing when not to prescribe is the larger part of the skill. Tics wax and wane, peak in the early school years and often settle through adolescence, so the management question is rarely "which medication" and much more often "does this child need any active treatment at all, and if so, what is the least intervention that restores function". The marks go to the candidate who holds that restraint and then doses each agent to its tic indication rather than to a number learned for a different one.

This section owns the treatment of tics in full: the decision to treat, the behavioural first line of habit reversal and its parent programme, the stepped sequence of drugs, and every tic-indication dose with its monitoring. The phenomenology of tics, the diagnostic criteria of Tourette syndrome and the comorbidity pattern of attention-deficit/hyperactivity disorder and obsessive-compulsive disorder are developed elsewhere in this chapter and named here only where a treatment decision turns on them.

Fig 23.22. Tic management ladder: habit reversal training within CBIT first for mild to moderate tics, then an alpha-2 agonist (clonidine or guanfacine), then an antipsychotic (aripiprazole or risperidone). Aripiprazole is the tic-licensed choice at the antipsychotic step, and its dose is given by weight band rather than as a single range because the upper end of that range is licensed only for patients of 50 kg or more. Clonidine, guanfacine and risperidone are used off-label and have no licensed tic dose, so no dose is printed for them: what is shown instead is the labelled absence itself, and for risperidone a named trial's mean, labelled as a trial mean and not a regimen. Haloperidol and pimozide are also tic-licensed but are held in reserve on tolerability and cardiac grounds.

33.1 Deciding to treat, and the order of treatment

Treatment is aimed at impairment, not at the tic count. A child whose tics are noticed but not distressing, not painful and not disrupting learning or relationships usually needs psychoeducation, reassurance and school liaison, and nothing more. Active treatment is reserved for tics that hurt, that draw bullying, that interrupt schooling or that the young person themselves wants reduced. When treatment is warranted it follows a deliberate sequence rather

than a menu: educational and behavioural treatment first, then an **alpha-2 agonist** if a drug is needed, then an antipsychotic, and only then the older or experimental agents. An alpha-2 agonist is generally tried before an antipsychotic because the adverse-effect cost of an antipsychotic can outweigh its benefit in a fluctuating, often self-limiting condition.

Comorbidity reorders the whole plan. Where attention-deficit/hyperactivity disorder or obsessive-compulsive disorder coexists, the comorbid disorder is frequently the greater source of impairment and is usually treated first, because controlling it can make the tics feel manageable without a tic-specific drug. This is also why the alpha-2 agonists are attractive: they address tics and the comorbid hyperactivity together.

Stimulants are not contraindicated in tic disorders, and believing that they are withholds the treatment most likely to help. Current labelling for methylphenidate and for amphetamine places tics and Tourette's under warnings and precautions only, never under contraindications, which list nothing but hypersensitivity and recent or concurrent monoamine oxidase inhibitor use; what the label actually requires is that the family history be taken and the patient clinically evaluated for tics before starting, that treatment be monitored regularly for the emergence or worsening of tics, and that it be discontinued if that becomes clinically appropriate. The group-level evidence is flat: a meta-analysis of 22 randomised placebo-controlled trials in 2,385 children found new-onset or worsening tics in 5.7% on psychostimulant against 6.5% on placebo, a risk ratio of 0.99 (95% CI 0.78 to 1.27), with no moderating effect of stimulant type, dose, duration of treatment, recorder or age. Because tics wax and wane on their own, a worsening that follows a stimulant start is much more likely coincidence than cause, and rechallenge may be considered. Where a child has both tics and ADHD, methylphenidate, clonidine, clonidine with methylphenidate, and guanfacine are each probably more likely than placebo to reduce tic severity as well as ADHD symptoms, and the clinician should ensure that functionally impairing ADHD is treated.

RULE **Behavioural treatment leads, an alpha-2 agonist comes before an antipsychotic, and most tics need no drug at all.** Escalation is stepwise and impairment-driven, and comorbidity is usually treated first. Opening with an antipsychotic skips the two steps before it and inverts the safety logic of the ladder.

Carry this into the exam: treat the impairment not the tic, lead with education and habit reversal, use an alpha-2 agonist before an antipsychotic, and treat the comorbidity first, stimulant and all.

33.2 Habit reversal training and CBIT: the behavioural first line

The first-line active treatment is behavioural, and it is more than distraction. Habit reversal training is the core of Comprehensive Behavioural Intervention for Tics, the first-line treatment when a child with mild to moderate tics needs active intervention. Its power rests on a physiological fact: most tics are preceded by a **premonitory urge**, an uncomfortable building

sensation that the tic momentarily discharges. Because the urge precedes the movement, the movement can be intercepted. The programme layers components onto that:

- awareness training, teaching the young person to detect the urge and the earliest signs of the tic;
- **competing response** training, in which they perform a voluntary movement incompatible with the tic and hold it until the urge subsides;
- relaxation training, contingency management and social support, with parents coached to prompt and reward home practice;
- relapse prevention, so gains survive the next natural worsening of the tics.

The mechanism is a controlled extinction of the urge-to-tic loop: repeatedly meeting the premonitory urge with a competing response rather than the tic weakens the urge over time, so the behaviour is not merely suppressed by willpower but progressively unlearned. This is a tic-specific technique and should not be confused with the exposure and response prevention used for paediatric obsessive-compulsive disorder; the competing response is a motor substitution against an urge, not a graded exposure to a feared thought. Behavioural treatment is well supported, with effect sizes broadly comparable to antipsychotic medication for tic reduction, which is precisely why it, and not a drug, sits at the head of the active-treatment ladder. Its practical limits are access and effort: it needs a trained therapist, a child old enough to report the urge and monitor themselves, and sustained home practice.

MNEMONIC

Aware, Compete, Relax, Reward, Sustain

The working parts in order: **aware**ness of the premonitory urge, a **compet**ing response against it, **relax**ation to lower baseline arousal, **reward** for practice through contingency management and social support, and relapse prevention to **sustain** the gains.

33.3 The alpha-2 agonists: clonidine and guanfacine

When a drug is needed, the alpha-2 agonists are the first rung and the safest place to start. The thing most sources leave out: in none of the labelling this chapter has read, that of the United Kingdom, the European Union, Australia and the United States, is either agent licensed for tics, so neither has a licensed tic dose. **Clonidine** is used off-label; every clonidine product this chapter has checked is licensed for something else: hypertension, ADHD in its extended-release form, or, in United Kingdom labelling, migraine prophylaxis and menopausal flushing. Current labelling supplies no starting dose, no frequency, no titration interval and no maximum for the tic indication, precisely because no clonidine product carries a tic indication, and no ceiling licensed for another indication may be imported as the tic ceiling. That absence is the teaching, not a gap in it: the weight-based figure circulating in the textbooks is an order-of-magnitude target, not a regimen, so it cannot be prescribed from and has no business on a dosing page. The American Academy of Neurology's 2019 practice guideline recommends clonidine for tics where the benefits outweigh the risks, and directs counselling about sedation, monitoring of heart rate

and blood pressure, and gradual tapering, but it publishes no dose at all. The United Kingdom position is blunter still: both UK oral clonidine labels state that there is insufficient evidence for its use in children and adolescents under 18 and that clonidine is therefore not recommended in them, and both record that its efficacy in Tourette syndrome has not been demonstrated. Its adverse effects are the reason to go slowly: sedation, postural hypotension and low mood. On the evidence, people with tics given clonidine are probably more likely than placebo to have reduced tic severity, and the effect is larger where ADHD is comorbid.

Guanfacine is the more selective alpha-2A agonist and tends to sedate and lower blood pressure less. It too is used off-label for tics and has no licensed tic dose; its licensed indications are hypertension and ADHD, and people with tics given guanfacine are only possibly more likely than placebo to have reduced tic severity, a weaker grade of evidence than clonidine's, with the effect again larger where ADHD is comorbid. The milligram figures most trainees recite for guanfacine are the ADHD figures, so any milligram number offered for guanfacine in tics must be traceable to a named tic trial and labelled as that trial's dose, never lifted from a monograph range. There is a tic-specific efficacy anchor: in an 8-week double-blind randomised trial of children who had both a tic disorder and ADHD, guanfacine reduced tic severity by **31%** against no change on placebo.

The monitoring for this class is mandatory, and it is not only about sedation. For either alpha-2 agonist in tics, the physician must counsel the patient about common adverse effects including sedation, must monitor heart rate and blood pressure, and must taper the drug gradually on discontinuation to avoid rebound hypertension; the class should be prescribed when the benefits of treatment outweigh the risks. Those are level A requirements, not counsels of perfection. Guanfacine also carries a cardiac requirement: a physician prescribing extended-release guanfacine must monitor the QTc interval in a patient with a history of cardiac conditions, in a patient taking other QT-prolonging agents, and in a patient with a family history of long QT syndrome, also a level A requirement. The cardiac warning does not begin at the classical agents; it begins on the second rung.

The alpha-2 agonists are also used in ADHD, where they do have a licence and a licensed dose, and for that alpha-2 dose the reader should cross-refer the ADHD and specific learning/communication disorders notes. Those numbers belong to that indication, and there is no tic-indication counterpart to which they could be transferred.

TRAP **Reciting the ADHD-indication alpha-2 dose as if it were the tic dose.** The same two drugs, clonidine and guanfacine, are prescribed for ADHD and for tics, and a candidate who has memorised one milligram figure will offer it for the other indication. This trap is sprung more often than any other on this topic, and printed sources spring it too: the guanfacine range in general circulation fuses the hypertension dose of the immediate-release form with the ADHD dose of the extended-release form, and neither has anything to do with tics. Neither agent has a tic-indication dose at all, which is the answer the examiner is listening for. Naming the drug is not enough; the examiner wants the dose scoped to the indication in front of you, and wants you to say so when no such dose exists.

PITFALL

Stopping clonidine suddenly. Because it is sedating and sometimes only modestly effective, a frustrated family may simply discontinue it, and an abrupt stop of a central alpha-2 agonist risks rebound hypertension. Whenever clonidine or guanfacine is prescribed for tics, the taper and the instruction never to stop it suddenly are part of the prescription, not an afterthought.

33.4 The antipsychotics for tics: aripiprazole and risperidone

When tics are severe or the alpha-2 step has failed, a dopamine blocker is the most reliable suppressor, at a price. Tics respond to dopamine D2 blockade, which is why antipsychotics work and why their metabolic and motor costs are the reason they are not first. **Aripiprazole** is the usual modern choice and the only one of the two antipsychotics taught at this step that carries a tic licence: it is FDA-approved for Tourette's disorder in children and adolescents aged 6 to 18, with a recommended range of **5 to 20 mg/day**. That range is a whole-population envelope, not a prescribable instruction, and it must never be quoted or printed without the weight bands beside it, because its upper end is licensed only for the heavier band. Three agents here hold a United States tic licence: aripiprazole, haloperidol and pimozide. A licence is not a place in the sequence: both classical agents are licensed and sit low anyway, on tolerability and cardiac grounds, and pimozide's licence is itself written only for patients who have failed to respond satisfactorily to standard treatment. What separates aripiprazole is not freedom from cost, but that its cost is not the sedation, extrapyramidal burden and QTc surveillance the classical agents bring.

The bands are these, of a tablet taken once a day: the labelled formulation here is the oral tablet, administered once daily without regard to meals. A patient weighing less than 50 kg is started at 2 mg/day, with a target of 5 mg/day after 2 days, which may be increased to 10 mg/day if optimal tic control is not achieved; 10 mg/day is that patient's maximum. A patient weighing 50 kg or more is started at 2 mg/day for 2 days, then 5 mg/day for 5 days, with a target of 10 mg/day on the eighth day; only where optimal tic control is not achieved may the dose rise in increments of 5 mg/day to a maximum of 20 mg/day. In either band, dosage adjustments are made gradually, at intervals of no less than one week. The one-week rule and the two-day step are not in conflict: the staged schedule above is the label's own initial titration, and the one-week interval governs the discretionary adjustments made after it. The staged steps are the schedule; everything after them is an adjustment, and adjustments wait a week. Two distinctions carry the marks and the safety. The first is the band boundary itself: less than 50 kg against 50 kg or more, so that a child of exactly 50 kg is not left between two regimens. The second is target against ceiling: for the heavier band 10 mg/day is the recommended dose and 20 mg/day is a ceiling for those who do not respond, not a destination. A bare "5 to 20 mg/day", carried into a viva or printed on a plate without its bands, licenses double the labelled maximum in a patient under 50 kg. The trial evidence supports both the drug and the low starting doses: in a placebo-controlled trial of children and adolescents with Tourette's, both a low-dose and a high-dose aripiprazole arm reduced tics, with roughly 69% much or very much improved on low dose and 74% on high dose against 38% on placebo, at a modest average weight gain.

Risperidone is also effective for tics, but off-label and with no licensed tic dose. Its licensed indications are schizophrenia in adolescents from 13 to 17 and in adults, acute manic or mixed episodes of bipolar I disorder from 10 to 17 and in adults, and irritability associated with autistic disorder from 5 to 16; tics are nowhere among them, and each of those indications carries its own separate dose. So the only risperidone figure that may honestly be quoted for tics is a trial's descriptive number, answering a question about the evidence, not about how to prescribe: in the landmark tic trial the mean effective dose settled at about **2.5 mg/day** over 8 weeks and cut the **Yale Global Tic Severity Scale** Total Tic score by about 32%, with a mean weight gain of 2.8 kg that captures the class liability in a single number. That Total Tic score is the standard primary endpoint against which tic treatments are judged. Risperidone is probably more likely than placebo to reduce tic severity, at a higher risk of drug-induced movement disorders, weight gain and somnolence. Say "the trial mean was about 2.5 mg/day, a trial mean rather than a regimen" and you have answered honestly. The figure carries no starting dose, no titration increment, no dosing interval and no maximum, which is what separates a descriptive number from a prescribable one. Offer instead the low paediatric range that circulates for risperidone and you have quoted a general envelope belonging to the autism and mania indications, sitting below the anchoring tic trial's own mean effective dose, which is the tell that it was never a tic dose.

GOOD TO REMEMBER

With any antipsychotic used for tics, the dosing method is start low, titrate slowly and monitor deliberately, because the tic dose is often lower than the antipsychotic dose you would reach for psychosis and because a growing child pays the metabolic bill. Track weight and metabolic parameters from the outset; the full adverse-effect and monitoring profile of these agents is developed with the antipsychotics themselves, but the weighing starts on the first prescription.

33.5 The classical agents: haloperidol and pimozide, and the ECG

The older high-potency agents still suppress tics powerfully, but tolerability and cardiac safety have moved them down the ladder. Haloperidol is FDA-approved for the control of tics and vocal utterances of Tourette's disorder in children and adults, and its labelled paediatric regimen is narrow: it is written for children between 3 and 12 years of age, in the weight range 15 to 40 kg, and is not intended for a child under 3. Within that band, therapy begins at the lowest dose possible, 0.5 mg/day, and is raised by increments of 0.5 mg at 5 to 7 day intervals until the desired therapeutic effect is obtained, with the total daily dose divided and given twice or three times a day, working toward a target of 0.05 to 0.075 mg/kg per day for Tourette's and the other nonpsychotic behaviour disorders, a deliberately lower target than the 0.05 to 0.15 mg/kg per day used for psychotic disorders. The maximum is an honest absence rather than a number: no maximum effective dosage has been established, and there is little evidence that improvement is further enhanced beyond 6 mg/day. That is a ceiling on expected benefit, not a licence to climb toward it. Haloperidol is often poorly tolerated, with sedation and extrapyramidal effects.

Pimozide, a classical D2 antagonist reserved for patients who have failed standard treatment, is used to suppress motor and phonic tics at a usual range kept under **10 mg/day**. Reliable dose-

response data for its effect on tics below the age of twelve are not available, which is itself worth saying aloud before prescribing it to a younger child. In children it is started at 0.05 mg/kg per day, preferably taken once at bedtime, and may be increased every third day to a maximum of 0.2 mg/kg per day, not to exceed 10 mg/day.

Those dose mechanics are governed by a pharmacogenomic gate, the part candidates omit. At doses above 0.05 mg/kg per day, CYP2D6 genotyping should be performed; in a poor CYP2D6 metaboliser the dose should not exceed 0.05 mg/kg per day and should not be increased earlier than 14 days. Roughly 5 to 10% of the population are poor CYP2D6 metabolisers, and they reach pimozide concentrations similar to those seen with a strong CYP2D6 inhibitor such as paroxetine; because their half-life is prolonged, steady state takes about 2 weeks to arrive, so alternative dosing strategies are recommended for them. The standard titration, moving every third day, therefore outruns the drug in exactly the patients who accumulate it, which is why the interval stretches to a fortnight rather than the dose merely being capped.

The safety-critical point is cardiac. ECG monitoring is essential for both haloperidol and pimozide when they are used for tics. Pimozide in particular causes dose-dependent QTc prolongation with a real risk of ventricular arrhythmia and sudden death, so before and during treatment one must obtain a baseline ECG and serum potassium and monitor periodically, especially during titration. Sudden unexpected deaths have occurred at high doses and patients must not exceed the prescribed dose. The interactions must be filed under the right enzyme and at the right severity. Pimozide is metabolised mainly by CYP3A4 and, to a lesser extent, by CYP1A2 and CYP2D6. Inhibitors of CYP3A4, including macrolides, azole antifungals and grapefruit juice, raise pimozide levels and arrhythmia risk. Strong CYP2D6 inhibitors are a different matter: paroxetine and the other strong CYP2D6 inhibitors are contraindicated with pimozide outright, pimozide 2 mg given with paroxetine 60 mg having raised pimozide AUC by 151% and Cmax by 62%. Filing paroxetine under CYP3A4 as a mere caution gets both the enzyme and the severity wrong, and the pairing is a live one: a child with tics and comorbid obsessive-compulsive disorder is exactly the patient in whom an SSRI and pimozide might otherwise meet.

33.6 Measuring response and assembling the plan

A defensible answer sets out the whole staged plan and says how response is judged. Response is tracked against a validated tic-severity endpoint rather than a clinician's impression, which is what lets a service tell genuine improvement from the natural waning of the tics. Put together: assessment and psychoeducation, comorbidity treated, habit reversal as the active first line, an alpha-2 agonist when a drug is needed, an antipsychotic for severe or refractory tics, and the classical high-potency agents held in reserve under cardiac monitoring. The figure and the table below set the agents against their line and their tic-indication dose. Read both with the same question in hand: for this agent, is there a licensed tic dose at all, and if not, what exactly am I quoting?

AGENT	PLACE IN THE LADDER	TICS: THE LICENSED DOSE, OR WHAT THE FIGURE ACTUALLY IS	CHIEF CAVEAT
Clonidine	Alpha-2 agonist; first drug step	Off-label; no licensed tic dose. No licensed start, interval or maximum for tics; UK labelling does not recommend clonidine under 18 at all	Sedation, postural hypotension; monitor heart rate and blood pressure; taper, never stop abruptly
Guanfacine	Alpha-2A agonist; first drug step	Off-label; no licensed tic dose and no tic-scoped milligram figure	Less sedating than clonidine, but extended release requires QTc monitoring where there is cardiac history, other QT-prolonging drugs or a family history of long QT; taper
Aripiprazole	Antipsychotic; for severe or refractory tics; the tic-licensed choice at this step	Under 50 kg: 2 mg/day, then 5 mg/day, maximum 10 mg/day. 50 kg or more: 2 mg/day, then 5 mg/day, target 10 mg/day, maximum 20 mg/day; steps no closer than a week apart	Metabolic effects, weight gain; the 5 to 20 mg/day envelope is never quoted without its bands
Risperidone	Antipsychotic; off-label for tics	Off-label; no licensed tic dose. The only figure is one trial's mean daily dose over 8 weeks, about 2.5 mg/day, and that figure carries no start, no titration and no maximum	Weight gain, hyperprolactinaemia, drug-induced movement disorders
Haloperidol	Classical agent; second-line; FDA-approved for tics	Children 3 to 12 years, 15 to 40 kg: start 0.5 mg/day, increments of 0.5 mg at 5 to 7 day intervals, divided twice or three times daily; target 0.05 to 0.075 mg/kg per day; no established maximum effective dose	Poorly tolerated; ECG monitoring essential
Pimozide	Classical agent; refractory only; FDA-approved for tics in patients who have failed standard treatment	Usual range under 10 mg per day; children start 0.05 mg/kg per day at night, raise every third day, maximum 0.2 mg/kg per day or 10 mg/day	Dose-dependent QTc prolongation; baseline ECG and potassium; CYP2D6 genotyping above 0.05 mg/kg per day; paroxetine contraindicated

Each agent's place in the sequence, its tic-indication dose where a licensed one exists and the caveat that governs its use; the behavioural first line precedes every row.

IN INDIA

The behavioural first line is the hardest part of this ladder to deliver here. Therapists trained in habit reversal and Comprehensive Behavioural Intervention for Tics are scarce outside a few academic centres, so in routine practice the active first line is often pragmatic psychoeducation, urge-awareness coaching and parent guidance built on the same principles rather than a manualised programme. One caution about the approvals named above: they are United States labelling, and the Indian licensing position for these agents was not verified for this chapter and must not be inferred from them. Where an Indian insert differs, it is the insert that governs the prescription written in India. What does travel is the order of the ladder. Reaching for a familiar antipsychotic first trades a fluctuating, often self-limiting condition for the metabolic and, with pimozide, the cardiac liabilities of a dopamine blocker, the inversion the evidence warns against.

Under 18 CLONIDINE IS NOT RECOMMENDED IN UK LABELLING	**31%** TIC REDUCTION, GUANFACINE TRIAL	**10 and 20 mg/day** ARIPIPRAZOLE TIC MAXIMUM, UNDER 50 KG AND 50 KG OR MORE	**under 10 mg/day** PIMOZIDE USUAL RANGE

34 · Differentiation of tics from stereotypies, chorea, dystonia and compulsions

A tic almost never announces itself with a label, and the child in front of you has simply been brought in for "abnormal movements", which is why the whole of this topic is a bedside discrimination and not a piece of knowledge. Tics belong to the wider family of childhood hyperkinetic movements, and the movements they are confused with - motor stereotypies, chorea, dystonia, myoclonus and, at the complex end, obsessive-compulsive rituals - each carry a treatable and prognostically different cause. Getting the movement wrong sends the child down the wrong pathway: a stereotypy worked up as a tic, a chorea missed as a habit, a dystonia treated as a quirk. The marks here are won not by naming the disorders but by knowing the single feature that pulls each one apart from a tic at the bedside.

This account owns the formal differentiation of tics from stereotypies, chorea, dystonia, myoclonus and compulsions. The diagnostic criteria of Tourette's disorder, the phenomenology of the premonitory urge and suppressibility, the classification of the tic disorders, and the management of tics each belong to their own accounts elsewhere in this chapter and are named here only as far as the discrimination needs them. Obsessive-compulsive disorder in its own right is developed in the paediatric obsessive-compulsive account; the general disorder is the Anxiety, OCD and trauma-related disorders notes'.

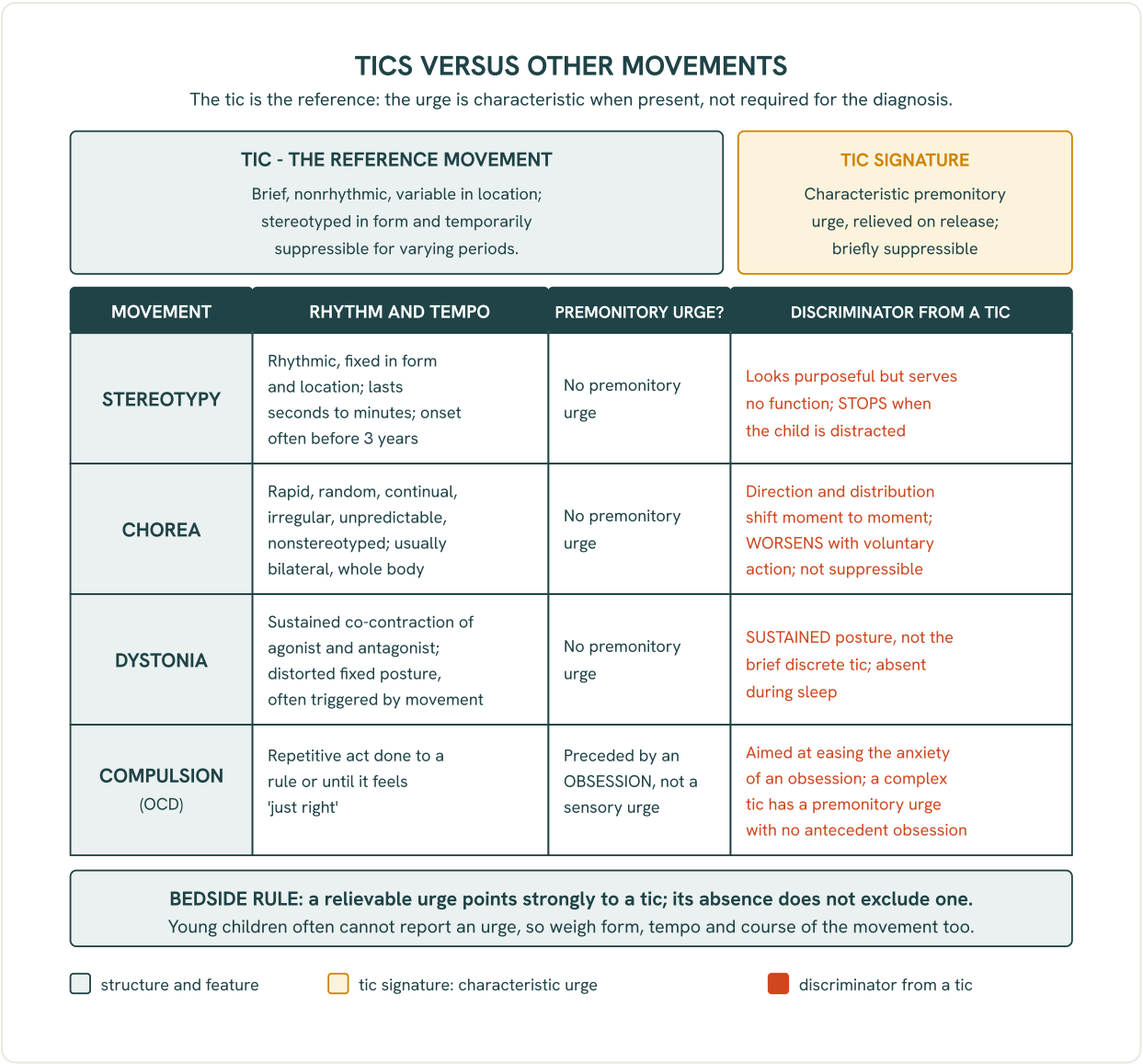

Fig 23.23. Tics versus other movements: the premonitory urge, characteristic of a tic when the patient can report it, and the brief suppressibility that goes with it, set against the discriminating feature of motor stereotypy, chorea, dystonia and obsessive-compulsive compulsion.

34.1 The signature of a tic: the reference frame for every comparison

Every discrimination below is really the same question asked six ways - does this movement carry the signature of a tic? A **tic** is a sudden, brief, non-rhythmic movement or vocalisation, and three linked features together form its signature. First, many tics are preceded by a **premonitory urge**, an uncomfortable localised sensation or inner tension that builds and is momentarily relieved by the movement; most patients old enough to describe one do, but younger children often cannot report it and not every tic carries it. Second, a tic shows partial **suppressibility**: the patient can hold it back for varying periods, at the cost of rising tension. Third, its form is stereotyped and recognisable yet its location and frequency vary over time. No other movement disorder in this differential carries all three, and a reported urge marks a movement as a tic more reliably than anything else, because it is a subjective report of an internal drive the involuntary hyperkinesias do not generate. An absent urge, though, never excludes a tic. The full teaching of the urge sits in the Tourette phenomenology account; what matters here is that you carry it as the yardstick against which each mimic is measured.

- Is there a premonitory urge, relieved by the movement? A reported urge points to a tic; the involuntary movement disorders generate none.
- Can the movement be briefly suppressed on request? A tic can; chorea and myoclonus cannot.
- Is the movement brief and non-sustained? A tic is; a dystonic posture is held.

RULE **The premonitory urge and brief suppressibility are the signature of a tic, and a positive report of them all but settles this differential.** A movement preceded by a relievable inner urge and capable of temporary suppression is a tic until proven otherwise; a movement that is purely involuntary, cannot be held back at all and carries no antecedent sensation is one of its mimics. Their absence carries far less weight: a young child may be unable to report an urge, so a negative answer sends you to form, tempo and course rather than out of the diagnosis.

MNEMONIC

Urge before, suppress on demand, brief by nature

The three-part signature in order: an **urge before** the movement that it relieves, where the patient can report one; the capacity to **suppress on demand** for a time; and a **brief, non-sustained** form. Test any suspicious movement against these three and the mimics fall away.

34.2 Tics versus motor stereotypies

This is the commonest and most examinable confusion, because both look patterned and both occur in children. **Motor stereotypies** are repetitive movements such as hand flapping, body rocking or finger wiggling that appear purposeful but serve no adaptive function. Several features separate them cleanly from tics. Stereotypies have an earlier onset, often younger than **three years**, whereas tics typically begin later in childhood. A single bout runs **seconds to minutes**, far longer than the flick of a tic. Crucially, a stereotypy is rhythmic and fixed in form and location, repeating the same movement in the same place, while a tic is brief, non-rhythmic and variable. And a stereotypy lacks any premonitory urge. The bedside discriminator that settles it is response to distraction: calling the child's name or touching them stops a stereotypy, because the child is typically absorbed and unbothered, whereas a tic is not abolished this way. Stereotypies are common in typically developing young children and more so in those with intellectual disability or autism; a child engrossed in a rhythmic, fixed pattern that a call to attention interrupts is showing a stereotypy, not a tic.

PITFALL

The costly error is to read a young child's rhythmic hand flapping or rocking as a tic and reach for a tic medication, or conversely to dismiss genuine tics as "just a stereotypy" and withhold assessment. Stereotypies rarely need pharmacotherapy and often settle with reassurance and behavioural strategies; misclassifying them exposes a child to antipsychotic or alpha-adrenergic agonist treatment they do not need. When form is fixed, onset is early and a call of the name interrupts the movement, think stereotypy before tic.

34.3 Tics versus chorea

Chorea is separated from a tic by its randomness and its relationship to movement. Chorea consists of movements that are rapid, random, continual, abrupt, irregular, unpredictable and non-stereotyped, usually bilateral and affecting all body parts, and that worsen during attempted voluntary action. Set that against the tic's signature and the contrast is sharp: a tic is stereotyped and can usually be held back for a time, and most patients able to report one describe a premonitory urge, whereas chorea has no fixed pattern, is not stereotyped, is usually bilateral across face, trunk and limbs, and its timing, direction and distribution shift from one moment to the next. The urge is asymmetric evidence and is worth using as such: a reported urge argues strongly for a tic, because the classification explicitly records the ABSENT urge for the two mimics where it states a position, stereotypies and myoclonus. It states no position on a premonitory sensation in chorea at all, so the absence of an urge argues nothing here in either direction, and against chorea the discriminators to lean on are the ones just given, its randomness, its lack of stereotypy and its worsening during attempted voluntary action. The dance-like flow of chorea across the body, flitting from limb to face to trunk without repeating itself, is nothing like the discrete, repeatable jerk of a tic. The most useful positive sign is that chorea disrupts voluntary movement, so attempts to hold a posture or perform a task fragment into extra involuntary flourishes - an active worsening rather than the tension-and-release cycle of a tic.

IN INDIA

Chorea is not an academic differential here. Sydenham chorea, the neurological manifestation of acute rheumatic fever following streptococcal infection, remains common in Indian children because rheumatic fever and rheumatic heart disease are still endemic, and it presents in the same paediatric age group in which tics appear. A new choreiform movement in an Indian child therefore mandates a search for a treatable systemic cause - a history of sore throat or migratory arthritis, examination for carditis, and the relevant work-up - rather than being logged as a tic. Missing it means missing rheumatic carditis, which is why the tic-versus-chorea distinction carries more weight here than the Western default suggests.

34.4 Tics versus dystonia

Dystonia is separated from a tic by duration and by the simultaneous pull of opposing muscles. Dystonia is the simultaneous sustained contraction of both agonist and antagonist muscles, producing a distorted posture or movement, often triggered by voluntary movement and not seen during sleep. The word to hold onto is *sustained*: where a tic is brief and non-sustained, a dystonic contraction locks the body part into an abnormal posture that is held. The co-contraction of agonist against antagonist generates that twisting, cramped quality and is mechanically the opposite of the clean single-direction flick of a simple motor tic. Two contextual features help: dystonia is frequently brought on by a particular voluntary movement, and it typically disappears in sleep. A movement that produces a held, distorted posture from opposing muscle pull is dystonia; one that is over almost as soon as it begins is a tic.

34.5 Tics versus myoclonus

Myoclonus is the supplementary movement-disorder differential, and it turns on the same two anchors. **Myoclonus** is a sudden, unidirectional, often non-rhythmic movement that may occur during sleep. It resembles a tic in being abrupt and shock-like, which is why it earns a place in the differential, but it is distinguished by its rapidity, its lack of suppressibility and the absence of a premonitory sensation or urge. Apply the signature: myoclonus cannot be voluntarily held back and carries no relievable inner urge, so both of the tic's defining subjective and volitional features are missing. That it may appear during sleep is a further pointer, locating it outside the semi-voluntary domain. When an abrupt jerk has neither an antecedent urge nor any capacity for suppression, and especially if it occurs in sleep, it is myoclonus and not a tic.

34.6 Complex tics versus obsessive-compulsive compulsions

This is the hardest separation, because a complex tic and a compulsion can look identical and the answer lies not in the movement but in what drives it. A **compulsion** in obsessive-compulsive disorder is aimed at preventing or reducing anxiety or distress and is performed in response to an obsession, for instance a washing ritual driven by a fear of contamination. A complex tic, by contrast, is driven by a premonitory urge and a need to perform the action until it feels "just right", without any antecedent obsession. The discriminating question is therefore about the antecedent: ask why the movement is done. If it neutralises a specific frightening thought, it is a compulsion; if it discharges a rising physical urge to reach a "just right" resolution with no idea being warded off, it is a complex tic. The difficulty is real, because complex tics and compulsions frequently co-occur and can be genuinely hard to separate, and many patients with a tic disorder also meet criteria for obsessive-compulsive disorder. The clinical resolution is to characterise each repetitive behaviour by its antecedent rather than by its appearance.

TRAP **Candidates try to separate a complex tic from a compulsion by how the movement looks; the discriminator is the antecedent.** Both can be elaborate, repeated and semi-purposeful, so appearance settles nothing. A compulsion answers an *obsession* and is done to reduce anxiety; a complex tic answers a *premonitory sensory urge* and is done until it feels "just right", with no frightening thought behind it. Examine the driver, not the choreography.

34.7 Putting the differential together at the bedside

Laid side by side on shared axes, the six movements separate cleanly on the two anchors and on a single cardinal feature each. The discrimination is made from a careful description of the movement and, where possible, direct observation or a home video, watching the child's response to distraction, to a request to suppress, and to attempted voluntary action.

MOVEMENT	DEFINING QUALITY	CARDINAL CONTRAST WITH A TIC
Tic	Sudden, rapid, recurrent, non-rhythmic and stereotyped, variable in location	The reference movement: urge-preceded when reportable, briefly suppressible
Motor stereotypy	Rhythmic, fixed in form and location, no urge, stops with distraction	Earlier onset and fixed pattern; interrupted by a call of the name
Chorea	Rapid, random, irregular, unpredictable, non-stereotyped; worsens with voluntary action	No fixed pattern and no urge; disrupts voluntary movement
Dystonia	Sustained co-contraction of agonist and antagonist; distorted held posture; absent in sleep	Sustained, not brief; a held abnormal posture
Myoclonus	Sudden, unidirectional, often non-rhythmic; may occur in sleep	Not suppressible and no premonitory urge
Compulsion	Performed in response to an obsession to reduce anxiety or distress	Antecedent is an obsession, not a sensory urge

The six movements of the differential on shared axes; the premonitory urge and suppressibility resolve most cases, and the final column gives the one feature that clinches each mimic.

GOOD TO REMEMBER

When a description alone leaves the movement ambiguous, do three things. Ask the older child whether a feeling builds before the movement and eases after it, since a positive report of a relievable urge is strong evidence for a tic, while a younger child unable to answer tells you nothing either way. Ask them to hold the movement back and watch for the tension-and-release of suppressibility. And observe it during an absorbing task and during a call to attention, since chorea worsens with voluntary action while a stereotypy stops when the child is addressed. A short home video often shows more than a clinic encounter.

Carry into the exam: a tic is defined by its form, sudden, rapid, recurrent, non-rhythmic and stereotyped, while the premonitory urge and partial suppressibility are its characteristic accompaniments, reported by most individuals but never required for the diagnosis - stereotypies are fixed, early-onset and stop with distraction; chorea is random and worsens with voluntary action; dystonia is a sustained held posture; myoclonus is unsuppressible; a compulsion answers an obsession while a complex tic answers a sensory urge.

Urge
CHARACTERISTIC OF A TIC, NOT REQUIRED FOR IT; RECORDED AS ABSENT IN STEREOTYPIES AND MYOCLONUS

Suppress
A TIC CAN BE BRIEFLY HELD BACK; CHOREA AND MYOCLONUS CANNOT

Brief
A TIC IS NON-SUSTAINED, UNLIKE THE HELD POSTURE OF DYSTONIA

< 3 yr
TYPICAL STEREOTYPY ONSET, EARLIER THAN MOST TICS

Elimination and feeding disorders

35 · Enuresis (types, etiology, assessment)

Bedwetting is the point at which normal development, organic disease and psychological disturbance all converge on a single symptom, and the marks are here because the candidate has to separate them. **Enuresis** is the repeated voiding of urine into inappropriate places - the bed or the clothing - in a child old enough that continence should by now have been achieved and in whom no medical cause accounts for the wetting. What makes it examinable is not the symptom, which any parent can describe, but the scaffolding around it: an age threshold below which the same wetting is developmentally normal, a frequency-or-distress requirement, a mandatory exclusion of organic disease, and a set of subtype and course distinctions that decide how the child is investigated and, later, treated.

This account owns the definition and diagnostic criteria of enuresis, its classification into subtypes, the pathophysiology that drives it, and the assessment that flows from the criteria. It does not teach the treatment: the behavioural measures, the enuresis alarm and the pharmacotherapy are taken up in their own account within this chapter, and the drug doses in particular are set out there and deliberately not restated here. Toilet-training norms and the ordinary timetable of continence belong to the Normal child development and milestones notes; what follows marks only the point at which the child has crossed from lateness into disorder.

35.1 The essential feature: Criterion A and the voiding itself

Criterion A defines the act, and it is written broadly on purpose. The requirement is the repeated voiding of urine into the bed or into clothes, and the manual is explicit that this may be either involuntary or, occasionally, intentional. The great majority of enuretic wetting is involuntary, and that is the default picture; but by admitting the rare intentional case the definition avoids excluding a child who, out of anger or protest, wets deliberately. The point behind the wording is that enuresis is defined by the event, not by the child's motive: the diagnosis turns on misplaced voiding at an age when it should not occur, and whether it was willed is a matter for formulation, not for the diagnostic threshold.

35.2 Criteria B and C: how often, for how long, and from what age

Criterion B sets the bar the wetting must clear, and it has two routes to satisfaction that candidates routinely collapse into one. The first route is one of frequency and persistence: the voiding must occur at least **twice a week** for a minimum of three consecutive months. The second route dispenses with the count altogether: even below that frequency, the diagnosis is met if the wetting produces clinically significant distress or impairment in social, academic or other important areas of functioning. A once-weekly wetter who is refusing school trips and sleepovers and is being shamed at home meets Criterion B by this second limb even though the frequency route is not reached.

The age criterion is where the diagnosis is separated from normal development. Criterion C requires that the child has reached a chronological age of at least **five years**, or, where

development is delayed, an equivalent developmental (mental) age of the same level. The floor is set at the point by which the ordinary child has established daytime and night-time control, so that wetting below it is lateness rather than disorder. Anchoring the threshold to developmental rather than chronological age matters in any child with global delay, in whom continence tracks developmental level and not birthdays: a chronologically seven-year-old functioning well below the five-year level has not failed at a task they were equipped to master, and should not be labelled.

TRAP **Reciting only "at least twice a week for three months" and forgetting the distress route.** Criterion B is satisfied by the frequency-and-duration standard OR by clinically significant distress or impairment at a lower frequency. Candidates who memorise the numeric limb alone will wrongly exclude the distressed, low-frequency wetter, who is precisely the child who reaches the clinic. The threshold is a disjunction, not a single rule.

35.3 Criterion D: the exclusion that becomes the assessment

Criterion D is a single clause that generates almost the whole of the medical work-up. The wetting must **not be attributable to the physiological effect of a substance or to another medical condition**. The substances named are the diuretics and the antipsychotics; the medical conditions span the endocrine, the structural and the neurological. Because enuresis is reached only after organic disease is cleared, the criterion is really an instruction to investigate, and the differentials it forces are the ones a careless clinician misses:

- endocrine causes of a genuine polyuria - diabetes mellitus and diabetes insipidus - in which the bladder is wet because urine output is genuinely high;
- urinary tract infection, the commonest reversible medical cause of new wetting, especially where there are daytime symptoms;
- structural anomalies of the lower urinary tract - an ectopic ureter, posterior urethral valves - which produce continuous or unusual wetting;
- neurological lesions of the spinal cord - spina bifida and a tethered cord - that disturb bladder innervation;
- a seizure disorder, where nocturnal incontinence is a manifestation of the seizure rather than of enuresis;
- an iatrogenic effect of a prescribed diuretic or antipsychotic.

RULE **Enuresis is a clinical diagnosis of exclusion made only from a defined age.** Two gates must both be passed: the child must be at least five years old chronologically or developmentally, and an organic cause - endocrine, infective, structural, neurological or drug-induced - must have been excluded. Skip either and the diagnosis is unsafe: below the age gate it pathologises normal development, and without the exclusion it renames a treatable medical illness as a behavioural one.

35.4 The subtypes by timing

The primary classification is by the time of day the wetting occurs. The condition is specified into three subtypes according to timing. The **nocturnal-only** form, in which urine is passed only

during night-time sleep, is much the commonest and is the classic monosymptomatic bedwetting of childhood. The **diurnal-only** form occurs during waking hours and, being daytime wetting, more often signals bladder dysfunction or a psychological problem. The third form combines both.

SUBTYPE	WHEN WETTING OCCURS	CLINICAL SIGNIFICANCE
Nocturnal only	Only during night-time sleep	Commonest form; typically the isolated bedwetting driven by the polyuria, bladder-capacity and arousal factors
Diurnal only	During waking hours	Daytime wetting; more likely to reflect bladder overactivity, voiding habit or emotional disturbance
Nocturnal and diurnal	Both daytime and night-time	The combined presentation, also termed the non-monosympto-matic form

The three timing-based subtypes of enuresis; the nocturnal-only form dominates in frequency, while daytime wetting shifts the differential toward bladder and psychological factors.

35.5 Monosymptomatic and non-monosymptomatic: the distinction that guides investigation

Cutting across the timing subtypes is a distinction the urological literature considers more useful still. **Monosymptomatic** enuresis is isolated night-time wetting with no daytime lower-urinary-tract symptoms - no urgency, frequency or daytime incontinence. **Non-monosymptomatic** enuresis is wetting accompanied by such daytime symptoms, and the combined nocturnal-and-diurnal subtype is the presentation this term describes. The distinction steers the work-up: the monosymptomatic child is usually the straightforward bedwetter, whereas daytime symptoms raise the suspicion of bladder pathology or infection. Within the daytime picture the manual recognises two mechanisms: **urge incontinence**, from an overactive, unstable detrusor that contracts before the child reaches a toilet, and **voiding postponement**, the habitual deferral of micturition until the bladder overflows, often in a child absorbed in play. They look alike at the moment of wetting and are told apart by the history of what precedes it.

35.6 Primary and secondary enuresis

A second course-based axis asks whether continence was ever attained. **Primary enuresis** is the form in which urinary continence has never been established, so that the wetting continues unbroken from infancy; by definition it can first be diagnosed at the age of five, the point at which the failure becomes a disorder. **Secondary enuresis** is the reappearance of wetting after a period of established dryness, its onset falling most commonly between the ages of five and eight but possible at any point in childhood. The clinically important fact is a negative one: there is no difference between the two in the prevalence of associated mental disorders, so the older teaching that secondary enuresis is the psychologically loaded form and primary the benign one is not supported. Secondary onset does, however, direct attention to what changed - a urinary

infection, a new medical illness, or a psychosocial stressor such as the birth of a sibling - because the return of wetting in a previously dry child asks for an explanation.

PITFALL

Treating new-onset secondary wetting as a behavioural relapse without asking what precipitated it. Because the child was previously dry, a fresh medical cause - a urinary tract infection, newly manifest diabetes with its polyuria - or an acute psychosocial stressor is likelier than in the never-dry child. Reaching for a behavioural programme skips the step that matters most in the secondary case: looking for the treatable thing that ended the child's dryness.

35.7 Epidemiology: common, male-predominant, and self-limiting

Enuresis is one of the commonest disorders of childhood, and its epidemiology is defined by a steady fall with age. Community prevalence of night-time wetting runs at around **5 to 10% at age five**, declining to 3 to 5% by age ten and to about 1% by age fifteen and beyond. The trajectory is the teaching point: prevalence falls year on year because the condition remits spontaneously in most children, so what is near-normal at four becomes, in the adolescent, uncommon and clinically weighty. Across these figures boys carry the higher prevalence, one of the more reliable features of the disorder.

5 to 10%	3 to 5%	about 1%	5 to 10%
PREVALENCE AT AGE FIVE	PREVALENCE AT AGE TEN	AT AGE FIFTEEN AND OLDER	REMIT SPONTANEOUSLY EACH YEAR AFTER FIVE

35.8 Etiology: three pathophysiological strands

The modern understanding of nocturnal enuresis is not one cause but the convergence of three, and a given child may sit on any one or a combination of them. The first is a mismatch between how much urine the kidney makes at night and how much the bladder can hold: a blunted nocturnal rise in arginine vasopressin, the antidiuretic hormone whose secretion normally climbs overnight to concentrate the urine, allows an overnight nocturnal polyuria that overwhelms the sleeping bladder. That this pathway is real is shown by vasopressin-deficient children becoming dry when the hormone is replaced, and by responders and non-responders differing in their vasopressin profile. The second strand is the bladder itself: a low **functional bladder capacity** with an unstable, overactive detrusor means the bladder signals fullness and empties at a smaller volume than it should, and a low functional capacity is a recognised predictor of failure to respond to the enuresis alarm.

The third strand frames enuresis as a **disorder of arousal**: the enuretic episode characteristically arises out of deep, delta-wave sleep, and the child fails to wake to the signal of a full or contracting bladder. Sleep studies bear this out - enuretic children received the arousal signal yet did not respond by waking - which reframes the wetting as a failure of arousal rather than of bladder or kidney alone. Voiding occasionally occurs in rapid-eye-movement sleep, when the child may later recall a dream of micturition, but the core deficit is the failure to arouse. The three strands are not rivals: a single child may carry mild polyuria, a modest reduction in bladder

capacity and a high arousal threshold together, and it is their combination that produces the wet bed.

MNEMONIC

Too much water, too small a bladder, too deep a sleep

The three etiological strands of nocturnal enuresis in a line. **Too much water**: the blunted overnight vasopressin rise and its polyuria. **Too small a bladder**: low functional bladder capacity with detrusor overactivity. **Too deep a sleep**: the failure to arouse from delta sleep to the bladder's signal. Any one may dominate, and most children carry a mixture.

35.9 The genetic contribution

Enuresis runs in families, and the familial loading is one of the strongest of any childhood disorder. A child's risk is substantially raised by a parental history: it is about **7.1 times** greater where the father was himself enuretic beyond early childhood, and about 5.2 times greater with a corresponding maternal history, and enuresis is correspondingly commoner in children with an affected parent. The heritability is high enough that a positive family history is both a useful diagnostic pointer - it reassures the family that the mechanism is constitutional rather than wilful - and a fair predictor that the child, like the parent before them, will in time grow out of it. The inherited component is best understood as acting through the mechanisms already described, shaping the overnight hormone rhythm, the bladder's functional capacity and the depth of sleep, rather than as a separate cause standing apart from them.

35.10 Assessment: history and examination first, investigation guided

The assessment is driven entirely by the criteria, and its architecture follows Criterion D. The history establishes the pattern - night, day or both; monosymptomatic or carrying daytime urgency, frequency and incontinence; primary or secondary, since a previously dry child who has begun to wet again is investigated differently from one who was never dry. It quantifies frequency and the distress caused, both of which the diagnosis needs, and takes the informative family history. The examination looks for the structural and neurological signs the exclusion criterion demands, the lumbosacral spine and perineal neurology especially. Investigation is then targeted rather than routine: a urinalysis for the glycosuria of diabetes and the pyuria of infection, with imaging or specialist referral reserved for the child whose daytime symptoms, abnormal stream or examination findings point to structural or neurological disease. A frequency-volume record of daytime voids and overnight wetting is the single most informative bedside tool, exposing both the true nocturnal urine output and the functional bladder capacity, and so pointing to which strand is operating.

GOOD TO REMEMBER

Two cheap steps carry most of the assessment. A urine dipstick excludes the two organic causes that must never be missed - the glycosuria of new diabetes and the leucocytes and nitrites of infection - and a few days of a home frequency-volume chart distinguishes the small-capacity bladder from the polyuric night and flags the daytime symptoms of non-monosymptomatic disease. Both precede any referral and turn a vague complaint of bedwetting into a mechanism you can act on.

35.11 Course, prognosis, and the line with normal development

The natural history is favourable and is itself part of the teaching. After the age of five the condition remits without treatment in a substantial share of children each year - on the order of 5 to 10% annually - so the pool shrinks steadily through childhood, and only about 1% of cases persist into adulthood. This high spontaneous remission is why treatment is weighed against watchful waiting, and why the alarm and drug measures are held for the child in whom wetting is causing real distress or failing to resolve. It also fixes the boundary with normal development: below the age of five, or below the equivalent developmental level, night-time wetting is a normal stage of a not-yet-complete process and is not enuresis at all. The disorder begins where the developmental grace period ends, which is why the age criterion, not the wetting, is the true threshold.

Enuresis is diagnosed only from a chronological or developmental age of five years, requires wetting at least twice a week for three months or else clinically significant distress, and is a diagnosis of exclusion made only once organic causes are ruled out.

35.12 ICD-11 and how the two systems differ

The two classifications agree on the concept but differ sharply in how they quantify it. Where the manual sets a numeric threshold, ICD-11 describes the frequency in words: enuresis is the repeated and persistent voiding of urine into the bed or clothes, occurring, in its wording, several times per week over several months, rather than the quantified twice-weekly-for-three-months standard of the manual. The age concept is expressed developmentally - continence is expected at a developmental age approximately equivalent to a chronological age of five years - and the same three timing subtypes are retained. The practical consequence is that the diagnosis rests on the descriptive "persistent and repeated" wording and on clinical judgement of what counts as developmentally overdue, rather than on a hard count, while the concept, the subtypes and the exclusion of organic disease are held in common with the manual.

IN INDIA

Because services here classify under ICD, the operative entity in the case file and the referral is the ICD one, coded 6C00, and the clinician diagnoses from its descriptive frequency and developmental-age wording rather than from a numeric manual threshold. The presentation is shaped by local realities: bedwetting is frequently normalised or, conversely, punished within the family, help-seeking is often delayed until an older child faces the shame of shared sleeping arrangements or a school trip, and the first port of call is usually the paediatrician rather than the psychiatrist. The clinician's early task is often as much to relieve the blame heaped on a constitutionally predisposed child, whose parent likely wet the bed too, as to investigate.

36 · Management of enuresis (behavioural, bell-and-pad alarm, desmopressin, imipramine)

The marks here sit on a sequence and a safety trap, not on a catalogue of drugs. What follows assumes the child has already been worked up, an organic and a daytime-symptom cause excluded, and the diagnosis of primary nocturnal enuresis secured. From that point the whole of management turns on two ideas. The first is that the least medical option is also the most durable, so the child who can wait is better served by conditioning than by a tablet. The second is that the one genuinely dangerous drug on the ladder is dangerous because the dose that works is so small.

Stated as a single sentence, the rational sequence is **behavioural intervention** first, with the enuresis alarm as its most durable member; then **desmopressin** as the drug reached for most often when a rapid pharmacological effect is needed; and **imipramine**, the **tricyclic antidepressant**, held back for refractory individuals. The classification and causal story upstream belong to the companion account of enuresis.

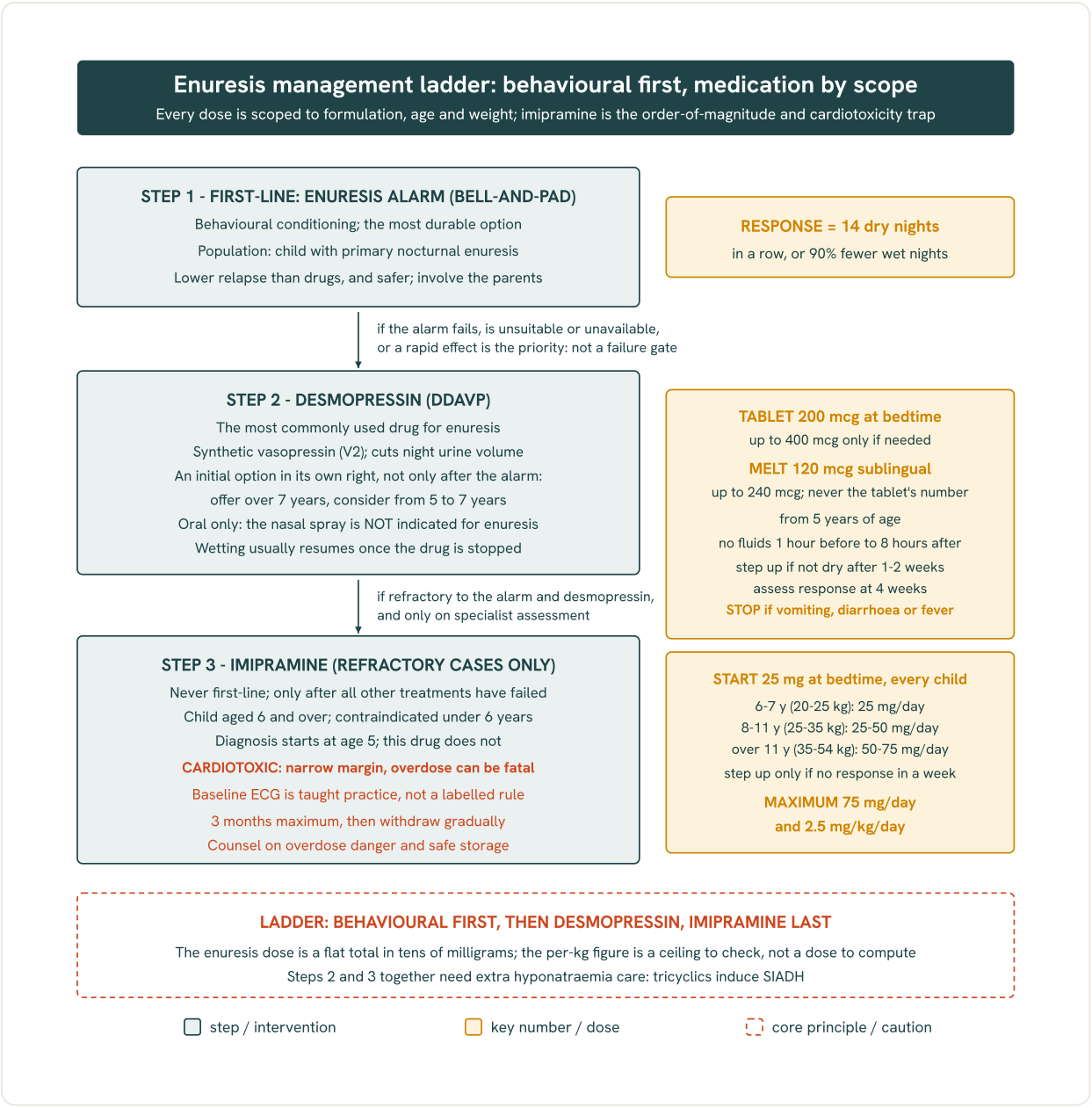

Fig 23.24. Enuresis management ladder: the bell-and-pad alarm is first-line and the most durable option, its benefit outlasting treatment where the drugs' does not, and a response is 14 consecutive dry nights or a 90 percent reduction in wet nights per week; desmopressin is the most-used drug and an initial option in its own right when a rapid effect is the priority or an alarm is unsuitable, given from 5 years of age as a 200 to 400 microgram tablet or a 120 to 240 microgram sublingual melt at bedtime, with fluids withheld from 1 hour before the dose until 8 hours after it, the dose stepped up in the child not completely dry after 1 to 2 weeks and the response assessed at 4 weeks, and the drug stopped during vomiting, diarrhoea or any acute illness upsetting fluid or electrolyte balance until the child is well again; and imipramine is reserved for the refractory child of 6 and over, started at 25 milligrams at bedtime, banded by age and weight, and never taken above 75 milligrams a day or 2.5 milligrams per kilogram per day, for no more than 3 months.

36.1 The ladder, and why its order is the answer

The order encodes a judgement about durability, safety and speed, in that priority. A graded approach begins with the recognition that many children become dry without any active treatment as they grow, so the first duty is to do no harm, to demystify the problem for a family that is often frightened and blaming, and to lift the shame that surrounds it. Against that background the least invasive effective option is preferred, and only when it fails or cannot be used does the clinician step up. A drug is added when the family needs a result faster than

conditioning can deliver it, for instance to get a child through a school trip or a family stay, and here desmopressin is the usual choice. The ladder is a priority order, though, not a queue in which each rung must fail before the next may be touched: desmopressin is an initial treatment in its own right, offered to the child over 7 years, and considered from 5 to 7 years, whenever a rapid or short-term improvement is the priority or an alarm is inappropriate or undesirable. The tricyclic is the one rung that genuinely is gated: it sits at the bottom, not because it does not work, but because it is no longer a first-line pharmacological choice and is reserved for refractory cases, and it is considered only for the child who has not responded to all other treatments and who has been assessed by a clinician with expertise in bedwetting, its cardiotoxicity making it the wrong drug to reach for early.

RULE **Conditioning teaches, drugs only suppress.** The alarm changes what the sleeping bladder does with a full-bladder signal, so its benefit can outlast the treatment; desmopressin and imipramine act only while they are being taken, and wetting characteristically returns once they are stopped. This single distinction is why the alarm leads the ladder and why a drug alone is a holding measure rather than a cure. When a family wants the wetting to end for good, the answer bends toward conditioning; when they want it to pause for a fixed occasion, the answer bends toward a drug.

MNEMONIC

First the bell, then the hormone, last the amine

The **bell** is the bell-and-pad alarm at the head of the ladder; the **hormone** is desmopressin, the vasopressin analogue, as the commonest drug; the **amine** is the tricyclic imipramine, kept last for the refractory child. The order of the phrase is the order of escalation, and it also tracks safety.

36.2 The behavioural foundation before any device or drug

Simple measures resolve a meaningful share of cases and make every later step work better. Before an alarm is fitted or a prescription written, a set of general behavioural measures is put in place, and in the milder or younger child they may be all that is needed.

- Motivational and educational work with the child and family, reframing the wetting as an involuntary developmental lag rather than laziness or defiance, which removes the punishment that entrenches it.
- Fluid and voiding scheduling: adequate daytime drinking, tapering intake in the evening, and a reliable bladder emptying at bedtime.
- Simple positive reinforcement of dry nights, the star-chart family of methods, in which the child records and is rewarded for success rather than blamed for failure.
- Bladder-training and lifting or scheduled-waking strategies in selected children, layered on when the basic measures are insufficient.

This is the substrate on which the alarm succeeds: a child who is engaged, unpunished and motivated will persist with a demanding conditioning programme, and a punished, ashamed

child will abandon it. It carries no risk at all, which is why it is never skipped even when a drug is going to be used.

36.3 The bell-and-pad alarm: the most durable single treatment

The alarm is a conditioning device, and its case rests on durability rather than on a headline success rate. The **bell-and-pad, or enuresis alarm**, works by classical conditioning: a moisture-sensitive sensor triggers a loud alarm at the very start of voiding, the child wakes and completes the void in the toilet, and over repeated pairings the sensation of a full bladder comes to substitute for the alarm as the waking or inhibiting signal. It is the first-line treatment once advice on fluids, toileting and an appropriate reward system has not worked, and its standing on the ladder rests on three comparative facts.

The first is that it gives up nothing while it is running: set against desmopressin, there is little or no difference in the number of children reaching a response by the end of treatment. The second is where the difference actually lies. Once treatment stops, the alarm may leave more children dry at follow-up than desmopressin does, and against imipramine the contrast is starker still, because the tricyclic's effect is not sustained after withdrawal and most children relapse. That persistence is the durability the drugs cannot match. The third is safety, and it is mechanical rather than statistical: a conditioning device cannot cause a hyponatraemic convulsion and cannot be taken in overdose.

One honest caution about the figures. The success percentages that older textbooks still print for the alarm come from reviews that the current Cochrane updates have replaced, and they move a great deal with how success was defined in the first place. The current reviews report relative effects on low-quality evidence and name no single success rate, and NICE says only that alarms have a high long-term success rate. What NICE does give is a definition rather than an effect estimate: a response is **14 consecutive dry nights**, or a **90 percent** improvement in the number of wet nights per week. The response to an alarm is assessed by 4 weeks, and treatment is stopped only if there are no early signs of response at all.

The alarm's chief limitation is that it demands sustained effort over weeks from an already tired family. Two features predict that it will fail: a low **functional bladder capacity**, and an inability to be aroused from sleep.

> **PITFALL**
>
> The commonest way the alarm fails in practice is not that it does not work but that it is abandoned too soon. Families expect a quick result, hear the alarm wake the whole household for several nights running with no dry mornings, and give up before the conditioning has had time to consolidate. Counselling that the method takes weeks of consistent use, and that early alarms without dry nights are the process working rather than failing, is what makes the device work.

36.4 Desmopressin: what it is and what it does

Desmopressin is a hormone analogue, not a sedative or a bladder drug. Desmopressin acetate, or DDAVP, is the most commonly used pharmacological intervention for primary nocturnal

enuresis. It is a synthetic vasopressin V2-receptor analogue that acts on the renal collecting duct to concentrate the urine and so reduce the volume of urine produced overnight. Because the drug corrects the water-handling rather than teaching the bladder anything, its effect is entirely dependent on continued administration. Wetting typically resumes once the drug is stopped, and the durable benefit off treatment is correspondingly low: on current evidence the alarm may leave more children dry at follow-up than desmopressin does. Against placebo the drug may reduce wet nights by roughly one to two a week, on low-certainty evidence, and probably increases the chance of achieving 14 consecutive dry nights, RR 3.18 (95% CI 1.75 to 5.80), on moderate-certainty evidence, and a raised chance of an endpoint is not a dry night promised to a particular child on a particular night. Guidance requires the family to be told, in these terms, that many children but not all will experience a reduction in wetness, and that many but not all will relapse when the drug is withdrawn.

36.5 Desmopressin: route, formulation and dose

Route is now a safety decision, and the oral route has won it. Desmopressin can be given intranasally or orally, and the modern position is that the oral route is preferred. The nasal spray is not a deprecated option but a withdrawn one: it is not an indicated formulation for primary nocturnal enuresis at all, because postmarketing reports showed a higher risk of hyponatraemia and hyponatraemic convulsions with the nasal spray than with desmopressin tablets. There is therefore no current bedtime nasal dose to carry for this indication.

The tablet and the melt are different strengths, and giving one the other's number is the error to avoid. Oral desmopressin is for the child from 5 years of age with normal urine-concentrating ability. The plain tablet is started at **200 micrograms at bedtime and increased to 400 micrograms** only if the lower dose proves insufficient, and the timing of that step matters: **the increase is considered in the child who is not completely dry after 1 to 2 weeks of the initial dose**, and **the response is assessed at 4 weeks**, **treatment being continued for 3 months** where there are signs of one and stopping considered where there are none. The sublingual melt is a different preparation at a different strength: it is started at **120 micrograms sublingually at bedtime and increased to 240 micrograms**, because 120 micrograms of melt is bioequivalent to 200 micrograms of tablet. Write the tablet's number on a melt prescription and the child receives about one and two-thirds of the intended exposure of the drug whose signature harm is a dose-dependent hyponatraemic convulsion. One jurisdictional divergence matters: United Kingdom labelling caps the tablet at 400 micrograms, while United States labelling permits titration to a higher maximum at bedtime. The lower ceiling is the one carried here. Continued treatment is not open-ended either: the need for it is reassessed after 3 months by means of a period of at least a week without the drug.

36.6 Desmopressin: the hyponatraemia caution that governs its use

The whole safety profile of desmopressin reduces to one preventable event. The most serious, if rare, adverse effect is **hyponatraemia**, and it can progress to seizures. It is the direct pharmacological consequence of the drug's action: a V2-mediated water retention that, if the child then drinks freely, dilutes the serum sodium into a dangerous water intoxication. The

management follows from the mechanism, and the rule is a window of time rather than an allowance of volume: **fluid intake must be limited to a minimum from 1 hour before the dose until the next morning, at least 8 hours after it**. Treatment without that reduction in fluid may lead to water retention and hyponatraemia, with or without the warning signs of headache, nausea and vomiting, weight gain and, in severe cases, convulsions; if they appear, treatment is interrupted until the child has fully recovered and the fluid restriction is enforced strictly on restarting. That symptom-triggered stop is the later of two stop rules. The tablet's labelling requires it to be discontinued during an episode of vomiting or diarrhoea until the child's fluid balance is once again normal, and the melt's labelling requires treatment to be interrupted during any acute intercurrent illness characterised by fluid or electrolyte imbalance, systemic infection, fever and gastroenteritis being the named examples. The fluid window and the warning signs both assume a well child drinking to a rule. The sick child has already lost control of his fluid and electrolyte balance, is being rehydrated by an anxious parent, and is on a drug that holds that water in, so waiting for the headache or the weight gain is waiting too long. The sick-day rule is therefore a second instruction of equal rank, not a footnote to the window. In a country where childhood gastroenteritis is common it is the more frequently needed of the two rules, and it is the one families are least often given. The same labelled warning tells the child to avoid swallowing water while swimming. Desmopressin must not be used in a child with primary or psychogenic polydipsia, in whom the compulsion to drink removes the one safeguard the treatment depends on; the polydipsia and any alcohol misuse are excluded before the drug is prescribed, not after. Desmopressin is a safe and useful drug whose single real hazard is entirely avoidable by a rule the family can follow.

Two further points belong to the rule rather than to the laboratory. Routine measurement of weight, serum electrolytes, blood pressure or urine osmolality is not recommended in the child treated with desmopressin for bedwetting, which puts the whole weight of safety on two spoken rules and no blood test: the fluid window on a well night, and the sick-day stop on any night that is not one. And there is one interaction: drugs that induce inappropriate antidiuretic hormone secretion, tricyclic antidepressants expressly among them, demand extra precautions against hyponatraemia when given with desmopressin, including careful attention to the fluid restriction and more frequent monitoring of the serum sodium.

GOOD TO REMEMBER

The instructions that actually prevent harm are not written on the prescription, they are spoken to the parent, and there are two of them. The first is a clock rather than a cup: drinks stop an hour before the tablet and do not resume until the morning. Teaching an allowed volume instead invites the family to give that allowance at two in the morning, deep inside the antidiuretic window. The second is the sick day: no dose on a night the child is vomiting, has diarrhoea or is febrile, and none again until he is well and his fluid balance is normal. Pair every prescription with both, instruct the parents explicitly to keep to them, and withhold the drug entirely from the compulsive drinker.

36.7 Imipramine: the tricyclic, and the order-of-magnitude dose

The imipramine enuresis dose is measured in tens of milligrams total, and this is the single most examined trap in the topic. Imipramine has a long history in this indication, and Indian pharmacology teaching lists enuresis among its uses. The age floor is the first thing to get right, because it is not the diagnostic floor: **the drug is for the child of 6 years and over, and it is contraindicated below 6**, whereas the diagnosis may be made from a chronological or developmental age of 5. The five-year-old who meets criteria is a child you may diagnose and must not give this drug to.

Every child starts at the same place, 25 milligrams at bedtime, whatever the age, and the dose is then set by age and weight band: **25 milligrams a day at 6 to 7 years and 20 to 25 kilograms, 25 to 50 milligrams a day at 8 to 11 years and 25 to 35 kilograms, and 50 to 75 milligrams a day over 11 years and 35 to 54 kilograms**. If a satisfactory response has not come within a week, the dose is stepped up, and it is stepped up slowly, because starting low catches the child who responds low. Two ceilings bound the indication: **75 milligrams a day in total, and 2.5 milligrams per kilogram per day**, whichever is reached first. The labelling is explicit that a daily dose above 75 milligrams does not enhance efficacy and tends only to increase side effects.

The rule is this: the enuresis dose is tens of milligrams as a total daily amount, and it is never converted into, nor borrowed from, a per-kilogram antidepressant figure. Outside nocturnal enuresis, imipramine has no established paediatric safety or efficacy at all; enuresis is the only childhood indication the drug holds. The enuresis dose is small on purpose.

TRAP **Do not read the enuresis dose as milligrams per kilogram.** The classic error writes the imipramine enuresis dose as a per-kilogram quantity, or imports a per-kilogram antidepressant envelope from elsewhere. The enuresis dose is a flat total of tens of milligrams, and the per-kilogram figure that exists in this indication is a cap of 2.5 milligrams per kilogram per day, not a target. A thirty-kilogram nine-year-old dosed to a per-kilogram misreading reaches a three-figure daily dose in a child whose labelled maximum is 75 milligrams.

36.8 Imipramine: cardiotoxicity, overdose and the safeguards

The reason imipramine is held back is a narrow margin between the effective and the fatal. The tricyclic has a **narrow therapeutic index**, and children, the poorly hydrated and those with cardiac disease are more susceptible to its cardiotoxicity than healthy adults. It prolongs the QTc interval and slows cardiac conduction, and in overdose it may be fatal, producing convulsions, cardiac dysrhythmias, severe hypotension, central nervous system depression, coma and characteristic ECG changes. The danger is compounded by the small effective quantity: the tablets a family holds are a plausible instrument of accidental poisoning by a younger sibling, or of a school-aged child's magical belief that swallowing the whole bottle will cure the bedwetting faster. For these reasons the drug must be dispensed and stored under control.

The monitoring follows the cardiac risk. A baseline electrocardiogram before starting is a widely taught practice point rather than a labelled requirement; neither the United Kingdom nor the United States labelling mandates one. What the labelling does record is the reason the ceiling sits where it does: electrocardiographic changes of unknown significance have been reported in

children given twice the maximum of 2.5 milligrams per kilogram per day. There is no per-kilogram threshold above which monitoring is switched on, and any such trigger set above the maximum would only ever fire outside the dose range the drug is allowed to occupy. The cap is the control, not the monitor.

Exposure is capped in time as well as in dose: the maximum treatment period is 3 months, including a gradual withdrawal, and the drug is never stopped abruptly. If the child relapses, a further course is not begun until a full physical examination has been made, and the child who needs repeated courses is reviewed medically every 3 months. The counselling is part of the safety: the family is told that the course lasts 3 months, that the dose is raised gradually, that it is taken at bedtime, that more than two out of three children relapse afterwards, and, in terms, about the particular dangers of overdose and the importance of giving only the prescribed amount and storing the tablets safely. A time-limited course is the control that stops an indefinite exposure to a cardiotoxic drug in a condition that most children grow out of anyway. Imipramine's toxicity is the whole reason it has fallen to the foot of the ladder even though it is an effective drug. It earns its place only in the refractory child in whom the safer options have genuinely been tried and have failed.

36.9 Choosing and combining, and the Indian reality

The three modalities are not interchangeable; each answers a different clinical question.

MODALITY	MECHANISM	DURABILITY OFF TREATMENT	CHIEF HAZARD	PLACE ON THE LADDER
Behavioural measures and the bell-and-pad alarm	Classical conditioning of the full-bladder signal	Highest: the benefit can outlast the treatment	None of consequence; demands weeks of effort	First-line; the most durable single treatment
Desmopressin	Vasopressin V2 analogue reducing nocturnal urine volume	Low: wetting typically resumes on withdrawal	Hyponatraemia and water intoxication; needs the fluid window	The commonest drug; for a rapid or occasion-limited effect, and an initial option when an alarm is unsuitable
Imipramine	Tricyclic; anticholinergic and other actions on the bladder	Low; relapses on withdrawal	Cardiotoxicity, lethal in overdose; dose and duration both capped	Reserved for refractory cases after specialist assessment; no longer first-line

The three treatments compared; the accompanying figure sets out the same escalation from behavioural methods through desmopressin to the reserved tricyclic.

The alarm and desmopressin are not mutually exclusive and are deliberately combined, the drug giving the family early dry nights that sustain their motivation to persist with the conditioning. Where bedwetting has not responded to an initial course of the alarm, the combination of alarm and desmopressin is offered, or desmopressin alone if continuing with the alarm is no longer acceptable to the family. The combination to think twice about is the other one: because tricyclic

antidepressants are among the drugs that induce inappropriate antidiuretic hormone secretion and so demand extra hyponatraemia precautions alongside desmopressin, the second and third rungs of this ladder interact. An anticholinergic is not offered in combination with imipramine.

IN INDIA

The ladder as taught is Western in its ordering, but the Indian reality tilts practice toward the drugs. Enuresis alarms are not widely available or affordable in most settings, so the behavioural first line, though correct, is often not a real option. That is not a licence to skip to the tricyclic. An alarm that cannot be obtained is exactly the case the guidance already anticipates: where an alarm is inappropriate or undesirable, desmopressin is offered as an initial treatment in its own right. Desmopressin as a bedtime oral tablet is therefore the pragmatic pharmacological choice, paired always with the fluid window. Imipramine, by contrast, is inexpensive, familiar and long-established in Indian teaching, where the standard pharmacology texts list enuresis among its uses, and that reachability is precisely why the cardiotoxicity caution matters more here, not less: the safeguards of a controlled supply, a capped dose and a time-limited course are easiest to neglect where the drug is easiest to get.

The doses in this account are taken from United Kingdom and United States labelling, read directly. The Indian regulatory position was not verified: no CDSCO-approved package insert for imipramine or desmopressin was consulted, and no claim is made here about what an Indian insert licenses, at what age, or at what ceiling. Where an Indian insert differs from the figures above, it is the insert that governs the prescription written in India.

The one line to carry: behavioural methods and the alarm are first-line and most durable, desmopressin is the commonest drug and an initial option in its own right when speed matters or an alarm is unsuitable but it relapses on withdrawal, and imipramine is a reserved, cardiotoxic drug for the child of 6 and over whose enuresis dose is a flat tens of milligrams capped at 75 a day, never calculated per kilogram. The per-kilogram figure is a ceiling, not a dose to compute from, and both caps bind and the first one reached is the one that governs.

14 dry nights IN A ROW, OR A 90% DROP IN WET NIGHTS: THE NICE RESPONSE	**200 to 400 mcg** DESMOPRESSIN TABLET, AT BEDTIME	**120 to 240 mcg** DESMOPRESSIN MELT, SUBLINGUAL	**75 mg/day** IMIPRAMINE MAXIMUM, AND NEVER ABOVE 2.5 MG/KG/DAY

37 · Encopresis (types, etiology, management)

The soiling that brings a child to the clinic is almost never the wilful act it is first taken to be, and the whole of this topic turns on that correction. **Encopresis** is the repeated passage of faeces into places where it does not belong, into the clothing or onto the floor, whether the child intends it or not. It is coded in the manual as F98.1, and it is examinable less for the wording of its criteria than for the physiology hidden beneath them: in most affected children the soiling is the visible end of a long chain that begins with one painful stool and ends with liquid faeces leaking helplessly around a hard, retained mass. Read that way, encopresis stops being a

behavioural problem and becomes a disorder of retention with a behavioural surface, and its whole management follows from that reframing.

This account owns the diagnostic criteria of encopresis, its subtypes, its epidemiology, the retention-constipation mechanism that generates the common form, and the staged treatment that flows from that mechanism. The parallel elimination disorder of the bladder, and the drug treatment of that disorder, are taught in their own accounts elsewhere in this chapter and are named here only where the contrast teaches something; the full paediatric assessment framework belongs to the Child psychiatry: assessment, treatment, services and protection notes.

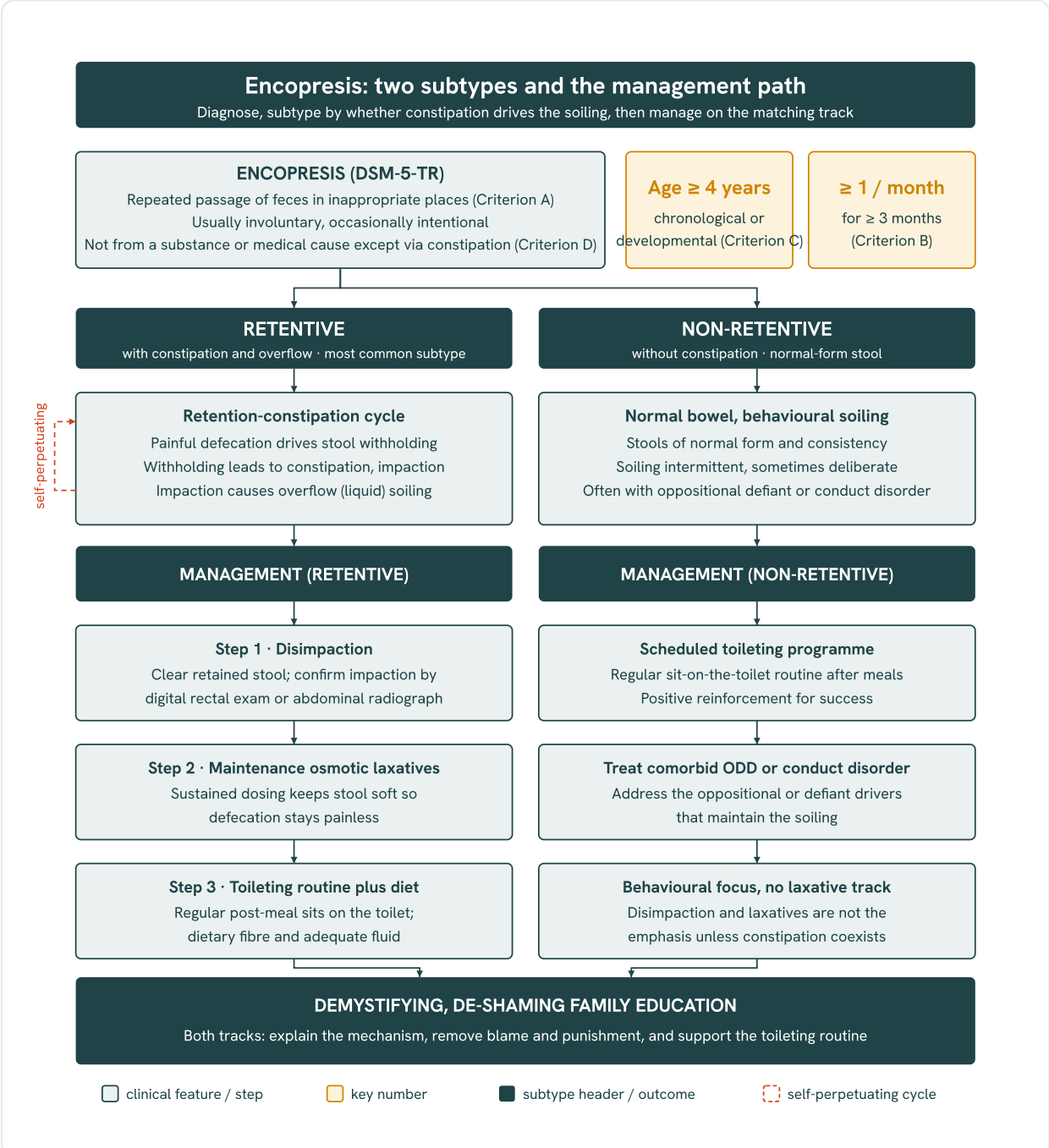

Fig 23.25. Encopresis subtypes and management: a shared DSM-5-TR diagnosis splits into a retentive track (constipation with overflow, managed by disimpaction, maintenance osmotic laxatives, and a toileting routine) and a non-retentive track (normal-form intermittent soiling, managed behaviourally and by treating comorbid oppositional defiant or conduct disorder), converging on demystifying family education.

37.1 Criterion A: the act, and why intent is beside the point

The defining feature is the repeated passage of faeces into inappropriate places, and the criterion is written to be neutral about whether the child meant it. The manual specifies that the passage is most often involuntary and only occasionally intentional, and this ordering is the clinically important part. A tired parent, and sometimes a hurried clinician, reads soiling as defiance, laziness or a bid for attention, because a school-aged child who fouls their clothes surely could stop if they wished. The criterion refuses that reading. In the great majority the child does not feel the stool coming and cannot hold it back, because a retained mass has blunted the normal call to stool and the liquid overflow slips past without warning. Occasionally the passage is deliberate, and that minority carries its own diagnostic weight, discussed with the subtypes below, but the default assumption in front of a soiling child is that the act is involuntary, and getting that starting assumption right is what separates a clinician who treats the bowel from one who punishes the child.

37.2 Criteria B and C: the frequency threshold and the age gate

Two criteria convert a symptom into a disorder: a minimum frequency and duration, and a minimum age. Criterion B sets the count. There must be **at least one such event each month**, sustained for **at least three months**. The threshold is deliberately low, because even monthly soiling in a school-aged child is not a variant of normal, and the three-month floor keeps a single episode of illness-related soiling from being over-labelled. Criterion C is the developmental gate, and it is the one candidates most often state loosely. The child must have reached a chronological age, or an equivalent developmental level, of **at least four years**. The gate is developmental, not merely chronological: continence is a milestone, and a child whose global development sits below the four-year level has not yet reached the stage at which reliable bowel control is normally expected, so the label cannot be applied to them however old they are in calendar years. This matters acutely in a population where intellectual disability and global delay are common, and it is the reason the developmental age, not the birth certificate, decides eligibility.

RULE **Encopresis is a diagnosis of the child who has, developmentally, arrived at continence and then lost or never consolidated it.** The age criterion is a floor set at the point where bowel control is normally achieved, and it is expressed as a developmental level rather than a birthday precisely so that a delayed child is not mislabelled for failing a task their nervous system has not yet reached. Establish the developmental age first; the chronological age alone can never make or exclude the diagnosis.

37.3 Criterion D: the exclusion with a deliberate carve-out

This is the cleverest criterion in the set, and the one most worth a mark. Criterion D states that the soiling must not be attributable to the physiological effect of a substance such as a laxative, or to another medical condition except through a mechanism that involves constipation. The exclusion clause is doing something unusual. Ordinarily a medical cause for a symptom rules a psychiatric label out. Here the manual carves an explicit exception: a medical condition is compatible with the diagnosis when, and only when, it produces the soiling by way of **constipation**. So a child with a low-fibre diet, or hypothyroidism, or coeliac disease, who

becomes constipated and then soils, still has encopresis, because constipation is the accepted final common pathway. By contrast, faecal incontinence arising through a mechanism that is not constipation - the chronic diarrhoea of a gut disease, or the neurogenic bowel of spina bifida, or an anal stenosis that leaks - does not warrant the diagnosis. The clause is not a loophole but the manual recognising that constipation-driven overflow is the disorder's own machinery, and that soiling produced by a different mechanism is a different problem wearing the same outward sign.

37.4 The two subtypes: retentive and non-retentive

The single most useful discrimination in the topic is between the constipated child and the unconstipated one, because they have opposite mechanisms, opposite associations and opposite treatments. The classification recognises exactly two forms, distinguished by whether constipation and overflow are present. The **retentive** form, with constipation and overflow incontinence, is much the commoner; the majority of children older than four who soil have this subtype. Here the stool is retained, the rectum is loaded, and what escapes is liquid overflow. The **non-retentive** form, without constipation and without overflow, is the smaller group: the stools are of normal form and are passed, intermittently, in the wrong place, and this pattern is more often linked with an oppositional or conduct disturbance than with a loaded bowel. The distinction is not academic. A child passing formed stools in inappropriate places without any constipation will not be helped by laxatives, and the clinical attention rightly shifts toward the emotional and behavioural picture, whereas the retentive child needs the bowel cleared before anything behavioural can work.

FEATURE	RETENTIVE (WITH CONSTIPATION AND OVERFLOW)	NON-RETENTIVE (WITHOUT CONSTIPATION)
Relative frequency	The majority of soiling children over four	The smaller group
Stool that soils	Liquid overflow leaking past a retained mass	Stools of normal form, passed intermittently
Constipation and impaction	Present and central	Absent
Typical association	The retention-pain cycle	Oppositional defiant or conduct disturbance
First move in treatment	Clear the impaction, then keep the stool soft	Address the behavioural and emotional picture

The two subtypes of encopresis run on opposite mechanisms; whether the bowel is loaded decides both the associations and the first step of treatment.

37.5 Epidemiology

The numbers are modest, age-dependent and sex-skewed, and each of those three shapes fits the mechanism. The prevalence runs at **one to four per cent** of children in high-income countries, with figures of two to eight per cent reported in some Asian countries. The disorder is far commoner in the younger school-aged child than the older: it is above four per cent between ages four and six and falls to below two per cent between ages ten and twelve, tracking the natural resolution of childhood constipation as the child grows, eats a fuller diet and consolidates

a toileting routine. It is more common in boys than in girls among older children. The age curve is the fact worth carrying: because most cases resolve with growth, persistence into later childhood marks the minority in whom the retention cycle has entrenched and warrants more determined treatment.

37.6 Etiology: the retention-constipation-overflow cycle

Everything about the retentive subtype makes sense once the cycle is drawn. The core mechanism is a self-perpetuating loop of painful defecation and withholding. A painful stool leads the child to withhold, withholding produces constipation, the retained stool becomes impacted, and liquid faeces then overflow around the impaction as incontinence. Once begun, the loop feeds itself: the longer the mass sits, the larger and harder it grows, the more painful its eventual passage, and the stronger the child's learned avoidance of the toilet. This is why **stool withholding** is the pivot of the whole disorder, a rational avoidance of pain that has catastrophic downstream consequences. The constipation that starts the loop may arise on either of two tracks:

- psychological, when the child withholds out of anxiety about defecating, a frightening earlier experience of a painful or hard stool, or oppositional refusal of the toilet;
- physiological, when the stool is hard and difficult to begin with, through ineffectual straining, a paradoxical contraction of the sphincter when the child bears down, a low-fibre and low-fluid diet, or an underlying condition such as hypothyroidism or coeliac disease.

An **anal fissure** closes the loop especially tightly, making the next stool acutely painful and so driving harder withholding still. Whichever track starts it, the destination is the same loaded rectum, and it is the loaded rectum, not the child's will, that produces the soiling.

37.7 Assessment: confirming the impaction

The assessment is short, physical and decisive, because it answers the one question that sorts the subtypes: is the bowel loaded. A careful history establishes the pattern of soiling, the stool form, the presence of pain or withholding behaviour, and the dietary and psychosocial context, and it screens for the red flags that would point away from simple constipation. The physical step that settles the subtype is the demonstration of retained stool: a **digital rectal examination** palpating a loaded rectum, or where that is not feasible or is equivocal, an abdominal radiograph showing faecal loading, confirms the impaction that defines the retentive form. This is not a formality: it converts a vague complaint of soiling into a mechanical diagnosis with an obvious first move, and it lets the clinician show the family that the problem is a full bowel and not a naughty child. The broader psychiatric and developmental workup, where the picture calls for it, follows the framework of the Child psychiatry: assessment, treatment, services and protection notes.

37.8 Management: clear, keep soft, retrain, de-shame

The treatment of the retentive form is a staged programme, and the order of the stages is the examinable core. The sequence is to first clear the retained stool by **disimpaction**, then to hold the bowel empty and the stool soft with **maintenance osmotic laxatives**, and to run alongside

these a **scheduled toileting programme** with dietary fibre, adequate fluid, and family education that demystifies and de-shames the problem. The order is not cosmetic. Disimpaction comes first because maintenance laxatives poured onto an impacted rectum simply add to the overflow; the mass must be gone before anything else can hold. Maintenance is then sustained deliberately over a long period, because the stretched, insensitive rectum needs an extended run of soft, easy, regular stools to recover its tone and sensation, and stopping the moment the soiling clears is the commonest route back to a loaded bowel. The behavioural programme, a regular unhurried sit on the toilet after meals to exploit the gastrocolic reflex, rebuilds the toileting habit the withholding destroyed. The family education carries as much weight as the drugs: a household managing the soiling as misbehaviour has to be brought round to seeing it as a bowel that overflowed, or the punitive dynamic will undo the regimen. This is behavioural-plus-laxative management, and it stands apart from the drug treatment of the bladder disorder taught elsewhere in this chapter, whose agents and doses do not belong here.

MNEMONIC

Clear, Keep, Sit, Teach

Clear the impaction by disimpaction; **Keep** the stool soft with sustained maintenance laxatives; **Sit** the child on a scheduled post-meal toileting routine with fibre and fluid; **Teach** the family that the soiling is overflow from a loaded bowel, not defiance. The order is not interchangeable: keeping, sitting and teaching all fail on a rectum that has not first been cleared.

PITFALL

The relapse trap is stopping the maintenance laxative too early. Soiling often clears within days of disimpaction and a few soft stools, and the natural instinct of family and clinician alike is to stop the medicine now that the child is clean. But the rectum is still stretched and under-sensing, and a bowel allowed to load again reproduces the whole cycle within weeks. Maintenance is continued well beyond symptom resolution and withdrawn slowly, not at the first clean week.

37.9 ICD-11, differentials and the examination traps

The two classifications agree, and the differentials are decided by mechanism rather than by the appearance of the soiling. The ICD-11 essential features mirror the manual, coding the disorder as 6C01 and setting the same approximate age-four developmental threshold. The differentials that earn marks all turn on the carve-out in Criterion D: soiling from chronic diarrhoea, from the neurogenic bowel of spina bifida, or from an anal stenosis is faecal incontinence, not encopresis, because the mechanism is not constipation. Within the diagnosis, the retentive and non-retentive forms are themselves a differential, resolved by whether the bowel is loaded. Two errors recur under examination pressure.

TRAP **Candidates read "except through a mechanism involving constipation" backwards.** The instinct is that any medical cause excludes the diagnosis, so a constipated child with hypothyroidism or coeliac disease gets marked as not having encopresis. The criterion says the opposite: a medical condition is compatible with encopresis precisely when it works through constipation. The exclusion removes only the non-constipation mechanisms, chronic diarrhoea and structural or neurogenic incontinence among them.

GOOD TO REMEMBER

When a school-aged child presents with what the family calls diarrhoea in the pants, think overflow before you think gastroenteritis. Frequent small leaks of foul liquid stool in an otherwise well child, especially with a history of hard or painful stools and toilet avoidance, is the retentive form declaring itself, and the abdomen or the rectal examination will usually find the loaded bowel that explains it. Treating that as an infective diarrhoea, or worse with a constipating agent, deepens the impaction it should be relieving.

Carry into the exam: encopresis is repeated faecal soiling from a developmental age of at least four years, at least monthly for three months, most often the involuntary overflow of a retentive, constipated bowel, managed by disimpaction, then sustained maintenance laxatives, a scheduled toileting routine and family education.

IN INDIA

Because services here classify under ICD, the operative label in case files is the entity coded 6C01, worked from its clinical descriptions. The disorder is genuinely common: prevalence figures in the region of two to eight per cent have been reported in some Asian countries, and low-fibre, low-fluid diets, cramped or shared and unhygienic toilets that a young child dreads, and the frank shame and punishment that soiling attracts in many households all feed the withholding that starts the cycle. Osmotic laxatives for disimpaction and maintenance are widely available and inexpensive, so the mechanical part of treatment is achievable; the harder work is the family education that reframes a shamed, punished child as a child with a loaded bowel, and it is that reframing that decides whether the routine holds.

1-4%	Age 4	≥1/month	3 months
PREVALENCE IN HIGH-INCOME COUNTRIES	MINIMUM CHRONOLOGICAL OR DEVELOPMENTAL AGE	SOILING EVENTS, THE FREQUENCY FLOOR	MINIMUM DURATION

38 · Pica

A child who eats what is not food, and keeps doing it. **Pica** is the persistent eating of **nonnutritive, nonfood substances** continued for **at least one month**. The substances vary with the child's age and with whatever is at hand: paper, soap, cloth, hair, string, chalk, paint, soil, clay, starch, ice, charcoal, pebbles. The defining oddity is that the child is not turning away from food. There is typically no aversion to nourishing food in general; the ingestion of nonfood matter sits alongside an otherwise ordinary appetite, which is exactly what marks pica out from

the disorders in which a child eats too little. Because the behaviour looks trivial to a lay eye and is easy to hide, the diagnosis rests on a clinician thinking to ask, and on separating a genuine disorder from a great deal of developmentally normal or culturally sanctioned mouthing and eating.

The marks here are discriminative and safety-driven. An examiner uses pica to test three things: whether a candidate can apply the developmental and cultural qualifiers that keep the diagnosis from swallowing normal behaviour, whether they know the company pica keeps, and whether they remember that this is a behaviour with hard medical consequences and not merely a curiosity. This topic owns pica in full: its criteria, the age floor, its substances and complications, its comorbidity, its differential and its management. The neighbouring feeding disorders of childhood are named here only where the reasoning needs them and are taught in their own right elsewhere in this chapter.

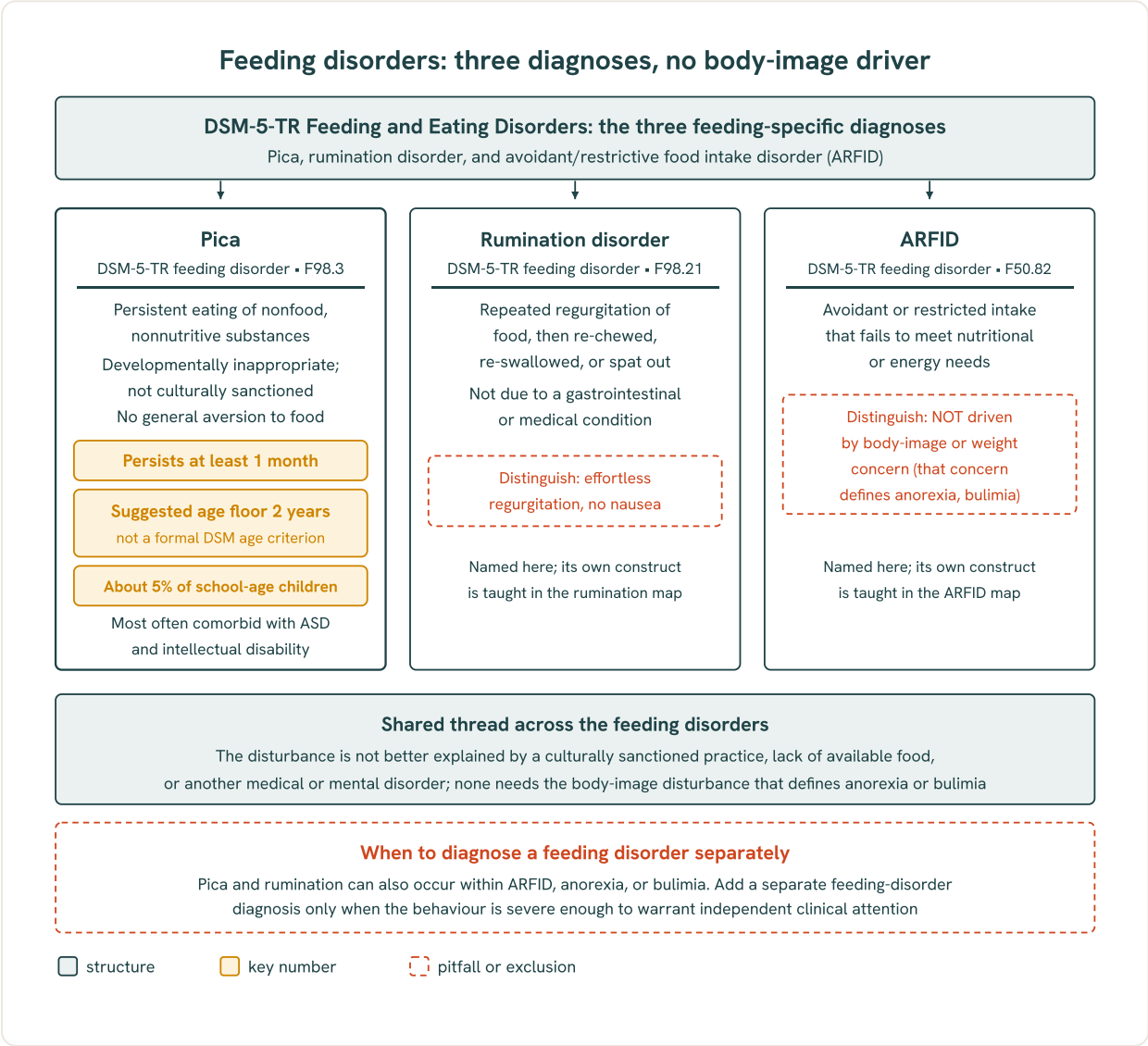

Fig 23.26. Feeding disorders map: pica taught in full, with rumination disorder and ARFID named as the sibling feeding diagnoses that share the absence of a body-image driver.

38.1 The essential feature: nonfood eating, persistent, with appetite intact

The behaviour is an addition to eating, not a subtraction from it. The core requirement is the repeated ingestion of substances that carry no nutritional purpose, sustained rather than a single curious episode, and present for a defined minimum period rather than a passing phase. What a candidate must hold is the qualitative shape of the behaviour. First, it is persistent: an isolated instance of a toddler swallowing a button is not the disorder. Second, the substances are genuinely nonnutritive, so the deliberate eating of low-calorie diet products, which do have some nutritional content, does not qualify. Third, and most usefully in the clinic, the child usually eats food normally as well; pica coexists with an ordinary appetite rather than replacing it. That last point is the fastest way to separate pica at the bedside from a restrictive feeding problem, where the whole difficulty is a narrowed or diminished intake of food itself.

38.2 The qualifiers that keep the diagnosis honest: development, culture and the age floor

Three gates stand between the behaviour and the diagnosis. Not every child who eats nonfood matter has a disorder, and the criteria are built to say so. The eating must be **developmentally inappropriate**, which excludes the universal hand-to-mouth exploration of infancy, when putting objects in the mouth and occasionally swallowing them is a normal stage rather than pathology. It must not be a **culturally supported or socially normative practice**, because in some communities the eating of particular earths or clays is a sanctioned custom and not a sign of illness. And where the eating occurs in the context of another disorder such as autism spectrum disorder, intellectual disability or schizophrenia, a separate diagnosis of pica is made only when the behaviour is severe enough to warrant clinical attention in its own right. To operationalise the developmental gate, a minimum age of **two years** is suggested before the diagnosis is entertained, so that the normal mouthing of the very young is not relabelled as a disorder.

- persistent nonfood eating for the required minimum period, with food eaten normally;
- developmentally inappropriate for the child's age and stage;
- not a culturally sanctioned or socially normative practice;
- if within another disorder, severe enough to deserve independent clinical attention.

RULE **Pica is nonfood eating that is developmentally inappropriate, not culturally sanctioned, and old enough to count.** The behaviour alone is never sufficient. It becomes a diagnosis only once it has cleared the developmental gate, the cultural gate and, where it rides on another disorder, the severity gate. Strip any one of these away and the criteria would capture the exploring infant and the observant clinician alike.

38.3 The substances, and why they are dangerous

What is eaten decides the harm. The preferred substances cluster into recognisable patterns that have acquired their own descriptive names, of which the eating of earth or soil, or **geophagia**, is the classic, alongside the eating of ice and of raw starch. The choice is shaped by age and by availability, which is why the substance changes as the child grows and why environment matters so much. The clinical weight of pica lies not in the strangeness of the

behaviour but in its consequences. Ingested nonfood matter can cause gastrointestinal obstruction, perforation and the formation of concretions in the gut; contaminated soil carries a risk of parasitic infestation; and the swallowing of paint or dust in older housing is a recognised route to lead exposure and its neurotoxicity. These are the reasons an examiner treats pica as a medical problem wearing a behavioural coat, and the reasons the assessment cannot stop at naming the behaviour.

38.4 Comorbidity: the developmental company pica keeps

Where you find pica, look for a developmental disorder. Pica is most commonly seen alongside **autism spectrum disorder** and **intellectual disability**, and the risk tends to rise with the severity of the intellectual impairment. To a lesser degree it is associated with schizophrenia and with obsessive-compulsive disorder. There is a further, characteristic pairing worth carrying into the exam: where a child is pulling out and then eating their own hair, or picking at and ingesting their skin, pica may accompany trichotillomania and excoriation disorder, and the swallowed hair can itself produce a gastric mass. Both autism spectrum disorder and intellectual disability are developed in their own right in the Intellectual disability and autism spectrum disorder notes; here they matter as the settings that should raise suspicion of pica and, conversely, as conditions the clinician must weigh when a child presents eating nonfood substances. The general point is that pica is rarely a lone finding in an otherwise typically developing older child, and its appearance should prompt a look at the wider developmental picture.

38.5 Differential diagnosis

The task is to separate pica from the behaviours it resembles. The accompanying figure maps the feeding and eating disorders of childhood and places pica alongside its neighbours, each of which is separated from it by a single clear feature. The commonest confusions are with normal infant mouthing, with the two other feeding disorders of childhood, and with the hair-and-skin conditions in which ingestion is secondary.

CONDITION TO DISTINGUISH	WHAT SEPARATES IT FROM PICA
Developmentally normal mouthing of infancy	Ingestion follows the ordinary oral exploration of the very young; the suggested age floor of two years and the developmental-appropriateness gate exclude it
Rumination disorder	Repeated regurgitation and re-chewing of already-swallowed food, not the eating of nonfood matter; a disorder of what comes back up rather than what goes down
Avoidant/restrictive food intake disorder	Avoidance or restriction of food leading to inadequate intake, with no ingestion of nonfood substances; the opposite failure of the feeding repertoire, and taught separately in this chapter
Trichotillomania or excoriation with ingestion	The hair-pulling or skin-picking is primary; pica is added only when the eating of the hair or skin is itself a clinical focus
Nonfood eating within another disorder	A separate pica diagnosis is warranted only if the behaviour is severe enough to merit independent attention, not whenever it appears in autism, intellectual disability or schizophrenia

The behaviours pica is most often confused with; each is separated by a single feature rather than by appearance, which is why the qualifiers and the age floor carry the differential.

38.6 Assessment and management

Investigate the body, then modify the behaviour. Because the danger of pica is physical, the assessment is as much medical as psychiatric. A clinician evaluates for the complications the ingested material can cause and for the deficiency states, iron and zinc among them, that are associated with the behaviour, screens for lead exposure where the history or housing suggests it, and looks for gastrointestinal obstruction or a concretion where symptoms point that way. Correcting an identified nutritional deficiency can itself reduce the behaviour, which is why the workup is not optional. Beyond the medical arm, management rests on behavioural strategies and on securing the environment: removing access to the preferred substances, close supervision, and where the child has a developmental disorder, structured behavioural interventions to reduce the ingestion and to build safer alternatives. In many young children the behaviour remits with maturation and with attention to the setting, but the coexisting developmental or nutritional problem often needs its own continuing care. There is no specific medication that treats pica as such; where drugs are used they target a comorbid condition, and any pharmacological choice should be confirmed against a pharmacology source rather than assumed.

Carry this into the exam: persistent eating of nonnutritive, nonfood substances for a month or more, developmentally inappropriate, not culturally sanctioned, in a child of at least two years, most often alongside autism spectrum disorder or intellectual disability.

TRAP **Diagnosing pica in the mouthing infant.** The classic examination error is to label the very young child who explores by mouth and swallows the occasional object as having pica. The suggested minimum age of two years and the requirement that the eating be developmentally inappropriate exist precisely for this child: below that stage the behaviour is a normal part of development, not a disorder.

PITFALL

The commonest clinical failure is to treat pica as a purely behavioural quirk and to skip the medical workup. A child eating soil, paint flakes or hair can arrive with iron-deficiency anaemia, lead exposure, worm infestation, a gut obstruction or a swallowed-hair mass, and none of these is found unless it is looked for. Naming the behaviour and stopping there misses the very harms that make pica worth diagnosing.

GOOD TO REMEMBER

When a child presents with pica, two reflexes change what you do that day: check iron status, because deficiency both drives and is driven by the behaviour and correcting it may resolve it; and take an exposure and access history, because what the child can reach and what the housing contains determine both the medical risk and the first line of management, which is simply removing access.

IN INDIA

Two realities shape pica in Indian practice. Geophagia, the eating of earth or clay, is in some communities a sanctioned custom, at times during pregnancy, and the cultural-exclusion criterion is written for exactly this: a socially normative practice is not a disorder, and the clinician must establish the community norm before reaching for the diagnosis. At the same time, the same soil-eating carries a real burden of iron-deficiency anaemia and of soil-transmitted helminth infestation, so a behaviour that may be culturally ordinary can still demand a medical response. Holding both truths at once, respecting the custom while treating the anaemia and deworming where indicated, is the practical skill this topic tests.

1 month	2 years	ASD, ID
MINIMUM DURATION OF THE BEHAVIOUR	SUGGESTED MINIMUM AGE FOR DIAGNOSIS	COMMONEST DEVELOPMENTAL COMORBIDITIES

39 · Rumination disorder

The child is not sick, and the food comes back up anyway. **Rumination disorder** is the repeated, effortless bringing back up of food that has already been swallowed, so that it can be re-chewed and then re-swallowed or spat out. The defining quality is that it happens without the child appearing ill: there is no nausea beforehand, no retching, no visible disgust, and often no obvious distress at all. Partially digested food simply reappears in the mouth minutes after a meal, is worked over again, and is either swallowed back down or ejected. To an observer who does not know the condition it can look like an odd habit, a digestive complaint, or a mild illness; to the clinician who does, that very absence of the machinery of ordinary vomiting is the diagnosis.

The marks here are about discrimination and about not medicalising the wrong thing. An examiner uses this topic to test whether a candidate can separate an effortless, behaviourally maintained regurgitation from true vomiting, from acid reflux, and from the several structural and neurological conditions that also send food back up. This topic owns rumination disorder in full: its core criterion, its phenomenology, its frequency and duration requirements, the medical and psychiatric conditions it must be told apart from, and its reach across the lifespan. The neighbouring feeding disorders are developed in their own right elsewhere in this chapter and are named here only where the reasoning needs the contrast.

39.1 The essential feature: effortless regurgitation and re-chewing

The whole diagnosis is a repeated, effortless return of already-swallowed food. The core requirement is a repeated **regurgitation** of food, in which previously swallowed, partially digested material is brought back up into the mouth and is then re-chewed, re-swallowed, or spat out. The word regurgitation is doing precise work: this is not the forceful expulsion of vomiting driven by a reflex arc, but a gentle, almost passive reappearance of a bolus that the child then processes again. Because the child produced the swallow in the first place and can carry the food back down afterwards, the act sits closer to a self-generated behaviour than to a symptom happening to the child, and it is this behavioural quality, more than any single feature, that the rest of the topic hangs on.

RULE **Effortless regurgitation without nausea, retching or disgust is rumination; forceful, distressed expulsion is not.** The presence of premonitory nausea, retching, autonomic distress or evident disgust points away from rumination and towards true vomiting or an organic cause. Rumination is quiet, repetitive and post-prandial, and the child frequently seems untroubled by it, which is exactly why it is missed.

39.2 The phenomenology that separates it from vomiting

Read the seconds after the meal, not the vomit bowl. The regurgitated material is characteristically food that was swallowed only minutes earlier and is still recognisable and only partially digested, brought up without the sour, fully acidified quality of gastric vomit and without the preceding wave of nausea. The episode typically begins soon after eating and can recur through the post-prandial period. Many affected children can be observed to initiate the return of food, for instance by particular movements of the tongue, jaw or abdominal wall or by posturing, and the whole sequence tends to be repetitive and stereotyped across meals. None of this resembles the single, effortful, distressing event of true vomiting. The clinical skill the examiner is probing is the willingness to watch a meal and the period straight after it, rather than to reach for an antiemetic or an endoscopy on the assumption that returning food must be a gut disease.

- the material is recently swallowed and only partially digested, not fully acidified gastric content;
- there is no premonitory nausea and no retching;
- the episodes are post-prandial, repetitive and often self-initiated;

- the child commonly appears indifferent rather than distressed.

39.3 How often and for how long: the frequency and duration thresholds

Occasional posseting is not the disorder; a sustained, frequent pattern is. For the diagnosis the regurgitation must have been present for **at least one month**, and it should be a frequent behaviour rather than a rare event, occurring on the order of **several times a week** and often daily. These thresholds do two things. The duration floor screens out the transient regurgitation that many infants show and grow out of, reserving the label for a pattern that has proven itself durable. The frequency expectation stops a very occasional episode from being over-read as a disorder. Together they mark rumination as an established, repeating behaviour, which matters because it is the persistence and the repetition, not the mere fact of food returning once, that carry the nutritional and developmental risk.

39.4 Excluding the medical mimics

Rumination is a diagnosis of a behaviour, made only once organic causes are excluded. The regurgitation must not be attributable to an associated gastrointestinal or other medical condition, and this exclusion is the heart of the differential. The obvious mimic is **gastroesophageal reflux**, in which acidic stomach content passes back up the oesophagus; the returning material there is sour and refluxed rather than re-chewed, and the mechanism is a failure of the lower oesophageal sphincter, not a learned behaviour. Structural obstruction such as **pyloric stenosis** produces true, often forceful vomiting from a mechanical block to gastric emptying, and delayed emptying from **gastroparesis** or an anatomical hiatal hernia can each mimic the picture. A particular neurological trap is **Sandifer syndrome**, in which reflux drives paroxysmal dystonic posturing of the head and trunk that can be mistaken for a movement disorder or for the posturing of rumination itself. Only when these have been considered and do not account for the picture is the behavioural diagnosis made.

CONDITION	WHAT SEPARATES IT FROM RUMINATION
Gastroesophageal reflux	Sour, acidified content refluxed passively through an incompetent sphincter; not re-chewed and re-swallowed, and typically with acid symptoms
Pyloric stenosis	Mechanical gastric outlet obstruction producing true, often projectile vomiting rather than effortless return of chewable food
Gastroparesis / hiatal hernia	Delayed emptying or an anatomical defect causing regurgitation or vomiting on a structural, not behavioural, basis
Sandifer syndrome	Reflux-associated dystonic posturing of the neck and trunk, a neurological mimic that must not be read as the disorder

The organic conditions rumination must be told apart from; each is separated by its mechanism rather than by the mere appearance of returning food, which is why the medical exclusion is done before the behavioural label is applied.

TRAP **Calling refluxed acid rumination, or calling rumination reflux.** The classic examination error is to collapse the two. Reflux brings up sour gastric content through a passive mechanism and is not re-chewed; rumination brings up recently swallowed, still-chewable food that the child works over again, with no nausea and no acid burn. Mistaking one for the other leads either to fruitless anti-reflux treatment or to a missed behavioural diagnosis.

39.5 The eating-disorder exclusion and the additional-attention rule

Two further exclusions keep rumination a distinct entity. The behaviour must not occur exclusively during the course of an eating disorder. If the regurgitation happens only within anorexia nervosa, bulimia nervosa, binge-eating disorder or **avoidant restrictive food intake disorder**, then that condition, not rumination, is the diagnosis; those disorders are taught in their own right, with ARFID developed elsewhere in this chapter and the classical eating disorders in the Eating and sleep-wake disorders notes. The final rule addresses the common situation in which rumination arises alongside another mental disorder, most often an intellectual or neurodevelopmental one: here the regurgitation is diagnosed as an additional condition only when it is severe enough to warrant independent clinical attention in its own right, rather than being folded into the background disorder. This prevents both the over-diagnosis of a minor behaviour and the neglect of a genuinely dangerous one.

39.6 Across the lifespan, and the association with intellectual disability

It is not only an infant's problem. Although rumination is classically described in infancy, it can be diagnosed across the lifespan, in older children, adolescents and adults, and it is seen with particular frequency in people with **intellectual disability**. In infants the behaviour is often understood as self-stimulating and self-soothing, emerging where the ordinary regulation and stimulation provided through feeding interactions is lacking; in individuals with significant intellectual disability it can become an entrenched, self-reinforcing habit. The practical importance of remembering the lifespan reach is that a non-verbal adolescent or adult with intellectual disability who repeatedly brings food back up is easily assumed to have a gastrointestinal problem, and the behavioural nature of the regurgitation is overlooked precisely in the group most likely to have it.

PITFALL

The commonest clinical failure is to subject a ruminating child to an escalating series of invasive gastrointestinal investigations and empirical anti-reflux treatments while never watching a meal. Because the returning food looks like a gut symptom, the workup runs down the organic track for months, when a period of direct observation after eating would have shown the effortless, self-initiated, re-chewed regurgitation and made the diagnosis at the bedside.

IN INDIA

Two realities shape recognition here. Rumination is most often encountered in children and adults with intellectual disability, many of whom are in family or institutional care with limited access to specialist feeding assessment, so the behaviour is frequently attributed to physical illness and worked up repeatedly before anyone names it. And because most services diagnose under ICD, the clinician should hold the same behavioural picture in mind under that framework and resist the pull towards a purely gastroenterological formulation, particularly in a non-verbal patient who cannot report the absence of nausea themselves.

39.7 Why it matters and the principles of management

Left unrecognised it costs weight, nutrition and, sometimes, life. The repeated loss of ingested food can produce weight loss, failure to thrive or malnutrition, dental erosion from the returned material, halitosis and social rejection, and in the most severe cases the nutritional compromise is dangerous. Because the disorder is behavioural, management is directed at the behaviour and its context rather than at the gut. The mainstay in infants and in those with intellectual disability is a behavioural and interactional approach: enriching the feeding relationship and the child's stimulation, and, where a specific technique is used, teaching an action incompatible with regurgitation such as diaphragmatic breathing after meals. The details of specific behavioural protocols and of any pharmacological adjunct lie outside what this topic establishes and should be confirmed against a dedicated source. What the candidate must carry is the logic: identify the effortless, re-chewed, post-prandial regurgitation, exclude the organic mimics, and treat it as a behaviour, not as a disease of the stomach.

GOOD TO REMEMBER

The single most useful investigation is often free: observe the patient during and for the half hour after a meal. Watching the recently swallowed food return effortlessly, be re-chewed and re-swallowed, with no nausea and no retching, settles the diagnosis more cheaply and more certainly than any imaging, and it also reframes the family's understanding of the problem from an illness to be cured to a behaviour to be reshaped.

Carry this into the exam: effortless, post-prandial regurgitation of recently swallowed food that is re-chewed, without nausea or retching, for at least a month and not explained by reflux or another medical condition, is rumination disorder.

1 month	several/week	daily
MINIMUM DURATION OF THE REGURGITATION	MINIMUM FREQUENCY EXPECTED	TYPICAL FREQUENCY AT PRESENTATION

40 · Avoidant restrictive food intake disorder (ARFID)

A child can starve without ever having a thought about weight or shape, and that is the whole point of this diagnosis. **Avoidant/restrictive food intake disorder** describes a persistent disturbance of eating or feeding in which the intake of food, in amount or in variety or in both, is

so restricted that the body or the life suffers for it, yet the restriction is driven by something other than a wish to be thin. The child eats too little, or too narrow a range, because food does not interest them, or because its look, smell or texture repels them, or because they are frightened of what eating might do to them. What is absent is any concern about becoming fat and any distortion in how their weight or shape is experienced. Holding those two facts together, real physical or functional harm on one side and a complete absence of body-image pathology on the other, is the entire topic.

The marks here are almost entirely discriminative. An examiner is testing whether a candidate can recognise a restrictive eating disorder that is not an eating disorder in the classical, weight-and-shape sense, and can therefore avoid the reflex of calling every underweight child who refuses food an anorexia case. This topic owns the criteria of the disorder and, above all, the boundary that separates it from the weight-driven eating disorders; anorexia nervosa and bulimia nervosa themselves belong to the Eating and sleep-wake disorders notes and are only named and cross-referred here, never taught.

40.1 Criterion A: a feeding disturbance that must cause real harm

The eating disturbance is only a disorder when it produces a consequence. The diagnosis is not made on fussy eating alone. There must be an eating or feeding disturbance that fails to meet the child's nutritional or energy needs, and that failure must show itself through at least one of **four** possible consequences. The restriction may cause significant weight loss, or in a growing child a failure to gain expected weight and **faltering growth**; it may produce a significant nutritional deficiency; it may force a dependence on enteral tube feeding or on oral nutritional supplements to keep the child fed; or it may cause a marked interference with psychosocial functioning, so that mealtimes, school trips, family meals and social life are disrupted by the eating problem. Any one of these clears the bar. The last of them matters more than candidates expect, because it means a child of entirely normal weight can still qualify: the harm can be to the life rather than to the body mass.

- significant weight loss, or faltering growth and failure to gain expected weight in a child;
- a significant nutritional deficiency;
- dependence on enteral feeding or oral nutritional supplements to meet needs;
- marked interference with psychosocial functioning.

PITFALL

Waiting for the child to be underweight before considering the diagnosis. Because one of the qualifying consequences is functional and social rather than nutritional, a normal-weight child whose extreme selectivity has made every meal a battle and every outing impossible can meet Criterion A in full. Anchoring on the scale alone will make the clinician miss exactly these children, who are often the ones causing the most family distress.

40.2 The three drivers of the restriction

Why the child is not eating tells you which ARFID you are looking at. The restriction is understood through **three** characteristic drivers, and a given child may sit on one or on more than one of them. The first is an apparent **lack of interest** in eating or in food, a low drive to eat in which the child simply does not seem hungry, forgets to eat, or tires of a meal after a few mouthfuls. The second is avoidance based on the sensory characteristics of food, the intense, selective refusal of foods by texture, smell, colour, temperature or appearance that produces the classic child who will eat only a handful of beige, dry, predictable items. The third is a concern about the aversive consequences of eating, a conditioned fear that follows a frightening experience such as choking, a traumatic gastrointestinal procedure or an episode of vomiting, and that then generalises into avoidance of the foods or the act of eating that the child associates with it, sometimes called functional dysphagia.

DRIVER	WHAT THE CHILD SHOWS	WHAT IT IS NOT
Apparent lack of interest in eating or food	Low appetite drive, early satiety, forgetting to eat, no pleasure in food	Not a wish to lose weight; the child is indifferent to food, not restricting it deliberately
Sensory-based avoidance	Refusal by texture, smell, colour or appearance; a narrow, fixed repertoire of accepted foods	Not calorie avoidance; the same food would be eaten if its sensory qualities were tolerable
Concern about aversive consequences	Fear of choking, vomiting or pain after a frightening event; avoidance of feared foods or of eating itself	Not fear of weight gain; the dread is of a bodily catastrophe while eating, not of fatness

The three drivers of avoidant and restrictive intake. Each explains the restriction by a mechanism that has nothing to do with weight or shape, which is what keeps the whole disorder distinct from the weight-driven eating disorders.

GOOD TO REMEMBER

The sensory-avoidant presentation overlaps heavily with autism spectrum presentations, where narrow, texture-bound eating is common. When you meet the child with an extremely restricted food repertoire, screen deliberately for the wider neurodevelopmental picture, and remember that the eating problem still needs its own nutritional and feeding-focused management rather than being written off as just part of the autism.

40.3 Criterion C: the absence of body-image disturbance is the whole diagnosis

ARFID is the restrictive eating disorder without the weight-and-shape motive. The single most important criterion is the one that draws the boundary with the classical eating disorders. The eating disturbance must not occur exclusively during the course of anorexia nervosa or bulimia nervosa, and there must be **no disturbance in the way the person's body weight or shape is experienced**. This is the crux. In anorexia and bulimia the restriction is powered by a fear of fatness and a distorted perception of the body; in ARFID it is powered by lack of interest, sensory aversion or fear of an aversive consequence, and the person has no drive for thinness and no distorted body image at all. Where genuine weight or shape concern is present and driving the restriction, the diagnosis is not ARFID. Anorexia nervosa and bulimia nervosa are developed in

their own right in **the Eating and sleep-wake disorders notes**; here they are the comparators against which ARFID is defined.

RULE **No weight or shape concern, no distorted body image: that absence is what makes it ARFID.** The restriction in ARFID is driven by disinterest, sensory aversion or fear of aversive consequences, never by a wish to be thin. The moment a distorted experience of body weight or shape is driving the eating, the clinician has left ARFID and is in the territory of anorexia or bulimia.

TRAP **Labelling every underweight, food-refusing child as anorexia nervosa.** The classic error is to reason from low weight and restricted intake straight to anorexia, without asking why the child is restricting. If there is no fear of weight gain and no body-image distortion, and the avoidance is sensory or a conditioned fear of choking or vomiting, the diagnosis is ARFID, and treating it as anorexia will target a motive the child does not have.

40.4 Criteria B and D, and where ARFID came from

Two exclusions keep the diagnosis honest. Criterion B requires that the disturbance is **not better explained by a lack of available food** or by a culturally sanctioned practice, so that a child who is undernourished because the family cannot afford food, or who eats little during a religious fast, is not captured. Criterion D requires that the eating disturbance is not attributable to a concurrent medical condition and is not better explained by another mental disorder; where an eating problem is fully accounted for by a physical illness or by another psychiatric condition, that condition is the diagnosis. ARFID is a positive entity, not a residual bin, and these two exclusions are what stop it swallowing every cause of low intake in childhood. Historically, the category was created to replace and extend the older DSM-IV feeding disorder of infancy or early childhood, carrying that clinical reality forward and widening it to older children, adolescents and adults, who were previously left without a home for a restrictive eating problem that was not anorexia.

40.5 Assessment and the company ARFID keeps

The assessment is built to answer one question before all others. Because the diagnosis turns on the motive for restriction and on the presence of harm, the assessment is directed at establishing both. The clinician documents the pattern and the range of foods eaten and refused, elicits which of the three drivers is operating, and actively probes for any weight or shape concern, since its presence or absence decides the diagnosis. In parallel the clinician establishes harm: the growth trajectory and any faltering, nutritional adequacy and specific deficiencies, any reliance on supplements or tube feeding, and the degree of psychosocial and family disruption. The differential is wide and must be worked through with care, because low intake in a child can arise from a medical cause of poor appetite or dysphagia, from a depressive disorder, from an anxiety or obsessive presentation, and, in the sensory-avoidant child, from an autism spectrum disorder whose restricted eating is part of a broader pattern. ARFID is diagnosed when the eating disturbance stands as a problem in its own right rather than being fully explained by one of these.

Carry this into the exam: restricted intake plus real harm, driven by disinterest, sensory aversion or fear of an aversive consequence, and with no fear of fatness and no distorted body image, is ARFID, not anorexia.

40.6 Principles of management

Feed the child, treat the driver, and do not import an eating-disorder model that does not fit. Management follows from the mechanism rather than from a single protocol. The first duty is the physical one: correcting undernutrition, restoring growth, replacing specific deficiencies and, where the child depends on tube feeding or supplements, working deliberately towards oral intake. Beyond that, the psychological work is matched to the driver. Sensory-based avoidance is approached with graded, patient exposure to new textures and foods and a widening of the repertoire, usually with close involvement of the family and often a feeding or occupational-therapy input. The conditioned fear of an aversive consequence is treated on an anxiety model, with graded exposure to the feared foods and to the act of eating, undoing the association that a choking or vomiting episode laid down. The child with a low drive to eat needs structure, appetite-supporting routines and attention to any contributing medical cause. Crucially, because there is no body-image pathology to address, the cognitive work aimed at weight and shape concern that anchors anorexia and bulimia treatment has no target here and should not be transplanted across; specific pharmacological agents and dosing lie outside what this topic establishes and should be confirmed against a pharmacology source.

IN INDIA

ARFID is under-recognised in Indian practice, partly because extreme faddiness is culturally read as ordinary childhood behaviour and met with coaxing or force-feeding rather than assessment. Two discriminations become especially live here. First, Criterion B does real work: in a setting where undernutrition from food insecurity is common, the clinician must establish that food was available and the child restricted it, not that the family could not provide it. Second, the very common paediatric complaint that "my child does not eat" hides a small number of genuine ARFID cases among a large majority of normal variation, so the useful clinical move is to ask specifically about harm, growth faltering, nutritional deficiency and the collapse of family and social life around meals, which is what separates the disorder from the ordinary fussy eater.

3	4	No
DRIVER PRESENTATIONS: DISINTEREST, SENSORY AVERSION, FEAR OF CONSEQUENCE	POSSIBLE CRITERION A CONSEQUENCES, ANY ONE SUFFICIENT	BODY-IMAGE DISTURBANCE, THE FEATURE SEPARATING ARFID FROM ANOREXIA AND BULIMIA

41 · Breath-holding spells and other transient childhood behavioural phenomena

A frightening event with a benign heart. Few things alarm a parent more than watching a toddler cry, stop breathing, change colour and go limp, and few things reassure a clinician more once the pattern is recognised for what it is. **Breath-holding spells** are involuntary, reflexive episodes in which a young child, provoked by a sudden emotional or physical stimulus, stops breathing in expiration long enough to lose colour, tone and consciousness, and then recovers

completely and spontaneously. The whole event is dramatic, stereotyped and self-terminating, and the single most important thing a candidate can say about it is that it is neither an act of will nor a form of epilepsy. The marks in this topic are earned by holding that reframe steady against the two intuitions that mislead everyone in the room, the parent who is sure the child is doing it deliberately, and the clinician who reaches for an anticonvulsant.

The topic also carries the broader heading of the transient behavioural phenomena of early childhood, the ordinary, self-limiting habits and reflexes that occupy the wide territory between normal development and disorder. Breath-holding is the one that reaches the examination as a discrete named entity, so it is taught here in full; the others are named and placed, because their value is precisely that they do not need treating.

41.1 A reflex, not a willed act

The child does not decide to hold their breath. The name is a misnomer that has cost countless toddlers a spanking and countless parents a needless guilt. What looks like a defiant, deliberate withholding of breath is in fact an involuntary reflex arc triggered by a provoking stimulus, running to completion outside the child's control and ending on its own. This distinction is not pedantic. If the behaviour were voluntary, the correct response would be to refuse to reward it; because it is reflexive and benign, the correct response is calm handling and the removal of the fear that surrounds it. The spell always follows a provocation and never occurs in sleep, and the child has no memory of the loss of consciousness itself. Recovery is complete within a minute, with no drowsiness, no confusion and no lasting deficit, which is exactly the profile a candidate must be able to recite to separate this from the seizure it imitates.

RULE **A breath-holding spell is an involuntary, benign, self-limiting reflex, not wilful defiance and not epilepsy.** Every management decision follows from this one sentence. There is no neurological damage from the spells themselves, the child cannot be disciplined out of them, and anticonvulsants have no place. The clinician's work is to recognise the pattern, exclude the small number of genuine mimics, and hand the family confident reassurance in place of dread.

41.2 Epidemiology and natural history

Common, early and outgrown. Breath-holding spells affect between **0.1% and 4.6%** of otherwise healthy young children, so they are common enough that a paediatric or child-psychiatry clinic will see them regularly, and they cluster in a narrow developmental window. Onset is usually between **6 and 18 months** of age, the period in which the autonomic and brainstem systems that drive the reflex are still maturing, and the great majority of children have stopped having spells by **5 years** of age. That trajectory, appearing in infancy and disappearing spontaneously by school age, is itself a reassuring feature and part of the natural history a candidate is expected to know. The examiner is not testing whether the child will grow out of it, because the answer is almost always yes; the examiner is testing whether the clinician can promise that outcome without first excluding the conditions that masquerade as it.

GOOD TO REMEMBER

The natural history is the therapy. Once the diagnosis is secure, the most powerful intervention available is telling the family, plainly and confidently, that the spells are harmless, that the child is not brain-damaged and is not being manipulative, and that the episodes will fade on their own within the first few years of life. Anxious, over-protective handling and repeated capitulation to the child prolong the pattern; matter-of-fact calm shortens it.

41.3 The two types: cyanotic and pallid

Two triggers, two colours, one benign end. Breath-holding spells come in two forms distinguished by their provocation and the colour the child turns. The **cyanotic type** is the more common. It is usually precipitated by anger or frustration: the child emits a short, loud cry, then involuntarily holds the breath in forced expiration, becomes cyanosed, goes rigid or limp with a transient loss of consciousness, and then takes a long inspiration and recovers. The **pallid type** is more often precipitated by pain or fear, such as a minor bump to the head or a sudden fright; here the crying may be minimal or silent, the apnoeic period is briefer, and the child develops pallor rather than cyanosis before losing consciousness. In both types the entire episode lasts approximately **10 to 60 seconds** and ends in full recovery.

FEATURE	CYANOTIC SPELL	PALLID SPELL
Relative frequency	The commoner of the two	The less common
Usual provocation	Anger or frustration	Pain or fear (a bump, a fright)
The cry	A short, loud cry precedes the apnoea	Crying may be minimal or silent
Colour change	Cyanosis, from breath held in forced expiration	Pallor, before loss of consciousness
Proposed mechanism	Respiratory, with autonomic dysregulation	Vagally-mediated cardiac inhibition, with reflex asystole or bradycardia
Course of the episode	Rigidity or limpness, brief loss of consciousness, then a long inspiration and full recovery	A briefer apnoeic phase, otherwise proceeds and recovers as the cyanotic spell

The two forms share a benign, self-terminating course; they differ in the stimulus that sets them off, the colour the child turns, and the physiology that drives them.

MNEMONIC

Blue with rage, pale with fright

The **cyanotic** child turns blue and is set off by anger or frustration; the **pallid** child turns pale and is set off by pain or fear. The colour names the type, and the colour points at the mechanism: cyanosis follows the held breath, pallor follows the vagal drop in heart rate.

41.4 Etiopathogenesis: why the reflex fires

The cause is multifactorial, and one strand of it is treatable. No single lesion explains breath-holding spells; the current understanding is that several immature or dysregulated systems converge to produce the reflex. **Autonomic nervous system dysregulation** underlies both types, with the pallid form in particular reflecting **vagally-mediated cardiac inhibition**, a reflex asystole or profound bradycardia mediated by an overactive vagal response to the provoking stimulus. Contributing to this immaturity is **delayed brainstem myelination**, consistent with the age window in which the spells appear and then remit as the nervous system matures. The strand that changes management is **iron deficiency anaemia**, which is associated with breath-holding and whose correction can reduce the frequency of spells, giving the clinician something to treat beyond reassurance. The mechanisms sit together rather than competing:

- autonomic dysregulation, the shared final common pathway of both types;
- vagally-mediated cardiac inhibition, the reflex asystole that defines the pallid spell;
- delayed brainstem myelination, fitting the infantile onset and the spontaneous resolution;
- iron deficiency anaemia, the one contributor that is worth actively seeking and correcting.

IN INDIA

Iron deficiency anaemia is highly prevalent among Indian infants and young children, which makes the iron strand of the etiology far more than a footnote in this setting. A child presenting with breath-holding spells warrants an assessment of iron status, and correcting a documented deficiency is a rational, low-risk intervention that can reduce the frequency of episodes. In a population where nutritional anaemia is common, this converts an otherwise "reassure and wait" condition into one with a concrete, treatable contributor worth pursuing.

41.5 Telling it apart from epilepsy, and managing it

The spell that imitates a seizure is not one. The central differential, and the one the examination targets, is the epileptic seizure. Breath-holding is a **nonepileptic** seizure-like behaviour: it is invariably preceded by a provoking stimulus and a cry or a fright, it never arises out of sleep, the loss of consciousness is brief and colour-linked, and recovery is immediate and without a post-ictal state. A genuine seizure has none of this dependence on provocation, can occur in sleep, and is typically followed by drowsiness or confusion. A prolonged pallid spell may end in a few anoxic jerks, and this is exactly where the unwary reach for an anticonvulsant, but the jerks are the consequence of transient cerebral hypoxia from the vagal asystole, not the expression of an epileptic focus. Management follows from the benign, self-limiting nature established above: confident reassurance and explanation are the cornerstones, the family is taught that the spells cause no harm and will remit, and any underlying contributor, iron deficiency in particular, is treated. Drugs directed at the brain are not indicated for the spells themselves.

TRAP **Diagnosing epilepsy and starting an anticonvulsant.** A pallid spell with a few terminal jerks, described second-hand by a frightened parent, is the classic set-up. Candidates who anchor on the convulsive movements and miss the provoking fright, the pallor and the instant recovery will label a seizure and prescribe accordingly, exposing the child to an unnecessary drug for a benign reflex. The discriminating features are the invariable trigger, the absence of sleep-onset events, and the immediate, post-ictal-free recovery.

PITFALL

The commonest clinician-and-family error is not misdiagnosis but management by appeasement. Once parents learn that a tantrum can escalate into a frightening spell, they begin to give the child whatever prevents the frustration, and the surrounding behaviour, though not the reflex itself, is powerfully reinforced. Advising a family to yield to the child in order to avert spells trades a benign, self-limiting reflex for an entrenched behavioural pattern. The guidance is the opposite: handle the trigger calmly and consistently, and do not let the fear of a spell dictate the parenting.

41.6 The wider family of transient childhood behavioural phenomena

Most of childhood's odd behaviours are developmental, not pathological. Breath-holding belongs to a larger group of transient phenomena of early childhood that share a benign, self-limiting character: the ordinary temper tantrum, thumb-sucking, nail-biting, rhythmic habits such as body-rocking and head-banging at sleep onset, and the brief motor mannerisms of the toddler years. The unifying clinical principle is that each is common, developmentally expected, and outgrown, and that the intervention of first resort is explanation and reassurance rather than treatment. The examiner's interest here is judgement: knowing when a behaviour sits within normal limits and when its intensity, persistence or associated impairment lifts it into a disorder that is taught in its own right. The threshold at which repetitive or rhythmic behaviour ceases to be a benign habit and becomes a stereotypy or a tic, and the boundaries of the individual childhood disorders, are drawn where those topics are taught; the discipline in this section is to recognise the transient phenomenon, place it, and resist the urge to medicalise it. The general developmental context for what is age-appropriate belongs to the Normal child development and milestones notes.

Breath-holding spells are involuntary, benign, self-limiting reflexes of early childhood, not wilful acts and not epilepsy; recognise the trigger-bound pattern, exclude the seizure, treat any iron deficiency, and reassure.

0.1-4.6%	6-18 mo	by 5 y	10-60 s
OF OTHERWISE HEALTHY YOUNG CHILDREN AFFECTED	USUAL AGE OF ONSET	USUAL SPONTANEOUS RESOLUTION	DURATION OF A TYPICAL EPISODE

Discriminate, apply and consolidate

42 · Telling the disorders apart, and from normal limits

The marks in this section are not for knowing a disorder but for telling it from its neighbours. By the time a candidate reaches this point the individual entities have each been learned in their own place, and the temptation is to treat that as the finish. It is not, because the examined presentation is almost never a clean single disorder announcing its name. It is a disruptive, irritable, rule-breaking child, or a frightened, clingy, wet one, described in a paragraph that fits three or four diagnoses at once and one normal childhood at the same time. The work that earns the marks is the sorting: seeing which axis a presentation turns on, which nearest neighbour it must be held against, and whether it is a disorder at all. This section does that work once, across the whole chapter, using only what the preceding entities have already established.

Two discriminations carry most of the weight, and the chapter has been built to set them up. The first is internal to the behavioural group: the several ways a child can present as disruptive look alike from across the room and separate only on the quality of what is happening underneath. The second runs the length of the chapter: nearly every symptom here also exists as an ordinary part of growing up, so each disorder has to be told not only from its diagnostic neighbours but from its own normal counterpart. Both are done here as maps rather than as fresh teaching, because the point of a synthesis is to lay finished pieces side by side.

CROSS-CUTTING DISCRIMINATION OF DISRUPTIVE BEHAVIOUR

Five patterns a difficult or disruptive child can show. This chapter names conduct disorder, ODD, DMDD and ADHD for contrast; read down each axis.

FEATURE	CONDUCT DISORDER	OPPOSITIONAL DEFIANT (ODD)	DMDD disruptive mood dysreg.	ADHD-DRIVEN DISRUPTIVENESS	NORMAL LIMITS (reference anchor)
CORE DISTURBANCE	Repetitive pattern that violates others' rights and major social norms	Angry, irritable mood plus argumentative, defiant, vindictive behaviour	Chronic irritability with recurrent severe temper outbursts	Disruptiveness secondary to inattention and impulsivity, not choice	Brief, developmentally expected defiance or high spirits
AGGRESSION, RIGHTS OF OTHERS	**YES - overt aggression,** property damage, deceit or theft present	**NO serious aggression to** people or animals, no property damage or theft	Outbursts occur, but the pattern is mood-driven, not rule-breaking	Impulsive, not planned; driven by poor response inhibition	**No persistent aggression;** incidents are minor and resolve
MOOD / IRRITABILITY	Not intrinsic to the disorder definition	Irritable, angry mood is intrinsic to it	**Persistently irritable between outbursts**	Frustration possible; not the core feature	Age-appropriate frustration only
BOUNDARY WITH NORMAL	Serious, persistent rule violation well beyond normal mischief	Defiance exceeds age norms and impairs across settings	Outbursts far exceed normal tantrums in force and frequency	Impairment from the deficit, not wilful opposition	**No cross-setting impairment - this is not a disorder**

READ THE DRIVER, NOT THE SURFACE BEHAVIOUR
A rule-violation pattern points to conduct disorder. Oppositional defiance points to ODD. A persistent irritable mood floor points to DMDD. A response-inhibition deficit points to ADHD. No cross-setting impairment is normal.

PITFALL: these patterns overlap and co-occur - ADHD often coexists with ODD or conduct disorder.
Diagnose the pattern that best explains the behaviour, not the first disruptive label you see.

structure / axis · entity cell · exclusion / not present · pitfall

Fig 23.27. Cross-cutting discrimination of disruptive behaviour: conduct disorder, oppositional defiant disorder, DMDD, ADHD-driven disruptiveness and normal-limits behaviour separated on core disturbance, aggression, mood and the boundary with normal development.

42.1 The disruptive-behaviour differential: four disorders and one normal childhood that all look alike

Disruptiveness is a final common presentation, not a diagnosis. A great many child referrals arrive as some version of the same complaint: the child is difficult, defiant, aggressive, will not do as told, and is trouble at home or at school. That surface is shared by several distinct conditions, and the examined skill is to look past it to what the behaviour actually is. Four disorders and one band of normal development converge on this presentation, and each has been taught in full elsewhere in this chapter or its neighbours: **conduct disorder**, in which the defining feature is that the line crossed is other people's basic rights and major age-appropriate norms; **oppositional defiant disorder**, in which the pattern is an angry, argumentative and defiant stance aimed largely at authority figures, without that step into violating others' rights; **disruptive mood dysregulation disorder**, in which the outbursts sit on top of a persistently irritable and angry mood between them and the condition is at bottom a disorder of mood rather than of conduct; and the disruptiveness that is **secondary to attention-deficit/hyperactivity disorder**, where rules are broken through failure of regulation and impulse rather than through hostile intent, taught in the ADHD and specific learning/communication disorders notes. Against all four stands the ordinary difficult behaviour of a well child, which is the fifth thing the paragraph might be describing and the one most often forgotten.

None of these is re-taught here; each owning section carries its own criteria, and the local contrasts a candidate meets first - conduct disorder against oppositional defiant disorder, disruptive mood dysregulation disorder against paediatric bipolar disorder - are drawn where those disorders live. What the synthesis adds is the single grid that holds all five at once, so that a mixed vignette can be resolved by asking, in order, what kind of thing the behaviour is.

42.2 Reading the grid: the axes that actually separate them

The five separate on a small number of questions, asked in the right order. The grid below is not a list of features to match but a sequence of discriminating axes. The first and most powerful is the nature of the line the behaviour crosses. The second is whom the behaviour is aimed at and whether an abnormal mood is present between episodes. The third, applied last, is whether the picture clears once the developmental baseline and the setting are taken into account, which is the question that separates every one of the disorders from normal difficult behaviour.

PRESENTATION	WHAT THE BEHAVIOUR ESSENTIALLY IS	THE DISCRIMINATING QUESTION	MOOD BETWEEN EPISODES
Conduct disorder	Violation of others' basic rights and major societal norms - aggression, destruction, deceit, serious rule-breaking	Is the line crossed other people's rights, not merely adult authority?	Not the defining feature
Oppositional defiant disorder	An angry, argumentative, defiant and often vindictive stance toward authority	Is defiance of authority the ceiling, without rights-violation?	Irritability is part of the pattern but is not a sustained mood disorder
Disruptive mood dysregulation disorder	Severe recurrent temper outbursts on a background of persistent irritable, angry mood	Is the mood between the outbursts itself persistently irritable?	Persistently and observably irritable or angry
Disruptiveness secondary to attention-deficit/hyperactivity disorder	Rule-breaking driven by inattention, impulsivity and overactivity, not hostile intent	Is the trouble a failure of regulation rather than of intent?	Not primarily a mood problem
Difficult behaviour within normal limits	Age-expected defiance, temper or rule-testing, situational and outgrown	Does it clear once age, setting and impairment are weighed?	Age-appropriate, not sustained

The disruptive-behaviour differential as one grid. Each row's full criteria are taught in its own place; what is set out here is the order of questions that resolves a shared surface into one of five answers. The comparison is read down the third column.

Read this way the differential becomes a decision rather than a memory test. A hostile, aggressive boy who steals and intimidates has crossed into others' rights and points to conduct disorder; the same-looking boy who argues, defies and loses his temper with adults but does not cross that line points to oppositional defiant disorder; the child whose outbursts erupt from a mood that stays sour and irritable in between points to disruptive mood dysregulation disorder; the child who breaks rules because he cannot sit, wait or attend points back toward attention-deficit/hyperactivity disorder as the primary problem. And a good many children fitting none of these are simply behaving as their age allows.

TRAP **Collapsing every disruptive child into conduct disorder.** Aggression and rule-breaking pull the eye straight to the most severe label, and the weaker answer stops there. The disciplined answer asks what the behaviour actually is before naming it: defiance of authority is not violation of rights, a temper outburst from a persistently irritable mood is a mood disorder and not a conduct one, and dysregulated impulsivity is a disorder of regulation. Naming conduct disorder because the child is difficult, rather than because others' basic rights are being violated, is the single commonest error this part of the chapter tests.

42.3 Disorder against normal limits: the cross-cutting map

Every family in this chapter has a normal counterpart it must be told from. The developmental continuum was set out at the opening as the principle underneath the whole chapter, and it comes due here, because the second great discrimination is not between two disorders but between a disorder and ordinary childhood. Tantrums, separation distress, fears, bedwetting and repetitive self-soothing are not abnormal in themselves; they are expected at particular ages and present in well children. What lifts any of them out of normal development and into a diagnosis is not the behaviour but four features acting together, the same four the chapter has used throughout: whether it is **persistent** rather than a phase, **pervasive** across settings rather than confined to one, developmentally out of step for the child's age, and genuinely **impairing**. Each owning section fixes its own threshold in numbers; the map here holds the comparisons together so the levers can be seen doing the same work every time.

NORMAL DEVELOPMENTAL PHENOMENON	THE DISORDER IT MUST BE TOLD FROM	WHAT LIFTS IT OUT OF NORMAL LIMITS
Toddler and preschool temper tantrums	Oppositional defiant disorder	A persistent, pervasive defiant and irritable pattern continuing past the age tantrums subside, impairing across settings
Separation distress of infancy and early childhood	Separation anxiety disorder	Separation fear excessive for the child's age, persistent, and impairing rather than a self-limiting developmental stage
Age-typical fears (dark, strangers, animals, imaginary threats)	The childhood anxiety disorders	Fear out of proportion to the threat, persistent, and disabling day-to-day function
Bedwetting before continence is developmentally expected	Enuresis	Wetting continuing beyond the developmental age of expected continence, at the criterion frequency and duration
Benign repetitive self-soothing (rocking, thumb-sucking)	Stereotypies and tic disorders	Movements that are prolonged, fixed and impairing, distinguished from the sudden non-rhythmic character of tics

The normal-limits map across the chapter. Each disorder's exact threshold is taught in its own section and is deliberately named here without its numbers; the constant is the set of levers in the third column, which do the same job for every row.

The map is worth carrying whole because the same reasoning recurs in disguise. A three-year-old having tantrums, a toddler distressed at a parent's departure, a five-year-old frightened of the dark, a young child still wet at night, and an infant who rocks to settle are, far more often than not, well children doing what their age allows. The corresponding disorder is diagnosed only when the four levers pull together, and the exact age or frequency that anchors each threshold belongs to the section that owns it and to the Normal child development and milestones notes.

RULE **A behaviour never makes a childhood diagnosis; persistence, pervasiveness, developmental incongruence and impairment do.** The presence of a tantrum, a fear, a wet bed or a defiant refusal settles nothing on its own, because each is a normal event at some age. The diagnosis lives in whether the behaviour has become durable, generalised across settings, out of step for the age, and a genuine source of impairment. Read every childhood presentation through those four levers before reaching for a label.

PITFALL

The error here is the mirror of missing a disorder: over-diagnosing ordinary development. A firm parent, an anxious teacher or a crowded clinic can turn an age-appropriate tantrum, a normal separation protest or a still-maturing bladder into a referral and then into a label. Reaching for a diagnosis because the behaviour is present, rather than because it is persistent, pervasive, developmentally out of step and impairing, over-pathologises childhood and does its own harm. The two failures - under-recognising the genuine disorder and over-labelling the normal child - are opposite ends of the same missing judgement.

42.4 Putting the chapter's map to work

The two discriminations run on one underlying method. Whether the question is which disruptive disorder a child has or whether he has one at all, the move is the same: refuse the surface, name the axis the presentation turns on, and hold it against both its nearest diagnostic neighbour and its normal counterpart before committing. That method is what lets a single mixed vignette be answered cleanly, and it is why the chapter was built as a shape rather than a list. A candidate who carries the family map, the disruptive-behaviour grid and the normal-limits levers into the hall can take almost any child presentation the paper offers and place it, which is precisely what these marks reward.

- Sort the presentation into its family first, and notice when it straddles two - the acting-out child hiding an internalising disorder is the commonest such crossing.
- Within the behavioural group, ask what the behaviour essentially is - rights, authority, mood or regulation - before naming a disorder.
- For any symptom with a normal counterpart, apply the four levers before diagnosing, and be as willing to withhold a label as to give one.
- Cross-refer outward where the presentation reaches beyond this chapter, to attention-deficit/hyperactivity disorder and to the wider systems of assessment and protection, rather than forcing the answer to stay inside these pages.

IN INDIA

Two features of Indian practice sharpen both discriminations. The operative classification is ICD-11, so the disruptive-behaviour labels that reach case files are its dissocial and oppositional categories rather than the DSM names, and the reasoning of the grid transfers unchanged even though the wording differs. More consequentially, most of these children are first seen by a paediatrician, a general physician or a school rather than a child psychiatrist, in a setting with few dedicated child mental health services. That raises the stakes on the normal-limits map in both directions: a genuinely disordered internalising child is easily missed because the distress is quiet, while an ordinary tantruming or bedwetting child is easily over-labelled by an anxious family or an overburdened clinic. The synthesis is not an academic tidy-up here; it is what protects the well child from a diagnosis and the unwell one from being overlooked.

GOOD TO REMEMBER

Before writing any childhood diagnosis, name its two comparators out loud: the nearest disorder it could be confused with, and the normal developmental phenomenon it grows out of. If the presentation cannot be told convincingly from both, the label is not yet earned. This single habit prevents the two errors that dominate this chapter's examined material at once.

MNEMONIC

Family, then Axis, then the four Levers

The chapter's whole method in three moves. First place the child among the five **Families** seeded at the outset - distress turned Out and distress turned In, with the tic, attachment, and elimination and feeding disorders apart. Then, within the disruptive group, read the **Axis** the behaviour turns on: whether it is about others' rights (conduct disorder), authority (oppositional defiant disorder), mood (disruptive mood dysregulation disorder) or regulation (attention-deficit/hyperactivity disorder). Last, before any label, pull the four **Levers** that separate every disorder from its normal counterpart: persistent, pervasive, developmentally out of step, and impairing.

Refuse the surface: place the child in a family, read what the disruptive behaviour essentially is - rights, authority, mood or regulation - and gate every childhood diagnosis through the four levers of persistence, pervasiveness, developmental incongruence and impairment before you name it.

Rights	Authority	Mood	Regulation
CONDUCT DISORDER	OPPOSITIONAL DEFIANT DISORDER	DISRUPTIVE MOOD DYSREGULATION DISORDER	ADHD-DRIVEN DISRUPTIVENESS

43 · Worked vignettes across the five groups

A map is only as good as the child in front of you. Everything set out across this chapter has been taught one disorder at a time, each in its own place and against its own threshold. The examination does not ask questions that way. It hands you a paragraph about a real-seeming child

and asks you to do, under time, what a clinician does across a first consultation: hear a presenting complaint, recognise which family of disorder it belongs to, read it against what is normal for that age, look for what travels alongside it, and land on a discriminator before committing to an answer. The worked cases below rehearse exactly that movement. They are deliberately built to be entered through the loudest symptom, because that is how children actually arrive, and then to turn on the quieter fact that decides the diagnosis.

Each vignette is arranged around one of the chapter's five families, so that the set as a whole demonstrates the spine already laid down: distress turned outward and distress turned inward as one axis, and the tic, attachment, and elimination-and-feeding disorders standing apart from it with their own logic. None of these cases re-teaches a disorder; where the full criteria, doses and course live, this chapter has already set them out, and the vignette only points back to that place. The work here is the reasoning between symptom and label, which is the part a candidate most often skips and an examiner most reliably tests.

43.1 The behavioural door: aggression that may be more than conduct

A defiant, aggressive boy is the commonest externalising vignette, and the commonest trap. Consider a school-age boy brought by a worn-out mother because he fights, lies, breaks rules at home and at school, and will not be told. The loudest reading is a disruptive behaviour disorder, and the first real task is to place it: a pattern of serious rights-violating and rule-breaking conduct points one way, while argumentativeness, temper and defiance directed at authority without that serious violation points to the milder oppositional pattern. That distinction is drawn in full where each disorder is taught, and the vignette only requires you to know which set of behaviours you are reading. A stronger answer then asks whether the boy shows the **callous-unemotional** features that mark the limited-prosocial-emotions variant, because they change the prognosis and the plan, and are set out where the conduct disorder specifier is taught rather than re-listed here.

The decisive move, and the one that separates a safe candidate from a fluent-but-shallow one, is to refuse to stop at the behaviour. The child who acts out is the child most likely to be carrying an anxiety, trauma or mood disorder underneath, presenting through irritability and rule-breaking that look purely behavioural. Ask, deliberately, what sits beneath the conduct. The hyperactive, impulsive overlap that so often accompanies this picture belongs to the ADHD and specific learning/communication disorders notes, and is named here only so that you screen for it rather than fold it into a behavioural label.

GOOD TO REMEMBER

When a child arrives through the behavioural door, treat the referral as telling you which door the family came through, not the whole of what is inside the room. The acting-out presentation earns a deliberate screen for the anxiety, trauma and mood that hide behind conduct, and for the attentional problem that so often rides with it. The loudest symptom is a starting point, never the finish.

43.2 The emotional door, arriving in the body: pain, and the child who will not go to school

Internalising distress is referred late, quietly, and often through the body. Consider a child with recurrent morning stomach aches and headaches who becomes distressed at the school gate and stays home, yet is well and cheerful on weekends and holidays. The physical complaints are genuine and the family may have cycled through paediatric investigations before anyone asks about mood or fear. This is the **somatisation** presentation of childhood distress, the way anxiety and depression declare themselves in a child who has few words for either, and its full teaching sits where the somatic-symptom and pain presentations of childhood are set out in this chapter. The candidate names it and moves to the discrimination the vignette is really testing.

That discrimination is between two ways a child ends up out of school. **School refusal** is anxiety-driven: the child is at home, the parents know, and the anxiety is often about separation, about the school situation itself, or about a mood disorder pulling the child down. **Truancy** is conduct-driven: the child conceals the absence and is elsewhere, and it belongs with the externalising picture of the section above. The features that separate them are worth holding as a short set:

- whereabouts and parental knowledge: at home with the family versus concealed and elsewhere;
- the affect that drives it: fear and distress versus indifference or defiance;
- the company kept: alone or clinging to a caregiver versus with a peer group away from adults;
- and the family the disorder belongs to: emotional and internalising versus behavioural and externalising.

Behind the refusal, the candidate looks for the specific anxiety or mood disorder generating it, each taught in its own place in this chapter, and for the masked, irritable presentation that childhood depression so often takes. The general adult somatic-symptom and conversion entities, which are a different construct from this childhood somatisation, are the province of the Somatic-symptom, sexual, impulse-control disorders and suicide notes.

43.3 A neurodevelopmental door: movements and sounds that are not habits

The tic vignette is won on phenomenology, not on the movement itself. Consider a boy whose parents describe eye-blinking and throat-clearing that have waxed and waned over months, now joined by brief involuntary sounds, worse when he is tired or stressed, briefly suppressible with effort, and preceded by an uncomfortable urge that the movement relieves. That signature is a tic disorder, and the vignette is testing whether you can separate it from its mimics rather than whether you can name Tourette. The discriminating features cluster tightly: the **premonitory urge**, the brief voluntary suppressibility at a cost, the waxing-and-waning course, and the aggravation by stress and fatigue. Where the classification of tic disorders and the phenomenology of Tourette are set out, this is taught in full; the vignette needs only the recognition.

Against that signature the candidate lines up the look-alikes and lets each fall away. **Stereotypies** are rhythmic, fixed, self-soothing and typically begin earlier, often in a child with autism or intellectual disability, and lack the urge-and-relief quality. Chorea is flowing and non-

suppressible; dystonia is sustained and postural; a compulsion is performed to neutralise an intrusive thought rather than to relieve a bodily urge. Each of these separations is developed where tics are differentiated from other movements, and the vignette rewards knowing which single feature does the work in each case. The impairment in such a child, when it comes, is usually driven less by the tics than by the attention-deficit and obsessive-compulsive problems that so commonly accompany them; those comorbidities are named here and taught, respectively, in the ADHD and specific learning/communication disorders notes and the Anxiety, OCD and trauma-related disorders notes.

TRAP **Reading suppressibility as proof that the movements are voluntary.** A child who can briefly hold a tic down, or a parent who reports that he "can stop when he wants to," tempts the candidate to call the movements a habit or attention-seeking behaviour. Brief, effortful, uncomfortable suppressibility followed by a rebound is a defining property of a tic, not evidence against it, and the premonitory urge is precisely what makes suppression cost something.

43.4 A relational door: the child who came from an institution

Two children from the same depriving start present as mirror images. Consider a young child adopted after early institutional care. One is watchful and emotionally withdrawn, does not seek comfort when distressed and does not respond to it when offered, and shows little social or emotional reciprocity. Another, from a similar history, is strikingly over-familiar, wanders off with unfamiliar adults without checking back, and shows no wariness of strangers. The first is the emotionally withdrawn, internalising pattern; the second is the indiscriminate, disinhibited pattern. The **reactive attachment disorder** and **disinhibited social engagement disorder** that these two pictures represent are each taught in full in their own place; the vignette tests whether you read the direction of the disturbance correctly and, crucially, whether you anchor both to their required cause.

That cause is a history of **pathogenic care**, grossly insufficient or repeatedly disrupted caregiving, and it is what turns this from a temperament question into an attachment one. The discrimination the examiner is usually reaching for is from autism: the socially odd, poorly reciprocal post-institutional child can look autistic at a glance. What separates them is the pathogenic-care history itself, the tendency to improve once a stable and sensitive caregiver is in place, and the absence of the restricted, repetitive repertoire and the pervasive social-communication deficit that define autism. That attachment-side comparison is developed where RAD and DSED are differentiated from autism in this chapter; autism itself is the subject of the Intellectual disability and autism spectrum disorder notes and is not re-taught here. The management follows from the cause and is not pharmacological: a stable, responsive caregiving relationship is the intervention, developed where the attachment disorders' management and the effects of institutional care are set out.

43.5 A bodily-function door: the bed that is wet, and the food that keeps shrinking

Two brief cases share a family. First, a child who still wets the bed at night. The whole discrimination turns on the developmental baseline: at one age this is ordinary and expected, and at a later age, persistent and frequent, it is a disorder, with the exact threshold taught where

enuresis is classified and assessed. The vignette rewards asking the age and the pattern before reaching for a label, and it hides a genuine clinical danger in its treatment.

> **PITFALL**
>
> The tricyclic used for enuresis is cardiotoxic in overdose, and the therapeutic-to-toxic margin in a small child is narrow. Reaching for imipramine casually, or leaving a supply within a young child's or a sibling's reach, is the way this benign-seeming problem turns dangerous. The dose, the cautions and the safer first-line options are set out where the management of enuresis is taught; the point to carry from the vignette is that the drug is not a soft option and the first line is behavioural.

Second, a child whose eating has narrowed to a few textures and who is losing weight, but who has no fear of fatness and no disturbance of body image. This is the picture of **avoidant restrictive food intake disorder**, driven by sensory aversion, low interest in eating, or fear of a feared consequence such as choking, and it is discriminated from anorexia and bulimia precisely by the absence of the weight-and-shape motive. ARFID and that distinction are taught in full in their own place; anorexia and bulimia themselves belong to the Eating and sleep-wake disorders notes and are named here only as the contrast. The younger child eating non-food substances, and the infant regurgitating and re-chewing, are the other members of this bodily-function family, each taught where it sits.

43.6 Pulling the doors together: one method for any child

Every vignette above was entered through a door and decided somewhere else. The loud presenting symptom named a family; the diagnosis was settled by a quieter discriminator and by reading the child against a developmental baseline that keeps moving. That is the single transferable method, and it is worth seeing all five cases laid against it at once.

PRESENTING DOOR	FAMILY	THE DISCRIMINATOR THE CASE TURNS ON	WHERE IT IS TAUGHT IN FULL
Aggression, defiance, rule-breaking	Behavioural (externalising)	Severity of rights-violation, the callous-unemotional variant, and what internalising disorder hides beneath	Where conduct and oppositional disorders are set out
Recurrent pain, will not go to school	Emotional (internalising)	Anxiety-driven refusal versus concealed truancy; somatisation as the presenting language	Where the childhood anxiety, mood and somatic presentations are set out
Waxing movements and sounds, briefly suppressible	Tic disorders	Premonitory urge and suppressibility against stereotypy, chorea, dystonia and compulsion	Where tic classification and Tourette are set out
Withdrawn, or overfamiliar, after early deprivation	Attachment disorders	The pathogenic-care history, and improvement with a stable caregiver, against autism	Where the attachment disorders and their differentiation are set out
Bedwetting; narrowed, weight-losing eating	Elimination and feeding	Age against the developmental baseline; absence of a weight-and-shape motive in ARFID	Where enuresis and the feeding disorders are set out

The five worked cases read against the one method: a presenting door names the family, a quieter discriminator settles the diagnosis, and the full teaching lives in each disorder's own place.

RULE **The presenting complaint names the door, never the diagnosis.** The loudest symptom reliably tells you which of the five families you are in, and reliably fails to tell you which disorder within it, or what else the child is carrying. Read every childhood vignette by placing the family from the presenting picture, reading the picture against the developmental baseline for that age, screening across the outward-inward axis for what hides beneath, and only then committing to the discriminator that the case actually turns on.

IN INDIA

Which door a child reaches care through is shaped by the setting. In a family-based, service-scarce system these children very often meet a paediatrician, a general physician or a school first, and the externalising presentations, which trouble the adults around the child, arrive far more readily than the internalising ones, which cause the child the greater private suffering and are the likeliest to go unrecognised. The labels in the case file are usually ICD-11's rather than the DSM equivalents. The examination vignette written for Indian candidates leans, accordingly, on the presentations that reach an Indian clinic, and the method above is what lets you answer them without a specialist's caseload behind you.

MNEMONIC

Out, In, and three apart

The whole set of vignettes cashes in the chapter's spine. Two families share one axis: the child who turns distress **Out**, met in the behavioural case, and the child who turns it **In**, met in the emotional case that arrived through the body. **Three** families stand **apart** with their own logic: the tic disorders (neurodevelopmental and motor), the attachment disorders (relational, rooted in early care), and the elimination and feeding disorders (of bodily function). Place the child among those five, read against the developmental baseline, and the vignette becomes answerable.

Door	**Baseline**	**Across**	**Home**
THE LOUDEST SYMPTOM NAMES THE FAMILY	READ IT AGAINST DEVELOPMENTAL AGE	SCREEN THE OTHER SIDE OF THE AXIS	THE DISCRIMINATOR SETTLES THE LABEL

A childhood vignette's presenting door names the family, never the diagnosis: place the child among the five families, read the picture against the moving developmental baseline, screen across the Out-In axis for what hides beneath, and commit only to the discriminator the case actually turns on.

44 · Consolidation and active recall

The last hour before an examination is not the time to learn a disorder; it is the time to make sure you can find one. Everything in this chapter has by now been taught in full, each entity in its own place, with its criteria, its mechanism and its management. What has not yet been done, and what decides the marks, is the retrieval: whether the shape of the chapter is available to you under pressure, so that a mixed child presentation resolves into the right family and the right discrimination rather than into a blank or a guess. This closing section does no new teaching. It hands back the whole set as one structure, gives you a way to test that the structure is genuinely yours rather than merely familiar, and leaves you with the single mnemonic the chapter was built to earn.

Read what follows as a rehearsal rather than a summary. A summary you nod along to and forget; a rehearsal makes you produce the answer before you are allowed to see it, and that act of producing is what fixes the material and what an examiner is, in effect, paying you to be able to do.

44.1 Why the close is retrieval and not re-reading

Knowing that you have read something is not the same as being able to produce it. The most reliable finding in the psychology of learning, and the one most useful on the evening before a paper, is that **active recall** - deliberately generating an answer from memory - consolidates knowledge far more durably than re-reading, however attentive the re-reading feels. Re-reading breeds a sense of mastery that outruns the reality, because a page you have seen before is easy to process, and the ease is misread as knowledge - the **fluency illusion** that quiet revision generates and that active production dispels. Producing the answer with the page shut removes that

comfort and shows you, honestly, where the gaps are while there is still time to close them. This is why the heart of this section is a list of prompts with no answers printed beside them, and why you should resist the pull to treat them as trivia you already know.

RULE **Retrieve before you re-read.** When a prompt stops you, the instinct is to turn back to the section and read it again; the stronger move is to sit with the difficulty, reconstruct as much as you can from the family shape, and only then check. The struggle to retrieve, not the smoothness of the re-reading, is what makes the item available next time.

44.2 The whole chapter as one shape

Five families, and for each the single question the examination turns on. The chapter opened by resolving a loose-looking list into five families on two principles; it closes by reassembling them, but now keyed to retrieval. For each family the useful thing to carry is not the full criteria - those are taught where they belong - but the **discriminating question**, the pivot on which the marks in that family actually turn. The map below sets the five side by side against that pivot. It names the disorders it compares and does not re-teach them; the cross-cutting comparison that works those distinctions in detail is drawn earlier in this chapter, and the point here is only to have the pivots in a single view.

FAMILY	THE PIVOT THE MARKS TURN ON	THE DISCRIMINATION IT FORCES (TAUGHT IN FULL EARLIER)
Behavioural (externalising)	What is the behaviour violating - a rule, another person's rights, or the child's own mood control?	Oppositional defiant disorder against conduct disorder against disruptive mood dysregulation disorder, and each against the disruptiveness that rides with attention-deficit/hyperactivity disorder
Emotional (internalising)	Is the fear, worry or low mood developmentally expected, or persistent, pervasive and impairing for this age?	The separation, social and specific presentations of childhood anxiety, and childhood depression with its irritable and somatic disguises
Tic disorders	Is the movement involuntary and urge-driven, or purposeful and self-soothing?	Tics against stereotypies, chorea, dystonia and compulsions
Attachment disorders	Is there a history of grossly deficient care, and does the child withdraw or approach indiscriminately?	Reactive attachment disorder against disinhibited social engagement disorder, and both against autism spectrum disorder
Elimination and feeding	Is the bodily-function problem beyond the age at which it is expected, and is it wet, soiled, restricted or aberrant?	Enuresis and encopresis against age-appropriate continence; pica, rumination and avoidant/restrictive intake against the eating disorders proper

The five families reassembled for the close, each against the one question that carries its marks. The disorders are named, not re-taught; the detailed discriminations sit in their own sections and in the cross-cutting comparison drawn earlier in this chapter.

The map is a route back into the detail, not a substitute for it. Its use in the examination is fast and specific: a vignette lands, you place it into one of the five families within a sentence, and the family hands you its pivot, which is the question you must be seen to ask. A candidate who can name the family and state its pivot has already structured the answer; the remembered criteria then hang off that structure instead of arriving as an unsorted heap. Two families - the behavioural and the emotional - answer to the single outward-inward axis; the other three keep their own logic, and forcing them onto that axis is the error the opening warned against and the close will not undo.

44.3 The active-recall prompts, by family

Cover the section, answer aloud, then check. These are deliberately answer-free. Work them with the relevant section shut: say the answer out loud or write it, and only then open the chapter to mark yourself. A prompt you glide past with a vague nod is a prompt you have not passed. They run in the chapter's own order, one cluster per family, and each cluster asks you to produce the discrimination that the family's pivot demands.

Behavioural. How does defiance without aggression differ from the violation of others' rights, and how does chronic, non-episodic irritability differ from both? Which onset pattern of the aggressive disorder carries the worse trajectory, and toward which adult disorder does it point? What does the callous-unemotional pattern add, and why does it change the prognosis rather than the diagnosis?

Emotional. On what single feature does a childhood fear become a disorder rather than a stage? How does depression disguise itself in a child who never says he is sad? When a child complains only of stomach aches and headaches, which two internalising disorders should you actively look for behind the body, and why is that presentation not the same thing as the somatic symptom and conversion disorders proper?

Tic. What distinguishes an involuntary, urge-driven movement from a purposeful, rhythmic, self-soothing one, and which two comorbidities so define the fuller tic phenotype that they, more than the tics, drive the impairment? Why is the first-line treatment of tics behavioural rather than pharmacological?

Attachment. What single element of the history is required before either attachment disorder can be considered at all? How does the emotionally withdrawn pattern differ from the indiscriminately sociable one, and what tells both of them apart from the superficially similar social difficulty of autism spectrum disorder?

Elimination and feeding. At what point does wetting or soiling stop being a stage and become a disorder, and which of the enuresis treatments is the order-of-magnitude and cardiotoxicity trap in a child? What separates avoidant and restrictive eating from the eating disorders driven by body image?

TRAP **Mistaking recognition for recall.** Reading a prompt, thinking "yes, I know that", and moving on is the commonest self-deception of the last evening. **Recognition**, the sense that you have met an idea before, is a far weaker signal than being able to generate it cold, and the examination asks only for the second. If you did not actually say or write the answer, treat the prompt as unpassed and come back to it.

44.4 What the paper actually asks of this chapter

The marks reward placement and discrimination, not recitation. Very little in a child-psychiatry question is won by reproducing a criteria list intact. The examinable move, almost every time, is to take an untidy real-child presentation, place it in its family, and then draw the one discrimination that the family turns on - defiance from rights-violation from mood-driven irritability, expected fear from disorder, tic from stereotypy, deprivation-rooted attachment disturbance from autism, developmental wetting from enuresis. This is why the map and the prompts above are built around pivots rather than around definitions: the definitions are the raw material, and the discrimination is the finished answer. The child who sits visibly on one side of the outward-inward axis and quietly on the other is **the mixed presentation** to expect, and the answer that notices both sides is the one that scores.

IN INDIA

Two features of local practice should shape how you carry this chapter into the paper. First, the operative classification here is ICD-11, so the family names and category labels you produce should be its labels, even where the teaching above has moved between systems for clarity. Second, these children very often reach a paediatrician, a general physician or a teacher long before a child psychiatrist, and the outward-inward axis predicts which of them arrive at all: the disruptive, externalising presentations are brought in because they trouble the adults, while the internalising disorders, which cause the child the greater private suffering, are the ones a service-scarce system most readily misses. An answer that names where a disorder is likely to be first seen, and by whom, reads as clinically grounded rather than merely memorised.

PITFALL

The revision-stage error is to spend the final hours deepening the disorders you already know and avoiding the discriminations you find slippery, because the familiar material is pleasant to re-read and the slippery pairs are uncomfortable to work. This inverts the return on effort. The marks lost in this chapter are lost at the boundaries - conduct disorder against oppositional defiant disorder against disruptive mood dysregulation, the attachment disorders against autism, tics against stereotypies - and it is precisely the uncomfortable pairs, not the comfortable entities, that repay the last hour.

44.5 The one shape to carry in

If everything else falls away under pressure, this is what should remain. The chapter was built so that a single memorable shape would survive the examination hall even when individual criteria did not. It is the frame the opening laid down and every section since has filled: two families that share one axis and three that stand apart from it, all read against a **developmental**

baseline that keeps moving as the child grows. Hold that, and any presentation has somewhere to go before you have recalled a single threshold.

MNEMONIC

Out, In, and three apart

The child who turns distress **Out** is the behavioural, externalising group; the child who turns it **In** is the emotional, internalising group; these two share one axis. **Three** families stand **apart** from that axis with their own logic: the tic disorders (neurodevelopmental and motor), the attachment disorders (relational, rooted in deficient early care), and the elimination and feeding disorders (of bodily function). Every one of the five is judged against the developmental baseline, never against the behaviour alone. Five families, two principles, one shape.

GOOD TO REMEMBER

On the day, lead with the family, not the diagnosis. Placing a presentation as behavioural, emotional, tic, attachment or elimination-and-feeding takes a sentence and immediately narrows the field to the discriminations that matter, which is the ground the marks are on. Reaching first for a specific label invites the loudest symptom to capture the answer; reaching first for the family keeps the discrimination in view.

The prompts are only useful if they are worked rather than read, so a short method for running them:

- take one family at a time and shut the section before you begin;
- say or write the answer in full before you look, treating any prompt you only recognise as unpassed;
- check against the family map above, confirming you produced the pivot and not merely a definition;
- return to the pairs that stopped you after a gap of some hours rather than immediately, so the second retrieval is a genuine test;
- and leave the comfortable entities to last, spending the scarce final time on the uncomfortable boundaries.

Two families on one axis - distress turned Out (behavioural) against distress turned In (emotional) - and three families apart from it (tic, attachment, elimination and feeding), every disorder read against the moving developmental baseline: place the family first, then draw the discrimination its pivot demands.

Out	In	Apart
PLACE IT: WHICH FAMILY?	ASK IT: WHAT IS THE PIVOT?	DRAW IT: WHICH DISCRIMINATION?

Previously asked

Genuine past questions from this chapter's territory, with their board of origin. No catalogued theory question falls inside the disruptive behaviour and conduct disorders, the childhood emotional disorders, the attachment disorders, the tic disorders or the elimination and feeding disorders in the examined window held in the intel index, so none is printed here. No question is reconstructed, paraphrased into a new one, or invented to pad the list. That a topic has not been asked as a set-piece essay does not lower its yield: conduct disorder against oppositional defiant disorder and disruptive mood dysregulation, the callous-unemotional specifier, tic pharmacotherapy scoped to its indication, the imipramine enuresis dose and its cardiotoxicity, and the attachment-disorder differential from autism are all live short-note and viva territory, and the chapter is built to answer them.

TRAP **An unasked topic is not a low-yield topic.** The absence of a past essay is a fact about one board's recent papers, not about what an examiner may ask this year. Prepare the discriminations and the doses, not the past paper.

The quick review

One table, read down the left column, cover the right. Every line is taught in full somewhere above; nothing here is new. Where the sources disagree, the disagreement is the answer.

ASK	HOLD
WHAT DSM-5 REQUIRES FOR CONDUCT DISORDER	A repetitive pattern violating the basic rights of others or major age-appropriate norms, drawn from four clusters: aggression to people and animals, destruction of property, deceitfulness or theft, and serious rule violations. At least three of the fifteen criteria in the past year, at least one in the past six months. The clusters and the count are the marks.
THE TWO ONSET SUBTYPES AND WHY THEY MATTER	Childhood-onset (at least one symptom before age ten) carries the worse prognosis, more aggression and the stronger link to antisocial personality disorder; adolescent-onset is more normative in its peer context and remits more often. The subtype is a prognostic statement.
THE LIMITED-PROSOCIAL-EMOTIONS SPECIFIER	The callous-unemotional phenotype, applied when at least two of four features persist over twelve months across relationships and settings: lack of remorse or guilt, callous lack of empathy, unconcern about performance, and shallow or deficient affect. It marks a distinct developmental pathway, not merely severe conduct disorder.

ASK	HOLD
CONDUCT DISORDER AGAINST OPPOSITIONAL DEFIANT DISORDER	ODD is angry and irritable mood, argumentative and defiant behaviour, and vindictiveness, without the aggression to people and animals, destruction, theft and serious rights-violation of conduct disorder. ODD can precede conduct disorder; it is not a milder synonym for it.
PHARMACOTHERAPY OF AGGRESSION IN CONDUCT DISORDER	Psychosocial treatment is first-line: parent management training for younger children, multisystemic therapy for adolescents. Medication (risperidone the best evidenced) is an adjunct for serious aggression only, scoped to that indication. It is not a conduct-disorder cure and it is not automatically disruptive-mood-dysregulation-disorder evidence.
WHAT DSM-5 REQUIRES FOR DMDD	Severe recurrent temper outbursts out of proportion to provocation, on average three or more times a week, with persistently irritable or angry mood between outbursts, for twelve months across at least two settings, onset before age ten, diagnosed between ages six and eighteen. It is a depressive-spectrum disorder.
DMDD AGAINST PAEDIATRIC BIPOLAR DISORDER	DMDD is chronic, non-episodic irritability; paediatric bipolar is episodic mania, discrete periods of elevated or expansive mood with the syndrome change. Blurring the episodic-versus-chronic line is the error; DMDD was created precisely to catch the chronically irritable child who was being over-diagnosed as bipolar.
SOMATIC PRESENTATION AGAINST SOMATIC SYMPTOM DISORDER	Recurrent abdominal pain and headaches are how childhood anxiety and depression often present, a somatisation of distress. Somatic symptom disorder proper does NOT require the symptom to be medically unexplained; conversion, now functional neurological symptom disorder, is a distinct entity and is not reducible to mood somatisation.
SEPARATION ANXIETY AGAINST NORMAL DISTRESS	Developmental separation distress peaks in the toddler years and settles; the disorder is developmentally excessive fear of separation, persistent for at least four weeks in a child, with impairment. School refusal is anxious non-attendance with the child at home and the parents aware, distinct from concealed truancy.
PAEDIATRIC OCD AND PANDAS	PANDAS and the broader PANS are a CONTESTED hypothesis of abrupt-onset OCD or tics after streptococcal or other infection, not settled causation. Teach the definition and the controversy; presenting the streptococcal link as an established cause is the error.
THE YOUTH ANTIDEPRESSANT WARNING	An SSRI, usually fluoxetine, is the first-line medication for moderate to severe childhood depression, alongside psychotherapy. The boxed warning reflects a small increase in suicidal ideation and behaviour in the pooled paediatric trials, roughly four percent against two percent on placebo, with no completed suicides. The answer is to monitor closely early, not to withhold treatment.

ASK	HOLD
REACTIVE ATTACHMENT AGAINST DISINHIBITED SOCIAL ENGAGEMENT DISORDER	Both require a history of pathogenic care. Reactive attachment disorder is the emotionally withdrawn, inhibited child who does not seek comfort; disinhibited social engagement disorder is the indiscriminately sociable child who wanders off with an unfamiliar adult. Two patterns, one adverse-care root.
ATTACHMENT DISORDERS AGAINST AUTISM	The attachment-side view: a history of pathogenic care and clear improvement with a stable, sensitive caregiver point to attachment disorder, whereas the pervasive social-communication deficit and the restricted and repetitive behaviours of autism persist across environments. Autism itself is the intellectual disability and autism spectrum disorder notes' to teach.
WHAT DSM-5 REQUIRES FOR TOURETTE SYNDROME	Both multiple motor tics and at least one vocal tic at some time, not necessarily concurrently, present for more than one year, with onset before age eighteen. The premonitory urge and the waxing-and-waning course are the phenomenology; comorbid ADHD and OCD drive most of the impairment.
TIC PHARMACOTHERAPY, SCOPED TO THE INDICATION	Behavioural treatment first: habit reversal training within comprehensive behavioural intervention for tics. Then the alpha-2 agonists guanfacine and clonidine, and the antipsychotics aripiprazole, risperidone and the classical agents, each at its TIC dose. The tic-indication dose is not the ADHD-indication alpha-2 dose and not the autism-indication antipsychotic dose; every dose carries its indication.
TIC AGAINST STEREOTYPY, CHOREA AND DYSTONIA	Tics are sudden, non-rhythmic, involuntary and briefly suppressible with a premonitory urge; stereotypies are rhythmic, fixed, soothing and present from earlier; chorea is flowing and unpredictable; dystonia is sustained and twisting. The premonitory urge and the suppressibility mark the tic.
ENURESIS MANAGEMENT AND THE IMIPRAMINE TRAP	Behavioural measures and the bell-and-pad alarm are the durable first line; desmopressin is the most-used drug, oral preferred over intranasal for the hyponatraemia risk. Imipramine is reserved for refractory cases: the paediatric enuresis dose is tens of milligrams total at bedtime (around 50 mg for ages six to twelve, 75 mg over twelve), NOT a large per-kilogram figure, and its cardiotoxicity and overdose lethality are the reason the dose and the electrocardiogram matter.
ARFID AGAINST THE EATING DISORDERS	Avoidant restrictive food intake disorder is a failure to meet nutritional needs from restricted intake WITHOUT the body-image disturbance or the drive for thinness of anorexia and bulimia. Anorexia and bulimia are the eating and sleep-wake disorders notes'; ARFID is named and distinguished here, not taught there.

ASK	HOLD
ENCOPRESIS	Repeated passage of faeces into inappropriate places from age four, most often retentive with overflow soiling on a background of constipation; management is disimpaction, then maintenance laxatives and a scheduled toileting programme, not primarily a psychiatric drug.
JUVENILE DELINQUENCY	A legal and social construct, not a DSM or ICD diagnosis. The psychiatric task is to assess for the conduct disorder, ADHD, substance use and mood or trauma that often underlie it; the juvenile-justice framework is named and cross-referred to the child psychiatry: assessment, treatment, services and protection notes, and any statutory specific is read from the primary Act, not asserted here.
WHAT THE SOURCES REFUSE TO TELL YOU	Several India-specific prevalence figures for these disorders, the exact standardised scoring bands of some rating scales, and the precise upper age of the extended antidepressant warning are not settled in the literature consulted. The chapter says so where it happens rather than inventing them.

References

Every specific in this chapter is bound to one of the sources below, and the count is the number of claims each carried. Books and guidelines are cited author, title and year in the house convention; no PMID is asserted for a book. For every diagnostic criteria set the primary manual (DSM-5-TR 2022 and ICD-11 CDDR) is the authority, not a textbook paraphrase. This chapter touches no statute it reproduces: item 88 names the juvenile-justice framework and cross-refers rather than stating any Juvenile Justice Act specific. 9 claims are recorded at partial confidence (an honest open gap or a figure to confirm against a primary source at review), never an invented number. The must-have-papers cross-check was NOT performed for this Paper III chapter, because the paper lists do not yet exist in the intel index; for Paper III the topic bank plus the syllabus scaffolding is the completeness authority.

SOURCE	CLAIMS CARRIED
DSM-5-TR (2022)	188
Kaplan and Sadock CTP 2024	33
Lewis Child and Adolescent Psychiatry	29
Rutter's Child and Adolescent Psychiatry	22
Maudsley 15th ed	19
Stahl Prescriber's Guide / Essential Psychopharmacology	15
ICD-11 CDDR	11
PubMed	10

SOURCE	CLAIMS CARRIED
Dulcan's Textbook of Child and Adolescent Psychiatry	5
Hammad	5
Developmental Psychopathology (Cicchetti	4
Bridge et al. 2007	2
Chapter cross-reference (a pedagogy pointer, not a sourced fact)	2
Day-23 _OWNERSHIP-MATRIX.md	2
not found in parsed corpus	2
Clinical Rating Scales and Assessment in Psychiatry and Ment	1
Goodman and Gilman's Pharmacological Basis of Therapeutics	1
Hammad 2006 (Arch Gen Psychiatry) and Bridge 2007 (JAMA)	1
KDT Essentials of Medical Pharmacology	1
New Oxford Textbook of Psychiatry	1
Shorter Oxford Textbook of Psychiatry	1
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