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Psychosis Spectrum

The schizophrenia-spectrum closer in one document: the other primary psychotic disorders and how a delusion forms and dissolves, the delusional misidentification and named syndromes, and catatonia from its signs to malignant catatonia and its emergency differential.

CONTENTS

- Other primary psychotic disorders
- Delusional misidentification and named delusional syndromes
- Catatonia
- Apply and consolidate

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PAPER II

Other psychotic disorders, delusional syndromes and catatonia

Four sections · one complete notes document

This material closes the schizophrenia-spectrum block, gathering into one document the psychotic disorders that are not schizophrenia, the delusional syndromes, and catatonia. The first band is **other primary psychotic disorders**: the schizophrenia-spectrum decision tree that separates them by duration and mood-loading, then schizoaffective disorder, delusional disorder, the life-cycle of a delusion from its formation to its dissolution, the acute and transient psychotic disorders, brief psychotic disorder, the induced and shared delusional disorders, postpartum psychosis and the late-onset psychoses, the attenuated psychosis syndrome, and the interleaved differential that tells them apart. The second band is **delusional misidentification and named delusional syndromes**: Capgras, Fregoli, intermetamorphosis and the subjective doubles, and the named monothematic syndromes of De Clerambault, Othello, Cotard and Ekbom, each keyed on its core delusional content. The third band is **catatonia**: the classical and modern signs and the examination, the psychiatric, neurological and medical causes and the transdiagnostic placement, the lorazepam challenge and benzodiazepine management, malignant catatonia and its differential from neuroleptic malignant syndrome, and the place of electroconvulsive therapy. The examinable skill is discrimination: to place a psychotic presentation on the spectrum by its duration, mood-loading and delusion-type, to name a syndrome by its core content, and to recognise catatonia and its malignant form and act.

HOW TO USE THESE NOTES

Read the three bands as one exercise in telling psychotic presentations apart. The **other primary psychotic disorders** band opens with the spectrum decision tree, then teaches each disorder against schizophrenia and against each other, and closes on the interleaved differential; each named entity ends with a **Good to remember** box giving what it is, the core criteria or the one discriminating feature, and the one number or eponym to hold. The **delusional misidentification and named syndromes** band draws the eponymous syndromes as grids keyed on the core delusional content, distinguishing which are the content of a delusion from which are named disorders. The **catatonia** band runs from the signs and their examination, through the causes and the transdiagnostic placement, to the lorazepam challenge and the malignant-catatonia emergency. Figures declare one axis and code it in colour: **petrol** marks structure and the neutral case, **coral** marks the trap or the danger, and **marigold** highlights the number or eponym to hold. Several boundaries are worth stating up front: the phenomenology, aetiology, criteria and course of schizophrenia itself are taught in the Schizophrenia: aetiology, phenomenology, classification and course notes and are not repeated here; the antipsychotic drugs and neuroleptic malignant syndrome as an entity belong to the Schizophrenia: management, antipsychotics and adverse effects notes, needed here only inside the malignant-catatonia differential; the technique of electroconvulsive therapy lives in the ECT and neurostimulation notes; the mood-episode criteria for schizoaffective disorder live in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; and the raw descriptive definitions of the individual delusions and misidentification phenomena live in the Psychometry, Assessment and Descriptive Psychopathology notes, applied here rather than re-derived. This is doctor-facing revision, so the full technical vocabulary is used, and every criterion, eponym and number is verified against a source.

Other primary psychotic disorders

1 · Orientation: the schizophrenia-spectrum decision tree

Why this matters. Schizophrenia does not stand alone. It sits at the centre of a **schizophrenia-spectrum** family of related psychotic disorders that share the same positive symptoms but are told apart by three things the examiner returns to again and again: how long the illness has lasted, whether a full mood episode is riding on top of it, and what kind of delusion is present. Get those three axes straight and the whole block resolves into a clean **decision tree**. This orienting topic builds that tree, seeds the master mnemonic that chains the chapter, and hands the reader a map to the delusional syndromes and to catatonia that follow. The illness schizophrenia itself is not re-taught here: its phenomenology, aetiology, criteria and course are owned by the Schizophrenia: aetiology, phenomenology, classification and course notes and are cross-referenced, not re-derived.

The governing idea: everything in this family is a positive-symptom psychosis, so you never separate them by "is there psychosis" but by the three discriminators. The **criteria map** below is worth memorising as a single object, because in the exam a stem rarely asks "define

schizophreniform"; it hands you a duration and a mood picture and asks you to place the patient. Nosology here is given ICD-11 primary (the current CDDR grouping, "schizophrenia and other primary psychotic disorders") with the DSM-5-TR labels cross-mapped, because the two systems draw the duration lines differently and the difference is itself examinable.

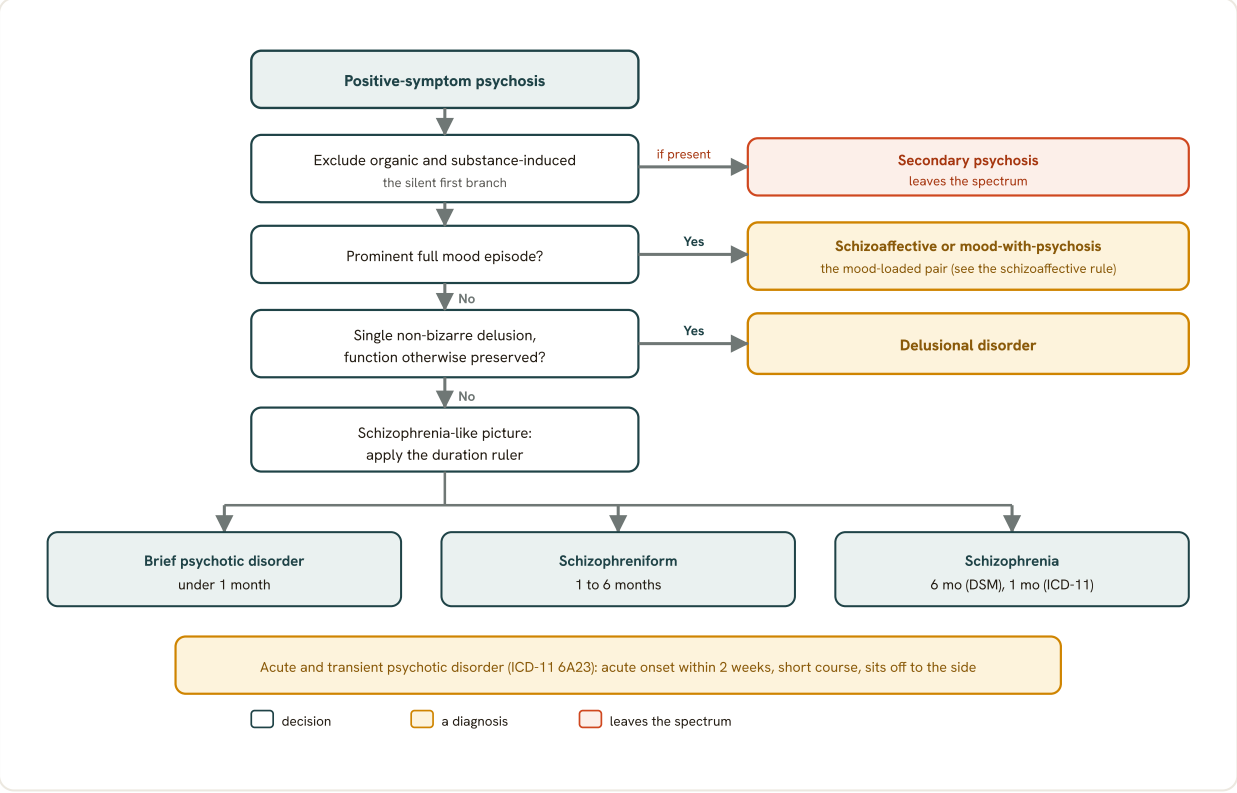


Fig 10.1 The schizophrenia-spectrum decision tree. Exclude the organic and substance-induced first (the silent branch), then walk three axes in order: mood-loading (a prominent mood episode pulls out schizoaffective disorder and mood-with-psychotic-features), delusion-type (a single non-bizarre delusion with preserved function is delusional disorder), and finally, for the remaining schizophrenia-like pictures, the duration ruler (under one month, one to six months, six months or more), with the acute and transient psychotic disorder sitting off to the side.

1.1 The spectrum as one object: what belongs, what does not

The schizophrenia-spectrum and other primary psychotic disorders, in the ICD-11 grouping, comprises schizophrenia, **schizoaffective disorder**, schizotypal disorder, the acute and transient psychotic disorders, and delusional disorder (ICD-11 CDDR). The DSM-5-TR chapter, "Schizophrenia Spectrum and Other Psychotic Disorders", covers the same ground plus brief psychotic disorder and schizophreniform disorder as separate short-duration entities, and organises itself explicitly along a gradient: sub-threshold conditions first, then single-domain conditions (delusional disorder, catatonia), then the time-limited psychoses, then schizophrenia (Kaplan & Sadock 2024). Two members earn their place here for reasons worth naming: **catatonia**, which the chapter carries because it is a psychomotor syndrome that cuts across the whole grouping, and **postpartum (puerperal) psychosis**, owned here as a psychotic presentation while the wider obstetric frame belongs to the Perinatal/women's mental health and emergency psychiatry notes.

What does not belong: a psychosis that is a direct physiological consequence of a substance, medication or medical condition is a secondary psychosis and is excluded before any spectrum

label is applied. That exclusion is the silent first branch of the whole tree, and forgetting it is the classic beginner error.

RULE **Exclude the organic and the substance-induced first, then place on the spectrum.** A secondary psychosis is not a point on this decision tree; it is what you rule out before you enter it.

1.2 Axis one, duration: the timeline that separates the short psychoses from schizophrenia

The first and most heavily tested axis is **duration**. Holding the same positive-symptom picture constant, DSM-5-TR splits the family purely by how long it has run. Brief psychotic disorder lasts at least one day and remits within one month, with full return to premorbid function; below one month is its signature (Kaplan & Sadock 2024). Schizophreniform disorder occupies the middle band, at least one month but less than six, and, crucially, does not require a decline in functioning. Schizophrenia is diagnosed once the illness has run six months or more (including at least one month of active-phase symptoms). Alongside these sit the acute and transient psychotic disorders (ATPD, ICD-11 6A23), defined not by a fixed ceiling but by an acute onset, psychotic symptoms emerging without a prodrome and reaching the full picture within two weeks, and a characteristically short, fluctuating course.

A trap the examiner sets deliberately: the two systems do not agree on where schizophrenia begins. ICD-11 requires symptoms for one month or more, so many cases that DSM-5-TR would call schizophreniform (one to six months) are already schizophrenia in ICD-11 (Kaplan & Sadock 2024). Read the stem for which system it is using.

under 1 mo	1 to 6 mo	6 mo or more	within 2 wk
BRIEF PSYCHOTIC DISORDER	SCHIZOPHRENIFORM (DSM-5-TR)	SCHIZOPHRENIA (DSM-5-TR)	ATPD ACUTE ONSET (ICD-11)

TRAP **Do not swap the duration thresholds between systems.** DSM-5-TR schizophrenia needs six months; ICD-11 needs one month, so DSM schizophreniform maps largely onto ICD-11 schizophrenia.

1.3 Axis two, mood-loading: does a full mood episode ride on the psychosis

The second axis, **mood-loading**, asks whether a prominent, full mood episode (depressive or manic) is part of the picture, and if so, how it sits in time against the psychosis. Two members of the family carry a prominent mood episode by definition: schizoaffective disorder and mood disorder with psychotic features. Two members carry none: delusional disorder and schizophrenia (where any mood symptoms are, by rule, a minority of the total course). The whole discrimination turns on a single temporal criterion. In DSM-5-TR schizoaffective disorder, a major mood episode is present for the majority of the total duration of the illness (Criterion C), and there must have been at least two weeks of delusions or hallucinations in the absence of a major mood episode at some point in the lifetime course (Criterion B) (Kaplan & Sadock 2024). If instead the delusions occur exclusively during mood episodes, the diagnosis is mood disorder with psychotic features. The mood-episode criteria themselves are cross-referenced to the Mood

disorders: classification, depressive and bipolar syndromes, neurobiology notes and not re-derived here.

TRAP **Never diagnose schizoaffective without the at-least-two-weeks-of-psychosis-without-prominent-mood rule.** Psychosis only ever during mood episodes is mood disorder with psychotic features, not schizoaffective disorder.

1.4 Axis three, delusion-type and bizarreness: where delusional disorder splits off

The third axis reads the **content and bizarreness** of the delusion and the state of the rest of the person. Delusional disorder is the anchor case: at least one month of one or more delusions, classically **non-bizarre** (though DSM-5-TR now allows a "with bizarre content" specifier), with no other active-phase symptom of schizophrenia and, strikingly, apart from the direct impact of the delusion itself, function and behaviour are not obviously bizarre and psychosocial functioning is largely preserved (Kaplan & Sadock 2024). This is the discriminator to hold: a well-systematised, encapsulated, often plausible-sounding delusion in an otherwise intact person points to delusional disorder, whereas the same content embedded in disorganisation, hallucinations and negative symptoms points to schizophrenia. Marneros frames it as a disorder in which the delusion is well-systematised and long-lasting while other mental and personality domains stay comparatively intact (New Oxford Textbook of Psychiatry). The raw descriptive definitions of the delusion types and of the misidentification phenomena are cross-referenced to the Psychometry, Assessment and Descriptive Psychopathology notes.

RULE **Delusional disorder is a single non-bizarre delusion in an otherwise preserved person.** No prominent hallucinations, no disorganisation, no negative symptoms, and no functional collapse beyond what the delusion itself drives.

1.5 The decision tree assembled: reading a stem in order

The three axes chain into an ordered walk, and reading them in this order is the fastest route through any placement stem. First, exclude the organic and substance-induced (the silent branch). Second, ask the mood question: is a full mood episode prominent, and what is its temporal relation to the psychosis, which pulls out mood-with-psychotic-features and schizoaffective disorder. Third, if not mood-loaded, ask the content question: is this a single non-bizarre delusion in an otherwise intact person, which pulls out delusional disorder. Fourth, for the remaining schizophrenia-like pictures, apply the duration ruler: under one month (brief psychotic), one to six months (schizophreniform), six months or more (schizophrenia), with the ATPD sitting off to the side as an acute-onset, short-course entity. The illness schizophrenia is the tree's endpoint and is fully taught in the Schizophrenia: aetiology, phenomenology, classification and course notes; here it is only the destination the other branches are defined against. This ordered tree is the concept figure for the block.

WORKED EXAMPLE

A 24-year-old man, well until three weeks ago, is brought with florid persecutory delusions and auditory hallucinations of abrupt onset; no drugs on screen, no mood episode, no prior episodes. Walk the tree: organic and substance-induced excluded; no prominent mood episode (not schizoaffective, not mood-with-psychosis); the picture is not a single encapsulated delusion in an intact person (not delusional disorder); on duration it is under one month with acute onset. The answer is brief psychotic disorder (DSM-5-TR), and given the acute, prodrome-free onset within two weeks, an acute and transient psychotic disorder (ICD-11). Only if it runs past one month, then past six, does it climb to schizophreniform and then schizophrenia.

1.6 The criteria map in one table

The single table below is the object to memorise: each row is a spectrum member, and the columns are the three discriminating axes. The highlighted cell in each row is the one feature that clinches that diagnosis against its neighbours.

DISORDER (DSM-5-TR / ICD-11)	DURATION AXIS	MOOD-LOADING AXIS	DELUSION / DISCRIMINATOR
Brief psychotic disorder (F23)	**at least 1 day, under 1 month, full recovery**	none required	any positive symptom; often stress-triggered
Schizophreniform disorder (F20.81)	**1 to 6 months**	none required	schizophrenia picture, no functional-decline requirement
Schizophrenia (F20.9 / 6A20)	6 months or more (DSM); 1 month or more (ICD-11)	mood a minority of total course	**full active-phase picture; taught elsewhere**
Schizoaffective disorder (F25 / 6A21)	concurrent, over the illness course	**mood episode for majority of course, plus at least 2 weeks psychosis without prominent mood**	psychosis and mood interwoven
Mood disorder with psychotic features	within mood episodes	**psychosis only during mood episodes**	mood-congruent or incongruent delusions
Delusional disorder (F22 / 6A24)	1 month or more of delusion	none prominent	**single non-bizarre delusion, function otherwise preserved**
Acute and transient psychotic disorder (6A23)	**acute onset, full picture within 2 weeks, short course**	variable	often polymorphic, rapidly shifting

The schizophrenia-spectrum criteria map: read each row across the three axes; the highlighted cell is the clinching discriminator.

MNEMONIC

"SPECTRUM by Duration, Mood, Delusion; then the DELUSIONAL syndromes; then CATATONIA moves."

The master chain for the whole chapter. First place the psychosis on the SPECTRUM using the three axes, Duration, Mood-loading, Delusion-type. Then work the named DELUSIONAL syndromes (the jealous, erotomanic, somatic and persecutory types, and the misidentification set). Then finish on CATATONIA, the transdiagnostic psychomotor syndrome that "moves" across the whole grouping. Duration, Mood, Delusion is the three-axis core the examiner rewards.

1.7 India and the systems footnote

The acute and transient psychoses are of particular Indian relevance: acute-onset, brief, full-recovery non-affective psychoses are seen commonly in Indian practice and carry, on the whole, a favourable outcome, which is why ATPD and its ICD-10 antecedents are heavily represented in Indian teaching and examination (Kaplan & Sadock 2024, on the cycloid-psychosis and ATPD overlap). Delusional disorder, by contrast, typically presents in middle to later adult life and is comparatively uncommon in first-contact samples. This material is largely generic and disorder-defining rather than drug-focused; where any pharmacology is needed it is given as a pointer, and the antipsychotic detail is cross-referenced to the Schizophrenia: management, antipsychotics and adverse effects notes.

INDIA

The acute and transient psychotic disorders are a familiar and clinically important presentation in Indian settings: an abrupt, florid, often polymorphic psychosis in a previously well adult, frequently remitting fully within a short course. Treat the episode, then watch longitudinally, because a minority declare themselves later as schizophrenia or a mood disorder on relapse.

HOLD THIS

Every disorder in this family is a positive-symptom psychosis; you separate them by three axes only, duration, mood-loading and delusion-type, after first excluding the organic and substance-induced.

PEARL

Schizophreniform disorder is often **provisional**: if you make it before six months have elapsed and the illness has not yet remitted, you are labelling a work in progress. About one-third of patients recover within the six-month window and keep the schizophreniform label; the majority of the rest convert to schizophrenia or schizoaffective disorder (Kaplan & Sadock 2024).

GOOD TO REMEMBER

- **Schizophrenia spectrum:** the ICD-11 grouping of primary psychotic disorders (schizophrenia, schizoaffective, schizotypal, ATPD, delusional disorder); told apart by duration, mood-loading and delusion-type after organic/substance causes are excluded.
- **Brief psychotic disorder (F23):** at least one day and under **one month** with full recovery; the shortest member; any positive symptom, often stress-related.
- **Schizophreniform disorder (F20.81):** the same picture lasting **one to six months** with no functional-decline requirement; provisional if diagnosed early; introduced by Langfeldt in 1939.
- **Schizophrenia:** DSM-5-TR **six months or more**, ICD-11 **one month or more**; the endpoint of the duration axis, taught in the Schizophrenia: aetiology, phenomenology, classification and course notes.
- **Schizoaffective disorder (F25 / 6A21):** a mood episode present for the majority of the illness plus **at least two weeks** of psychosis without prominent mood (DSM-5-TR Criteria B and C); the two-week rule is the whole discrimination.
- **Delusional disorder (F22 / 6A24):** at least **one month** of a single non-bizarre delusion with function otherwise preserved and no other active-phase symptom; the one-domain psychosis.
- **Acute and transient psychotic disorder (6A23):** acute onset reaching the full psychotic picture **within two weeks**, short fluctuating course, often polymorphic; favourable outcome, clinically common in India.

2 · Schizoaffective disorder

Why this matters. **Schizoaffective disorder** is the schizophrenia-spectrum diagnosis the examiner uses to test whether you can hold two ideas at once: that a patient can have psychosis meeting the full schizophrenia bar together with a full mood episode, and that this is still not enough to call it schizoaffective unless a precise temporal rule is satisfied. The whole topic turns on one gate and one balance: a period of psychosis without a prominent mood episode, and mood symptoms occupying the majority of the illness. Get those two right and the diagnosis is secure; blur them and you have simply relabelled a mood disorder with psychotic features.

The governing sentence: schizoaffective disorder requires that, over the lifetime of the illness, delusions or hallucinations were present for a period of at least **two weeks** in the *absence* of a prominent mood episode, while mood symptoms meeting full criteria were present for the majority of the total duration of the illness (Kaplan & Sadock 2024, DSM-5-TR 2022). The disorder itself, its phenomenology, aetiology, criteria and course, is owned by the Schizophrenia: aetiology, phenomenology, classification and course notes and is not re-taught here; the raw mood-episode criteria (major depressive, manic, mixed) are cross-referenced to the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; and the drug detail is cross-referenced to the Schizophrenia: management, antipsychotics and adverse effects notes.

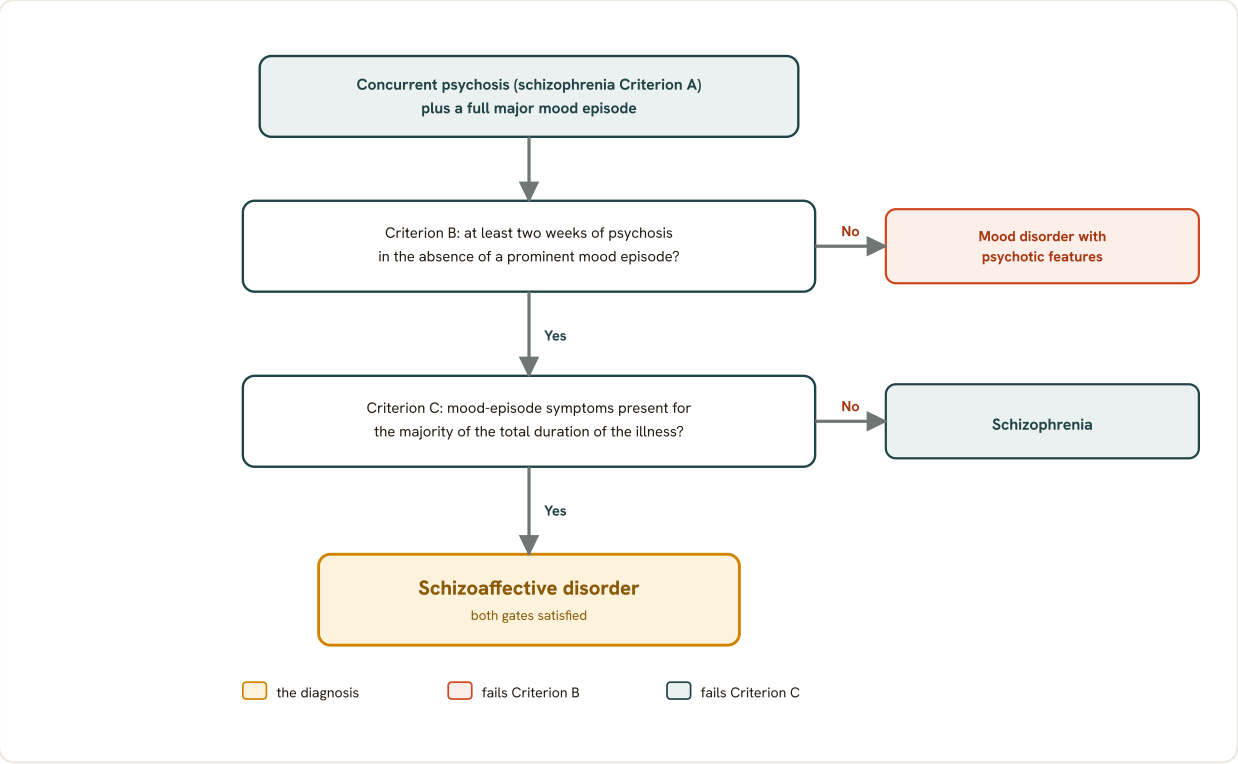


Fig 10.2 The schizoaffective decision rule (DSM-5-TR). Criterion B, at least two weeks of psychosis in the absence of a prominent mood episode, separates it from a mood disorder with psychotic features; Criterion C, mood-episode symptoms for the majority of the total illness, separates it from schizophrenia. Both gates must be satisfied. ICD-11 substitutes onset timing (simultaneous or within a few days, each lasting at least one month) and roughly equal prominence within a single episode.

2.1 The origin: Kasanin and a diagnosis born between two houses

The term schizoaffective psychosis was coined by Jacob Kasanin in a 1933 paper, describing a subgroup of nine young patients with good premorbid functioning, sudden onset of both schizophrenic and mood symptoms, and good outcome (Kasanin 1933, Kaplan & Sadock 2024). Kraepelin had already noticed that his own rich case material included many patients with features of both dementia praecox and manic-depressive illness, and Bleuler held that the co-occurrence of mood and schizophrenic symptoms in the same episode did not change the diagnosis of schizophrenia. Kasanin's category has survived every classification since, but a modern reading of his original case histories finds that all nine could today be diagnosed as a mood disorder with psychotic features, which is a fair emblem of the diagnosis it seeded: real enough to keep, unstable enough to argue about.

2.2 The DSM-5-TR criteria: the two-week gate and the majority rule

DSM-5-TR frames the diagnosis around an uninterrupted period of illness and four criteria (DSM-5-TR 2022). **Criterion A** requires an uninterrupted period during which there is a major mood episode (major depressive or manic) that is a **concurrent mood** episode running alongside psychosis meeting Criterion A of schizophrenia; because anhedonia is common in schizophrenia, a qualifying major depressive episode must include depressed mood itself (Criterion A1), not merely loss of interest. **Criterion B** is the defining rule: delusions or hallucinations for two or more weeks in the *absence* of a major mood episode during the lifetime duration of the illness. **Criterion C** requires that mood-episode symptoms are present for the majority of the total

duration of the active and residual portions of the illness. **Criterion D** excludes substances and another medical condition.

The two criteria that do the diagnostic work point in opposite directions. Criterion B separates schizoaffective disorder from a mood disorder with psychotic features: it demands a stretch of *pure* psychosis, at least two weeks, with no prominent mood episode running. Criterion C separates it from schizophrenia: it demands that mood symptoms dominate the majority of the illness, not a brief slice of it. A presentation can fail either way, and the diagnosis lives only in the narrow band where both hold.

RULE **Two weeks of psychosis without a prominent mood episode, plus mood for the majority of the illness.** Criterion B (the two-week pure-psychosis window) rules out a mood disorder with psychotic features; Criterion C (mood for the majority of the total duration) rules out schizophrenia. Both must hold.

TRAP **Calling any co-occurring psychosis and mood "schizoaffective".** Concurrent psychotic and mood symptoms alone do not make the diagnosis: without the two-week period of psychosis in the absence of a prominent mood episode, this is a mood disorder with psychotic features, where psychosis is confined to the mood episode.

2.3 A worked temporal example

DSM-5-TR gives a canonical pattern worth memorising. An individual has prominent auditory hallucinations and persecutory delusions for two months before a major depressive episode begins; psychosis and the full depressive episode then run together for four months; the depression lifts but the psychosis persists a further one month before it too clears (DSM-5-TR 2022). Total illness about seven months: psychosis alone in the first two months and the last one month (three months of pure psychosis, well over the two-week gate), with depression present for four of the seven, a majority. Both Criterion B and Criterion C are satisfied, so this qualifies as schizoaffective disorder.

WORKED EXAMPLE

Contrast the DSM-5-TR counter-case: a patient with a four-year history of active and residual schizophrenia develops depressive and manic episodes that, taken together, occupy less than one year of those four years. Criterion C fails, mood does not reach the majority of the illness, so the diagnosis is schizophrenia, not schizoaffective disorder, even though full mood episodes did occur.

2.4 Bipolar type and depressive type

DSM-5-TR requires a subtype specifier. The bipolar type (F25.0) applies if a manic episode is part of the presentation, whether or not depressive episodes also occur; the depressive type (F25.1) applies if only major depressive episodes are part of the presentation (DSM-5-TR 2022). The split is not cosmetic: bipolar type tends to present in younger adults, depressive type in older adults, and the bipolar/unipolar division maps onto the split running through the mood disorders themselves, whose full episode criteria are set out in the Mood disorders: classification,

depressive and bipolar syndromes, neurobiology notes. A **with catatonia** specifier is available and coded separately.

2.5 DSM-5 versus ICD-11: two framings of the same problem

The two systems agree on the spirit but differ in the letter, and the examiner likes the contrast. DSM-5-TR builds the diagnosis on the lifetime-course architecture above: a **concurrent mood** episode alongside schizophrenia-criterion psychosis, plus the two-week rule (Criterion B) and the majority rule (Criterion C). ICD-11 collapses the frame onto the current episode: its essential feature is that all the diagnostic requirements for schizophrenia are met concurrently with mood symptoms meeting the requirements of a moderate or severe depressive episode, a manic episode or a mixed episode, with the two symptom sets roughly equally prominent within the same episode (ICD-11 CDDR). The ICD-11 boundary text is exact: the onsets of the psychotic and mood symptoms are either simultaneous or occur within a few days of one another, and both must last at least one month; if the psychotic symptoms without mood symptoms end up much longer than the concurrent phase, the episode is better called schizophrenia. ICD-11 diagnoses episode by episode, so a previous schizoaffective episode does not preclude a later diagnosis of schizophrenia, and vice versa.

FEATURE	DSM-5-TR	ICD-11 CDDR
Unit of diagnosis	Lifetime, uninterrupted period of illness	**Current or most recent episode**
Core requirement	Mood episode concurrent with schizophrenia Criterion A	Schizophrenia requirements met concurrently with a full mood episode, roughly equally prominent
Pure-psychosis rule	**Delusions or hallucinations for at least two weeks in the absence of a prominent mood episode (Criterion B)**	No two-week rule; uses onset timing (simultaneous or within a few days) and duration
Mood-burden rule	**Mood symptoms for the majority of the total duration (Criterion C)**	Psychotic and mood symptoms concurrent and each lasting at least one month
Subtypes	Bipolar type (F25.0), depressive type (F25.1)	Course specifiers (first/multiple episode); type read from the mood episode

The two-week gate and the majority rule are DSM-5-TR devices; ICD-11 substitutes onset-timing and equal-prominence within a single episode.

2.6 The validity controversy: a diagnosis that will not sit still

Schizoaffective disorder is the classic example of a category of debated reliability and validity that sits between the mood and the psychotic disorders, and whose membership shifts with the criteria used (Kaplan & Sadock 2024). The instability is documented, not rhetorical. In a series of roughly 700 first-admission patients with psychotic features, 6% received a diagnosis of schizoaffective disorder at discharge, but only half of these (about 3% of the total) met DSM-III-R research criteria on review; two years later, only one-third of those originally diagnosed were still

called schizoaffective, one-third had become schizophrenia, and one-third an affective disorder (Kaplan & Sadock 2024). Family, twin and adoption studies find raised risks of both schizophrenia and affective disorder in relatives, marking it as a heterogeneous condition, and recent phenomenological work has failed to support the validity of the very two-week cutoff on which the DSM diagnosis rests. In practice the diagnosis is made more often than a strict application of the criteria would justify.

COMMON TRAP

Do not treat the schizoaffective label as a stable, once-and-for-all diagnosis. Because the relative proportion of mood to psychotic symptoms changes over time, DSM-5-TR itself expects the diagnosis to migrate: a severe four-month depressive episode in the first six months of a chronic psychosis may qualify as schizoaffective, then be revised to schizophrenia if psychosis persists for years without a further mood episode. The diagnosis is provisional by design.

at least 2 weeks PSYCHOSIS WITHOUT A PROMINENT MOOD EPISODE (CRITERION B)	**about one-third as common** AS SCHIZOPHRENIA	**0.3%** LIFETIME PREVALENCE, FINNISH SAMPLE (DSM-5-TR)

2.7 Course, prognosis and where it sits on the spectrum

Prognostically the disorder behaves as its name suggests, intermediate: outcome is better than schizophrenia but worse than a pure mood disorder (Kaplan & Sadock 2024, DSM-5-TR 2022). Negative symptoms and cognitive deficits occur but tend to be less severe and less persistent than in schizophrenia, and insight, though often impaired (anosognosia is common), may be less globally lost. Prevalence is about one-third that of schizophrenia, with a lifetime figure of **0.3%** in a Finnish sample and a female preponderance under DSM-IV criteria; the DSM-5 Criterion C (mood for the majority of the illness) is more stringent than the DSM-IV requirement of a merely substantial portion, so measured rates are expected to fall. This intermediate position, better than schizophrenia and worse than mood disorder, is exactly what makes its place on the schizophrenia spectrum contested.

2.8 Management pointers

This is a diagnosis topic, not a treatment topic, so the management is given as pointers only. The principle is combination: an **antipsychotic** to treat the psychotic dimension plus **mood-targeted treatment** for the affective dimension (a mood stabiliser or lithium for bipolar type, an antidepressant added with the usual caution for depressive type), the two chosen to match the subtype (Kaplan & Sadock 2024). Several second-generation antipsychotics carry mood benefit that suits this population, and the full drug detail, mechanisms, dosing and adverse effects, is cross-referenced to the Schizophrenia: management, antipsychotics and adverse effects notes; the mood-episode criteria that determine which affective arm to treat are set out in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes. Paliperidone is the one antipsychotic with a specific schizoaffective licence, but treat brand and licensing detail as belonging to the management notes.

INDIA

In Indian practice the diagnosis is used but, as elsewhere, applied loosely, and the same instability applies: an initial schizoaffective label frequently resolves to schizophrenia or to a mood disorder with psychotic features on longitudinal follow-up. Management follows the same combination logic (antipsychotic plus mood-targeted agent); brand-level detail belongs to the Schizophrenia: management, antipsychotics and adverse effects notes.

MNEMONIC

2 WEEKS PURE, MOOD FOR MOST

Two weeks of pure psychosis (no prominent mood episode) is the gate that excludes a mood disorder with psychotic features (Criterion B); mood for most of the illness is the balance that excludes schizophrenia (Criterion C). Hold both halves and the DSM-5-TR diagnosis is complete.

HOLD THIS

Schizoaffective disorder = at least two weeks of psychosis in the absence of a prominent mood episode (Criterion B), with mood symptoms present for the majority of the total duration of the illness (Criterion C); miss the two-week rule and it is only a mood disorder with psychotic features.

GOOD TO REMEMBER

- **Schizoaffective disorder:** a chronic psychosis with a full mood episode concurrent with schizophrenia-criterion psychosis; the discriminating rule is delusions or hallucinations for at least **two weeks** in the absence of a prominent mood episode (DSM-5-TR Criterion B); coined by Kasanin (1933).
- **Criterion B (the two-week gate):** at least two weeks of psychosis without a prominent mood episode over the lifetime of the illness; separates it from a mood disorder with psychotic features; the exam number is **two weeks**.
- **Criterion C (the majority rule):** mood-episode symptoms present for the majority of the total duration of the active and residual illness; separates it from schizophrenia; the phrase to hold is "majority of the total duration".
- **Bipolar type (F25.0) vs depressive type (F25.1):** bipolar type if a manic episode is part of the presentation (younger adults); depressive type if only major depressive episodes (older adults); the distinguishing feature is the presence or absence of a manic episode.
- **ICD-11 framing:** schizophrenia requirements met concurrently with a full mood episode, roughly equally prominent within the same episode, onsets simultaneous or within a few days, each lasting at least 1 month; no two-week rule, the number to hold is 1 month.
- **Validity controversy:** debated reliability and validity, sitting between mood and psychotic disorders, membership shifting with criteria; the demonstrative figure is that at 2-year follow-up only about one-third of those first diagnosed retain the label (Kaplan & Sadock 2024).

3 · Delusional disorder

Delusional disorder is the diagnostic home for the patient who holds one or more fixed false beliefs yet is, in every other respect, unremarkable: the personality is intact, the affect fits the belief, work and relationships hold up outside the delusional zone, and there are none of the hallucinations, thought disorder or negative symptoms that would push the diagnosis toward

schizophrenia. The examiner loves it precisely because it is a disorder defined by what is *absent* as much as by what is present, and because the classic error, calling it schizophrenia the moment a delusion appears, is so easy to make. This is a clinical-disorder topic: the schizophrenia symptoms it is contrasted against are owned by the Schizophrenia: aetiology, phenomenology, classification and course notes, and the raw descriptive definitions of each delusion type (persecutory, grandiose, jealous, erotomanic, somatic) are cross-referenced to the Psychometry, Assessment and Descriptive Psychopathology notes rather than re-derived here.

How to use this page. Fix the core first (a **non-bizarre** delusion for at least a month, function otherwise preserved, no prominent hallucinations or other schizophrenia symptoms), then the **subtypes** by delusional theme, then Kendler's **dimensions of delusional** severity as a rating frame that cuts across the subtypes, and finally the epidemiology and the management pointers. The duration threshold is the single fact most often swapped in a viva: DSM-5-TR asks for one month, ICD-11 for three.

3.1 The core: a non-bizarre delusion with preserved function

What defines it. Delusional disorder is the presence of one or more persistent delusions, classically **non-bizarre** (plausible in principle, drawn from ordinary life: being followed, deceived by a spouse, loved from afar, infested), in the *absence* of the other features that would make the diagnosis schizophrenia (Kaplan & Sadock 2024, ICD-11 CDDR). There are no prominent or persistent hallucinations, no formal thought disorder, no passivity or first-rank phenomena, and no negative symptoms; apart from the actions and attitudes flowing directly from the delusional system, affect, speech and behaviour are typically unaffected. Personality is relatively preserved, and social and occupational functioning show much less deterioration than in schizophrenia (ICD-11 CDDR). The one nuance introduced by DSM-5 is that the *non-bizarre requirement was removed* from the core criterion and replaced by a "with bizarre content" specifier, so a bizarre delusion no longer excludes the diagnosis in DSM; the non-bizarre character remains the prototype and is retained in the ICD tradition (Kaplan & Sadock 2024).

RULE **A non-bizarre delusion with otherwise preserved functioning.** The whole diagnosis is one or more circumscribed delusions with the personality, affect and function outside the delusional zone essentially intact, and none of the hallucinations, thought disorder or negative symptoms of schizophrenia.

TRAP **Calling it delusional disorder when function is broadly impaired or other psychotic features dominate.** If there is global deterioration, persistent hallucinations, formal thought disorder or negative symptoms, the diagnosis is schizophrenia (or a mood-with-psychosis disorder), not delusional disorder.

3.2 Duration and the diagnostic boundaries

The duration threshold, the fact most often swapped. The delusion must persist for a minimum period: DSM-5-TR requires the delusion(s) for **at least 1 month**, whereas ICD-11 (6A24) requires it **typically for at least 3 months** and often much longer (DSM-5-TR 2022, ICD-11 CDDR). Both systems permit hallucinations only if they are non-prominent, non-

persistent and thematically tied to the delusion, the classic example being tactile hallucinations in delusional infestation. The differential is drawn against three neighbours and one exclusion, and holding the discriminator for each is the exam-yield here.

CONTRAST	SHARED FEATURE	DISCRIMINATOR (WHAT MAKES IT DELUSIONAL DISORDER)
Schizophrenia	Persistent delusions	**No prominent hallucinations, thought disorder, passivity or negative symptoms; personality and function preserved; later onset**
Mood disorder with psychotic features	Persecutory, grandiose or somatic delusions	**Delusions occur at times *without* any mood episode; a full manic/depressive syndrome is absent or brief relative to the delusion (mood criteria in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes)**
Paranoid personality disorder	Suspiciousness, guardedness, litigiousness	**A frank, held delusion is present, not merely a pervasive distrustful trait; may be diagnosed alongside the personality disorder**
Illness anxiety / OCD / body dysmorphic disorder	Somatic or contamination preoccupations	**Belief is held with delusional conviction and insight is absent; if only an overvalued idea, code the primary disorder with the "absent insight / delusional beliefs" specifier**
Organic / substance cause	Delusions (often persecutory)	**Excluded first: delirium, dementia, basal-ganglia and limbic disease, amphetamine/cocaine; if present, code psychotic disorder due to another condition**

The differential is drawn by absence: delusional disorder is what remains once schizophrenia, a mood syndrome, a personality trait, an overvalued idea and an organic cause are each excluded (Kaplan & Sadock 2024, ICD-11 CDDR).

3.3 The subtypes by delusional theme

Named by content, not by severity. Both DSM-5-TR and ICD-11 classify delusional disorder into **subtypes** according to the predominant delusional theme (DSM-5-TR 2022). The raw descriptive definition of each delusion is owned by the Psychometry, Assessment and Descriptive Psychopathology notes; here the subtype is defined operationally by its central theme.

Persecutory type

The central theme is being conspired against, cheated, spied on, followed, poisoned, maligned or obstructed; the commonest subtype.

Grandiose type

The central theme is a great unrecognised talent, insight or discovery, or a special identity or relationship.

Jealous type

The central theme is that a spouse or partner is unfaithful (delusional jealousy, the Othello theme); the one subtype more common in men.

Erotomanic type

The central theme is that another person, usually of higher status, is in love with the patient (De Clerambault syndrome, psychose passionelle).

Somatic type

The central theme is bodily: infestation, malfunction, deformity or odour (older term monosymptomatic hypochondriacal psychosis); presents to dermatology, surgery or dentistry, rarely to psychiatry.

Mixed type

No single theme predominates; more than one of the above coexists. (An unspecified type covers themes that fit none of the above.)

MNEMONIC

PG-JES-M

The subtypes in one string: **P**ersecutory, **G**randiose, **J**ealous, **E**rotomanic, **S**omatic, **M**ixed. Persecutory is the commonest; jealous is the one that skews male; erotomanic is the one with the eponym (De Clerambault).

3.4 The named eponyms attached to the subtypes

Three eponyms travel with the themes. The examiner expects the eponyms, but also expects you to know they are *presentations*, not diagnoses in their own right: each can arise in delusional disorder, in schizophrenia, in a mood disorder or organically. De Clerambault syndrome is the erotomanic theme (a person of higher status is secretly in love with the patient, who reads rejection as concealed courtship). Othello syndrome is the delusional-jealousy theme (the conviction of a partner's infidelity, with checking behaviour and a real risk of violence). Ekbom syndrome is delusional infestation or delusional parasitosis, the prototypical somatic-type presentation, often with linked tactile hallucinations of things crawling on or under the skin.

TRAP **Calling any morbid jealousy Othello syndrome.** Othello syndrome is *delusional* jealousy; obsessional or overvalued jealousy, or jealousy secondary to alcohol or a mood state, is not, and the same theme can be a symptom of schizophrenia or a mood disorder rather than delusional disorder.

3.5 Kendler's dimensions of delusional severity

A rating frame, not a subtype scheme. Where the subtypes classify a delusion by *content*, Kendler's **dimensions of delusional** experience classify it by *form and severity*: a set of largely independent axes along which any single delusion can be rated, and along which two patients with the "same" persecutory belief may differ enormously (Kendler 1983). The point for the exam

is that **delusional severity** is multidimensional, so a delusion is not simply "present or absent" but strong or weak on several separable measures, and these dimensions do not correlate tightly with each other.

Conviction
The degree of certainty with which the belief is held, from partial doubt to absolute incorrigibility.

Extension
How far the delusion reaches into other areas of the person's life and world (a single encapsulated idea versus a pervasive delusional system).

Bizarreness
How implausible or impossible the belief is by ordinary cultural standards.

Disorganisation
How internally consistent and systematised the belief is, versus fragmented and contradictory.

Pressure
The degree of preoccupation, the amount of mental space and time the delusion occupies.

Affective response (a commonly-taught later addition, not in Kendler's original five)
The intensity of emotion attached to the belief.

Deviant (acting-out) behaviour (a commonly-taught later addition, not in Kendler's original five)
The extent to which the person acts on the delusion (stalking, litigation, assault), the dimension of greatest forensic weight.

Because these are independent, they are useful for *rating* and tracking a delusion (and for understanding why an encapsulated, low-pressure, non-acted-on delusion is compatible with preserved function) rather than for assigning a subtype. This dimensional view sits alongside the standard classifications and is worth naming when a question asks how a delusion is characterised beyond its theme (Kendler 1983).

> **PEARL**
> Content and form answer different questions. If asked to *classify* a patient's delusion, give the subtype (persecutory, jealous and so on); if asked to *characterise or rate* the delusion, reach for the dimensional frame: Kendler's original five (conviction, extension, bizarreness, disorganisation, pressure), with affective response and acting-out commonly added. The "acting-out" dimension is the one that drives risk and admission, whatever the theme.

3.6 Epidemiology and course

Uncommon, and later than schizophrenia. Delusional disorder is relatively uncommon: a general-population study from Finland found a lifetime prevalence of DSM-IV delusional disorder of **0.18%**, and in clinical series it accounts for roughly **1% to 4%** of psychiatric admissions (Kaplan & Sadock 2024). The **age of onset** is characteristically later than in schizophrenia, generally in the **mid- to late-30s**, usually insidious, and the delusions once formed tend to be stable and fixed (Kaplan & Sadock 2024, ICD-11 CDDR). The persecutory subtype is the most common; other than the jealous type, which is more common in men, the

disorder is roughly equally distributed between the sexes, with males tending to a younger onset. Functioning is generally preserved, with adverse outcomes arising mainly when the person acts on the belief; a minority progress to schizophrenia over time, and a premorbid personality disorder (classically paranoid) is over-represented.

0.18%	mid to late 30s	1 mo / 3 mo
LIFETIME PREVALENCE (FINLAND, DSM-IV)	TYPICAL AGE OF ONSET	MINIMUM DELUSION DURATION (DSM-5-TR / ICD-11)

3.7 Management pointers

Engagement first, then cautious pharmacology. The management of delusional disorder is dominated by one problem: these patients rarely see themselves as ill, so they present late, reluctantly, and often to a non-psychiatrist. The first task is therefore *engagement and the therapeutic alliance*, working with the distress and the functional consequences rather than confronting the belief head-on, since a frontal challenge to a fixed delusion tends to entrench it and end the consultation. Pharmacologically, antipsychotics are used but the *evidence base is limited* and largely uncontrolled: response is generally more modest and less reliable than in schizophrenia, and the drug detail (agent choice, dosing, adverse effects) is cross-referenced to the Schizophrenia: management, antipsychotics and adverse effects notes rather than re-taught. Comorbid *depression* is common (sometimes an understandable response to the delusional predicament) and should be identified and treated, as it worsens course. Risk assessment is integral, particularly in the jealous and erotomanic subtypes where acting-out (violence, stalking) is the main danger.

WORKED EXAMPLE

A 42-year-old, employed and socially intact, presents only because his wife insists, convinced over eight months that a neighbour is conducting an affair with her; he checks her phone and clothing and has confronted the neighbour once. There are no hallucinations, no thought disorder, no negative symptoms, and no mood episode; outside this belief he functions normally. This is delusional disorder, jealous subtype (Othello theme): non-bizarre delusion, over the duration threshold, preserved function, no other schizophrenia features. The clinical priority is risk (delusional jealousy carries a real risk of violence), then engagement, then a cautious antipsychotic trial and screening for comorbid depression, with the belief itself worked around rather than argued against.

INDIA

In Indian practice, delusional disorder classically presents to non-psychiatric specialists first: the somatic type (delusional infestation, Ekbom syndrome, and delusional parasitosis) reaches dermatology and general physicians, and the jealous type surfaces in the context of marital and family conflict, so much of the early clinical experience is of engaging a patient who does not accept a psychiatric frame. Diagnosis follows the ICD-11 (and long-familiar ICD-10) three-month non-bizarre-delusion template that Indian teaching texts use. The system-wide rule that no diagnosis is named to the patient sits comfortably here: management is framed around the distress and its consequences rather than the label, which also aids engagement. Generic antipsychotics dominate and are affordable; verify the local brand before naming one.

HOLD THIS

Delusional disorder is one or more non-bizarre delusions for at least a month (DSM-5-TR; three months in ICD-11), with personality, affect and function preserved outside the delusion and none of the hallucinations, thought disorder or negative symptoms of schizophrenia; classified by theme into persecutory (commonest), grandiose, jealous (male-predominant, Othello), erotomanic (De Clerambault), somatic (Ekbom) and mixed subtypes, onset in the mid- to late-30s.

GOOD TO REMEMBER

- **Delusional disorder (the diagnosis):** one or more persistent non-bizarre delusions with function otherwise preserved and no other schizophrenia symptoms; the discriminator from schizophrenia is preserved personality/function plus absence of prominent hallucinations, thought disorder and negative symptoms; the number to hold is the duration, at least 1 month (DSM-5-TR) or 3 months (ICD-11 6A24).
- **Subtypes (by theme):** persecutory, grandiose, jealous, erotomanic, somatic, mixed; the discriminator is the predominant delusional content; persecutory is the commonest and jealous the one more common in men.
- **De Clerambault syndrome (erotomanic type):** the delusion that a higher-status person secretly loves the patient; the discriminator is the reading of rejection as concealed courtship; the eponym to hold is De Clerambault (psychose passionelle).
- **Othello syndrome (jealous type):** the delusion of a partner's infidelity; the discriminator from ordinary or obsessional jealousy is delusional conviction; the fact to hold is its association with violence risk.
- **Ekbom syndrome (somatic type):** delusional infestation / delusional parasitosis, often with tactile hallucinations; the discriminator is the fixed conviction of infestation despite normal examination; presents to dermatology, not psychiatry.
- **Kendler's dimensions of delusional severity (the rating frame):** the original five are conviction, extension, bizarreness, disorganisation and pressure (Kendler 1983), with affective response and acting-out commonly added in later descriptive-psychopathology teaching; the discriminator from the subtypes is that these rate form and severity, not content.
- **Epidemiology and management (the pointers):** lifetime prevalence about 0.18%, onset mid- to late-30s, function preserved; engagement and alliance first, antipsychotics with limited evidence, treat comorbid depression; drug detail cross-referenced to the Schizophrenia: management, antipsychotics and adverse effects notes.

Figure. A concept figure contrasting delusional disorder with schizophrenia along two axes (breadth of impairment and presence of non-delusional psychotic features), with the delusional

disorder region marked as narrow-impairment, delusion-only, is flagged for central drawing.

4 · The life-cycle of a delusion: formation and dissolution

A delusion is not a static false belief that a patient simply "has"; phenomenologists describe it as a process with a beginning, a crystallisation and, in recovery, a dissolution. Two German-tradition observers frame the two ends of that arc. **Klaus Conrad** traced the *formation* of an evolving psychosis as a staged transformation of the whole field of experience, and **Karl Jaspers** named the discrete primary *forms* in which a true (schizophrenic) delusion first appears. At the far end, Eugen Bleuler's clinical observations of how a settled delusion coexists with, or is sealed off from, ordinary reality describe the ways a psychosis is contained or worked through.

Why it matters. This topic supplies the descriptive spine for the delusion viva: it lets you narrate *how* a persecutory system came into being, distinguish a primary delusion (heavily weighted in the diagnosis of schizophrenia) from a secondary one, and explain why a chronically deluded patient can hold a bizarre belief and a mundane life side by side without felt contradiction. The raw definitions of each delusional form are cross-referenced to the Psychometry, Assessment and Descriptive Psychopathology notes rather than re-derived here; the disorder schizophrenia itself (its phenomenology, criteria and course) is owned by the Schizophrenia: aetiology, phenomenology, classification and course notes.

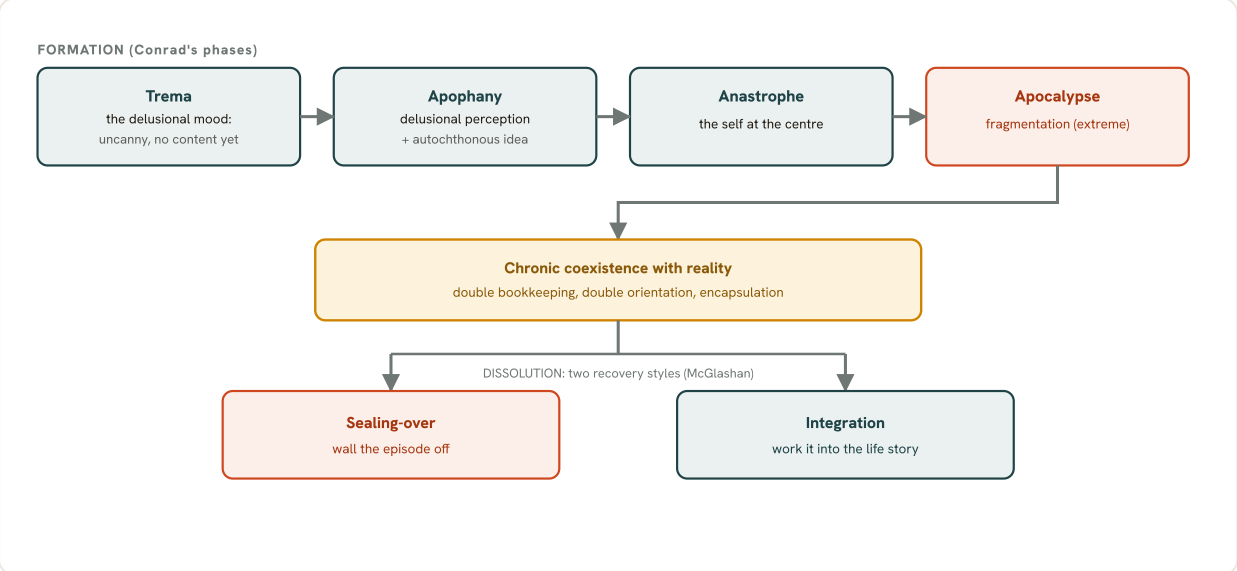


Fig 10.3 The life-cycle of a delusion. Formation runs through Conrad's phases, the trema (delusional mood, an uncanny atmosphere) preceding the apophany (the crystallised delusional perception), then anastrophe and, at the extreme, apocalypse. Once formed, a delusion coexists with reality (Bleuler's double bookkeeping and double orientation, and encapsulation), and at recovery is either sealed over or integrated into the life story.

4.1 Formation: Conrad's phases of an evolving psychosis

Klaus Conrad (1958), studying young soldiers with acute schizophrenia, described the beginning of psychosis as a sequence of gestalt transformations rather than a single event (Conrad 1958; reviewed by Mishara 2010). The sequence is a favourite viva scaffold because it maps the subjective experience onto Jaspers' forms.

Trema

The opening delusional mood (Conrad's word, borrowed from the German for pre-performance stage-fright): a tense, uncanny atmosphere that something momentous is impending, a total change in the perception of the world with no explicit content yet (Mishara 2010).

Apophany

The apophanous phase: the sudden crystallisation of a specific meaning where the patient finds new significance for events. This is the moment of the delusional perception and the autochthonous delusional idea, when "now it all makes sense" (Mishara 2010).

Anastrophe

The world comes to revolve around the self; the patient is placed at the centre of the reorganised field of meaning, heightening the psychosis.

Apocalypse

At the extreme, the disintegration or fragmentation of experience, with dissolution of the coherent world into perplexity and terror.

Conrad's fuller German scheme runs to six phases in all, the four acute phases above followed by *consolidation* (a new psychological world is built on the new meanings) and *residuum* (an eventual defect or autistic end-state); the AIIMS Clinical Methods summary gives a five-stage version (trema, apophany, anastrophe, consolidation, residuum) that folds away the apocalyptic phase (Conrad 1958; AIIMS Clinical Methods 2024, citing Mishara 2010). For the exam the load-bearing point is the ordering of the early phases: the **trema** (delusional mood) is the *prodromal atmosphere* that precedes the **apophany** (the crystallised delusional perception).

RULE **Mood before meaning.** Conrad's trema, the delusional mood, precedes the apophanous delusional perception: the uncanny atmosphere comes first, the specific "now it all makes sense" crystallisation comes second.

4.2 Jaspers' primary delusional forms, applied to this arc

Karl Jaspers separated **primary** delusions (arising *de novo*, ununderstandable, un-derivable from any preceding mental event, and given considerable diagnostic weight in schizophrenia) from **secondary** delusions (psychologically understandable from mood, culture or other symptoms). Four primary forms are recognised; each is a moment on Conrad's formation arc. The crisp descriptive definitions live in the Psychometry, Assessment and Descriptive Psychopathology notes and are only cross-referenced here (Sims 2015; Shorter Oxford 2018).

PRIMARY FORM	ONE-LINE CHARACTER	POINT ON THE ARC
Delusional mood (delusional atmosphere)	**A state of perplexity: something uncanny is going on that involves the patient in unspecified ways, content not yet fixed**	Conrad's *trema* (prodromal)
Delusional perception	**A real, normal percept is invested with a new, self-referential delusional meaning not derivable from mood; Schneider's "two-memberedness"**	Conrad's *apophany*
Sudden delusional idea (autochthonous delusion / delusional intuition)	**A fully formed conviction appearing in an instant "like a bolt from the blue", a single-stage event (no antecedent percept)**	Conrad's *apophany*
Delusional memory (retrospective delusion)	A past memory (real or false) is reinterpreted delusionally, often to explain the illness	Later elaboration

Jaspers' four primary delusional forms mapped onto Conrad's phases. Note the discriminator between delusional perception (two stages: normal perception, then delusional meaning) and the autochthonous sudden idea (one stage: the idea simply arrives), verified against AIIMS Clinical Methods 2024 and Kaplan & Sadock 2024.

WORKED EXAMPLE

A 25-year-old man saw the yellow leaves of the tree outside his house falling and, in that instant, knew this was a signal from God that he was "the chosen one", the sole person able to intercept it. The *percept* (yellowing leaves) is entirely normal; the delusion is the immediate, self-referential meaning attached to it, with no pre-existing delusional system to derive it from. That is a **delusional perception** and its "two-memberedness" (percept, then meaning) is Schneider's diagnostic hallmark (AIIMS Clinical Methods 2024).

4.3 Dissolution: how a delusion is held, contained and given up

Once formed, a delusion may persist, encapsulate, or dissolve. The descriptive vocabulary for the *persistence-alongside-reality* end is Bleulerian; the vocabulary for *recovery* comes from the schizophrenia outcome literature.

Double bookkeeping

Bleuler's observation that the delusion and everyday reality are held in parallel *without felt contradiction*: the patient who insists their food is poisoned will nevertheless eat it, and the espoused belief is not consistently acted upon (Bleuler; Kaplan & Sadock 2024).

Double orientation

The delusion is held with conviction yet has little influence on feelings and actions: a patient may believe himself a member of the royal family while living contentedly in a group home; this separation usually occurs in chronic schizophrenia (Shorter Oxford 2018).

Delusional encapsulation

An *encapsulated delusion* is walled off so that it has limited impact on the person's overall behaviour, the belief coexisting with otherwise intact functioning (AIIMS Clinical Methods 2024).

At recovery, two contrasting *recovery styles* are described (McGlashan, Levy and Carpenter 1975). In sealing-over, the patient walls the psychotic episode off, minimising and encapsulating it as alien and best not examined ("sealing over"). In **integration**, the patient works the experience into the life story, remaining curious about it and weaving it into a continuous sense of self. Integration is generally associated with better insight and engagement; sealing-over with more guarded, defended recovery.

TRAP **Coexistence is not insight.** Do not read double bookkeeping (delusion and reality coexisting, the belief not acted upon) as regained insight: the conviction is intact, only the behavioural consequence is muted.

PEARL

Double bookkeeping, double orientation and encapsulation all describe a delusion that *survives* alongside reality; sealing-over and integration describe two ways the episode is *metabolised* in recovery. Keep the two axes separate in an answer: the first is about how the belief coexists with reality during illness, the second about the stance the patient takes toward the episode afterwards.

4.4 India note and the figure to hold

India. In much Indian public-sector practice the delusional-formation vocabulary is examined from the descriptive-psychopathology tradition (Fish, Sims, and the AIIMS Clinical Methods text), and delusional perception and the autochthonous sudden idea are high-yield viva points precisely because they are the primary forms carrying the greatest diagnostic weight for schizophrenia. Candidates are expected to name the eponyms (Conrad, Jaspers, Schneider, Bleuler) and to distinguish the one-stage sudden idea from the two-stage delusional perception; misattributing these, or swapping the order of trema and apophany, is a classic error.

HOLD THIS

Trema (delusional mood) precedes apophany (delusional perception): mood first, crystallised meaning second, world-around-self (anastrophe) next, fragmentation (apocalypse) at the extreme.

GOOD TO REMEMBER

- **Conrad's phases:** the staged formation of an evolving psychosis; core sequence trema (delusional mood) to apophany (delusional perception) to anastrophe (self at centre) to apocalypse (fragmentation); eponym Conrad 1958.
- **Trema:** the opening delusional mood, a tense uncanny "something is impending" atmosphere with no fixed content; discriminator = prodromal, precedes any specific delusion.
- **Apophany:** the apophanous crystallisation where a specific meaning appears and "now it all makes sense"; discriminator = the moment of the delusional perception and autochthonous idea.
- **Delusional perception:** a normal percept given new self-referential delusional meaning; discriminator = Schneider's "two-memberedness" (two stages), eponym Schneider 1959.
- **Sudden (autochthonous) delusional idea:** a fully formed delusion arriving in one step "like a bolt from the blue"; discriminator = single-stage, no antecedent percept.
- **Delusional memory:** a past memory reinterpreted delusionally (retrospective delusion); discriminator = the falsification lies in the recall, not a current percept.
- **Double bookkeeping:** delusion and reality held in parallel without felt contradiction, belief not consistently acted on; eponym Bleuler.
- **Double orientation:** delusion held with conviction but little effect on feeling or action, typical of chronic schizophrenia; discriminator = conviction intact, behaviour unaffected.
- **Delusional encapsulation:** the belief walled off with limited impact on overall behaviour; discriminator = preserved functioning around a contained delusion.
- **Sealing-over vs integration:** two recovery styles, walling the psychosis off (sealing-over) versus working it into the life story (integration); eponym McGlashan 1975.

5 · Acute and transient psychotic disorders

The **acute and transient psychotic disorders** are the schizophrenia-spectrum group defined by their *tempo* rather than their content: an abrupt onset from a well state into full psychosis, a rapidly shifting clinical picture, and a short, self-limiting course with a characteristically **good outcome**. They are the nosological home of a family of historical syndromes, the French bouffee delirante among them, that European and non-European psychiatrists kept re-describing under different names because the phenomenon is real and recurrent (Kaplan & Sadock 2024). The exam wants three things: the **icd-11** category structure, the abrupt-onset short-course rule with its good prognosis, and the follow-up caveat that a minority re-diagnose over time.

This is a discrimination topic. Master the boundary against schizophrenia (duration and symptom stability) and against mood disorder (whether an affective episode is sustained), and hold the one management pointer: treat the episode, but *follow up*. The raw descriptive definitions of the delusions, hallucinations and perplexity referenced below sit in the Psychometry, Assessment and Descriptive Psychopathology notes; the disorder schizophrenia itself is owned by the Schizophrenia: aetiology, phenomenology, classification and course notes and is not re-taught here.

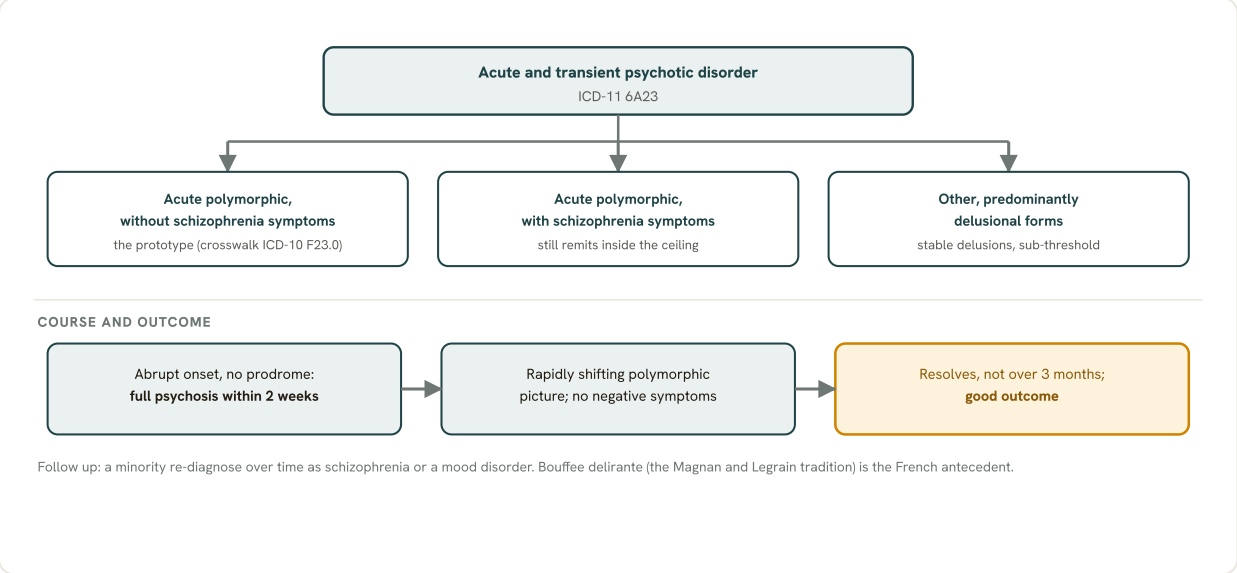


Fig 10.4 The ICD-11 acute and transient psychotic disorders, category and course. The single 6A23 category holds the acute polymorphic form (with or without symptoms of schizophrenia) and the other predominantly-delusional forms. The tempo defines it: abrupt onset to full psychosis within two weeks, a rapidly shifting polymorphic picture with no negative symptoms, and resolution within three months with a characteristically good outcome, though a minority re-diagnose on follow-up.

5.1 The icd-11 category and its essential features

One code, one gestalt. The **icd-11** collapses the older subtypes into a single category, acute and transient psychotic disorder (6A23), whose essential features are: acute onset of psychotic symptoms (delusions, hallucinations, disorganised thinking, or experiences of influence, passivity or control) *without a prodrome*, progressing from a non-psychotic to a clearly psychotic state within **2 weeks**; symptoms that *change rapidly* in nature and intensity, sometimes over the course of hours; *absence of negative symptoms*; and a duration that does not exceed **3 months**, most often lasting from a few days to 1 month (ICD-11 CDDR). Psychomotor disturbance including catatonia may be present. The picture must not be organic, substance-induced, or better explained by schizophrenia or another primary psychotic disorder.

RULE **Tempo defines it.** Acute onset (within 2 weeks) plus a rapidly shifting picture plus a short duration (not over 3 months) with a good outcome is **atpd**, but follow up because some become schizophrenia.

5.2 The acute polymorphic ("kaleidoscopic") picture

Symptoms that will not sit still. The prototype is the acute polymorphic psychotic disorder: an abruptly onset, rapidly shifting, "kaleidoscopic" picture of changing delusions, hallucinations and affect, with the type and intensity of symptoms fluctuating in relatively rapid succession, and marked emotional turmoil, perplexity or confusion superimposed (ICD-11 CDDR). The polymorphic form comes in two flavours, *with or without symptoms of schizophrenia*: if schizophrenia-type symptoms are present but the whole episode still remits inside the duration ceiling, it remains within this group rather than becoming schizophrenia. The other forms are the *other acute, predominantly-delusional* presentations, in which comparatively stable delusions or hallucinations dominate but do not meet the schizophrenia threshold. In the icd-11, this

fluctuating polymorphic quality is the *defining* feature of the single 6A23 category (ICD-11 CDDR).

TRAP **Do not fix schizophrenia on a first brief episode.** A first, brief, polymorphic episode with rapidly shifting content and full remission is **atpd**, not schizophrenia, even if the cross-section technically shows schizophrenia-type symptoms.

5.3 Course, good outcome and the follow-up imperative

Mostly remits, a minority declares itself later. The natural history is a rapid deterioration in social and occupational functioning, then full remission with return to the premorbid level. Favourable outcomes are associated with acute onset, short duration, good premorbid functioning and female gender (ICD-11 CDDR). A proportion of patients, however, re-diagnose over time as schizophrenia, another primary psychotic disorder, or a mood disorder, so the diagnosis is always provisional and follow-up is mandatory. Male gender and younger age of onset carry the greater risk of subsequent schizophrenia. Episodes are often stress-precipitated (an acute stressor is commonly reported but is not required), and the disorder is commoner in women and in developing countries (Kaplan & Sadock 2024).

WORKED EXAMPLE

A 24-year-old woman is brought in three days after a family bereavement: over hours her talk swings between grandiose religious certainty and terrified persecutory fear, voices come and go, and her affect lurches from ecstasy to dread. She is fully alert and oriented. Ten days later she is symptom-free. This is an acute polymorphic presentation of **atpd** (6A23.0, first episode): acute onset under 2 weeks, kaleidoscopic shifting picture, no negative symptoms, full remission. You still book follow-up, because a first episode does not exclude later schizophrenia or a mood disorder.

within 2 weeks	3 months	1 day to under 1 month
ONSET TO FULL PSYCHOSIS (ICD-11)	MAXIMUM DURATION; USUALLY DAYS TO 1 MONTH	DSM-5-TR BRIEF PSYCHOTIC DISORDER

5.4 Bouffee delirante and its nosological place

The French antecedent. Descriptions of acute-onset, remitting psychoses recur across traditions: Meynert's *amentia*, Kleist and Leonhard's cycloid psychosis, the Scandinavian *reactive (psychogenic) psychosis*, and the French bouffee delirant of the late 19th century (Kaplan & Sadock 2024). The nosological place of the bouffee delirante is precisely as the historical antecedent of the acute polymorphic category: an abrupt "delusional flash" out of a clear sky, richly polymorphic, with rapid full recovery, later distilled (with the cycloid and reactive concepts) into ICD-10's acute and transient category and then ICD-11's 6A23 (Kaplan & Sadock 2024). The classic attribution is to the French degeneration-school tradition of Magnan and his pupil Legrain (Kaplan & Sadock 2024 dates the joint concept to 1895), so name the tradition rather than betting on a single author or year.

PEARL

The recurring theme across all these historical labels is identical: acute onset, polymorphic content, rapid full recovery. That the same syndrome was independently described across cultural and linguistic boundaries is exactly why a unified acute and transient category was worth creating.

5.5 The icd-10 to icd-11 shift, and DSM-5

From subtypes to a single tempo-defined code. ICD-10 split the group into acute polymorphic psychotic disorder (with or without symptoms of schizophrenia) and an acute schizophrenia-like psychotic disorder, each further specified for the presence or absence of an associated acute stressor. The **icd-11** unified all of this into one category, 6A23, in which the rapidly fluctuating polymorphic picture is the essential feature; the official crosswalk maps 6A23 back to ICD-10 F23.0, acute polymorphic psychotic disorder without symptoms of schizophrenia (ICD-11 CDDR). Course is then coded by specifiers only: first episode (6A23.0) or multiple episodes (6A23.1), each further as currently symptomatic, in partial remission, or in full remission.

DSM-5 has no exact equivalent. The DSM-5-TR has no dedicated acute-and-transient section; its nearest match is brief psychotic disorder, defined by a duration of **at least 1 day but less than 1 month** with full return to premorbid functioning (Kaplan & Sadock 2024). Most patients meeting icd-11 **atpd** criteria map onto DSM-5-TR brief psychotic disorder, but a substantial minority fall instead into schizophreniform or other specified schizophrenia-spectrum categories, so the fit is inexact.

INDIA

The acute, benign-course psychoses that this category captures were reported with high frequency in India, Africa and the Caribbean, and the diagnosis is used heavily in Indian practice. Many psychiatrists, especially from developing countries such as India, drove recognition of the acute polymorphic picture as a distinct, good-prognosis entity rather than early schizophrenia.

FEATURE	ACUTE AND TRANSIENT (ICD-11 6A23)	SCHIZOPHRENIA	MOOD DISORDER WITH PSYCHOSIS
Onset	**Abrupt, no prodrome, within 2 weeks**	Often insidious, poor premorbid adjustment	Follows onset of the mood episode
Symptom stability	**Rapidly shifting, polymorphic, over hours to days**	Stable, fixed (same delusion for months)	Mood-congruent, tied to the episode
Negative symptoms	**Absent during the episode**	Often present	Not the defining feature
Duration	Does not exceed 3 months (usually days to 1 month)	At least 1 month florid; typically much longer	Mood episode persists days and usually much longer
Outcome	**Good; full remission to premorbid level**	Variable, often chronic	Episodic, defined by mood recovery

The three-way discrimination: tempo and symptom instability separate **atpd** from schizophrenia; a sustained affective episode separates it from a mood disorder. The mood-episode criteria themselves are in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes.

MNEMONIC

ABRUPT

Acute onset (within 2 weeks), **B**rief (not over 3 months), **R**apidly shifting polymorphic picture, **U**nremarkable premorbid state, **P**rognosis good, **T**rack them (follow up: some become schizophrenia or a mood disorder).

GOOD TO REMEMBER

- **Acute and transient psychotic disorder (icd-11 6A23):** abrupt-onset, short-course psychosis with a good outcome; discriminator is the rapidly shifting polymorphic picture with no negative symptoms; the number to hold is onset within 2 weeks, duration not over 3 months.
- **Acute polymorphic psychotic disorder:** the prototype and the icd-11 crosswalk to ICD-10 F23.0; discriminator is kaleidoscopic changing delusions, hallucinations and affect, with or without symptoms of schizophrenia; the point to hold is that it is the defining form of 6A23.
- **Bouffee delirante:** the French late-19th-century acute polymorphic psychosis, a "delusional flash" with rapid recovery; discriminator is its historical role as the antecedent of the acute polymorphic category; eponym to hold is the Magnan and Legrain tradition (Kaplan & Sadock 2024).
- **Cycloid psychosis:** Kleist and Leonhard's acute-onset, good-prognosis, frequently-recurring psychosis; discriminator is the same acute-remitting theme in a different tradition; hold Leonhard as the name attached to the concept.
- **DSM-5-TR brief psychotic disorder:** the nearest DSM match, with no dedicated acute-and-transient section; discriminator is the duration window; the number to hold is at least 1 day but less than 1 month with full remission.

HOLD THIS

Acute onset within 2 weeks, a rapidly shifting polymorphic picture with no negative symptoms, and full remission within 3 months is acute and transient psychotic disorder (icd-11 6A23), good outcome expected, but follow up because a minority declare as schizophrenia or a mood disorder.

6 · Brief psychotic disorder

The **brief psychotic disorder** of DSM-5-TR is the shortest-lived of the schizophrenia-spectrum entities: a storm of positive psychotic symptoms that erupts suddenly, burns out inside a month, and leaves the person back at their premorbid baseline. It is the diagnosis you reach for when a previously well patient presents floridly delusional or hallucinated after, say, a bereavement or a delivery, and then clears rapidly. For the exam it is a duration problem first and a differential problem second: get the **less than a month** boundary right, name the specifiers, and know that the good prognosis is real but conditional on follow-up.

Why it earns a place. It anchors the short end of the psychosis duration ladder (brief psychotic, then schizophreniform, then schizophrenia), it carries the historical concepts examiners love (brief reactive psychosis, bouffee delirante), and it forces you to weigh a benign transient episode against the first presentation of a lifelong illness. The raw descriptive definitions of the delusions and hallucinations themselves are cross-referenced to the Psychometry, Assessment and

Descriptive Psychopathology notes; the disorder schizophrenia itself is owned by the Schizophrenia: aetiology, phenomenology, classification and course notes and is not re-taught here.

6.1 The DSM-5-TR entity: sudden onset, days to weeks, full recovery

Core criterion. Brief psychotic disorder (DSM-5-TR, coded **F23**, page 108) requires the sudden onset of at least one positive psychotic symptom, and one of them must be delusions, hallucinations or disorganised speech (grossly disorganised or catatonic behaviour alone does not qualify). The episode lasts **at least a day but less than a month**, with an eventual full return to the premorbid level of functioning (APA 2022; Kaplan & Sadock 2024). Tasman frames it crisply as a transient psychotic state, not caused by a medical condition or a substance, lasting at least one day and up to the one month upper bound (Tasman 2015).

The bracketing. The one month upper bound is the hinge. Cross the one month line and, if criterion A for schizophrenia is met, the diagnosis becomes schizophreniform disorder (at least one month but less than six months); cross six months and it becomes schizophrenia. The whole point of the entity is that the psychosis is over inside a month and the person is restored.

RULE **Under a month, full recovery.** Brief psychotic disorder resolves within one month with a full return to premorbid functioning; if it runs past a month it is no longer brief psychotic disorder.

HOLD THIS

Brief psychotic disorder = sudden positive psychosis lasting at least a day but less than a month, with full return to premorbid functioning; the post-stressor form is the old brief reactive psychosis.

6.2 The three specifiers: stressor, post-stressor, peripartum

The stressor axis. DSM-5-TR tags the episode *with marked stressor(s)* or *without marked stressor(s)*. The **post-stressor** form, coded when florid psychosis follows an event that would be markedly stressful to almost anyone in that culture, is the modern home of the historical brief reactive psychosis of DSM-III and DSM-III-R, whose criteria explicitly required a severe precipitant such as a major loss or a combat-type trauma (Kaplan & Sadock 2024). The without-stressor form has no identifiable precipitant.

The peripartum axis. The third specifier is *with peripartum onset*, applied when onset is during pregnancy or within four weeks postpartum. This is the DSM-5-TR route by which a postpartum psychotic episode that clears inside a month is captured; the wider perinatal frame for postpartum psychosis as a psychiatric emergency is developed in the Perinatal/women's mental health and emergency psychiatry notes.

SPECIFIER	WHEN APPLIED	HISTORICAL / LINKED CONCEPT
With marked stressor(s)	**Onset shortly after an event markedly stressful to almost anyone**	Old brief reactive psychosis (DSM-III/III-R); the post-stressor form
Without marked stressor(s)	No identifiable precipitant	Distinguishes reactive from non-reactive episodes
With peripartum onset	**Onset in pregnancy or within four weeks postpartum**	Captures brief postpartum psychotic episodes

The three DSM-5-TR specifiers for brief psychotic disorder; the marked-stressor form is the direct descendant of brief reactive psychosis.

6.3 The ICD-11 near-equivalent: acute and transient psychotic disorder

The counterpart. The ICD-11 acute and transient psychotic disorder (ATPD) is the near-equivalent of brief psychotic disorder: an acute onset of psychotic symptoms that fluctuate rapidly, characteristically with a polymorphic, shifting picture, resolving within a short period. Kaplan & Sadock explicitly link the two, noting that both overlap with the older concept of cycloid psychosis (Kaplan & Sadock 2024). ATPD in turn absorbs the historical European syndromes: the French bouffee delirante and the Scandinavian reactive psychosis tradition (Shorter Oxford 2018).

Where they diverge. The ICD lineage is more elaborate than the single DSM criteria set. ICD-10 split acute and transient psychotic disorders into several types by symptom picture and course, with the maximum duration varying by type: roughly one month for schizophrenia-like symptoms and up to three months for the predominantly delusional form (Tasman 2015). DSM-5, by contrast, uses one criteria set and a single maximum of one month. When mapping in an exam, note that ICD-11 places catatonia transdiagnostically as a separate cross-cutting entity rather than tucking it inside these categories.

TRAP **Do not swap the durations.** Brief psychotic disorder tops out at one month; the predominantly delusional acute and transient psychotic disorder in the ICD lineage may run longer, and schizophreniform runs one to six months. Mismatching these loses the mark.

6.4 Differential from early schizophrenia, and why follow-up is the whole game

The problem of the first episode. The single hardest discrimination is between a true brief psychotic disorder and the opening act of schizophrenia (or a mood disorder with psychotic features). At presentation they can look identical; only duration and outcome separate them. This is why the diagnosis is provisional in spirit: it is confirmed only in retrospect, once the episode has fully resolved inside a month and the person has been observed to hold their recovery.

Diagnostic instability. The prognosis is genuinely good, but the label is unstable on follow-up: in one clinical series only **23%** of patients given a discharge diagnosis of brief psychotic disorder still held that diagnosis a year later, a proportion having declared a chronic psychotic or mood illness on longer observation (Kaplan & Sadock 2024). The New Oxford Textbook makes the same operational point: individuals who have shown a brief psychotic episode must be monitored to assess the stability of function and the possible emergence of further brief or

chronic disorders (New Oxford 2020). Disorder-specific management is short-course antipsychotic cover for the acute episode plus structured follow-up; the antipsychotic drug detail is cross-referenced to the Schizophrenia: management, antipsychotics and adverse effects notes.

TRAP **Not following up.** Treating a resolved brief psychotic episode as closed and discharging without review misses the substantial minority who convert to a chronic psychotic or mood illness; the good prognosis is contingent on surveillance.

under 1 mo	1 to 6 mo	23%
BRIEF PSYCHOTIC DISORDER DURATION	SCHIZOPHRENIFORM DISORDER	RETAINED THE DIAGNOSIS AT 1 YEAR (REST RECLASSIFIED)

WORKED EXAMPLE

A 26-year-old woman, previously well, becomes floridly paranoid and hears accusatory voices ten days after her father's sudden death. She is perplexed, sleepless and frightened; there is no drug use and the physical workup is clean. She is given brief antipsychotic cover and settles completely over three weeks, back to her baseline job and relationships. The provisional formulation is brief psychotic disorder, with marked stressor(s), the post-stressor form. Because a minority declare schizophrenia or a mood illness later, she is not discharged outright but reviewed; if a fresh episode or persistent symptoms emerge, the diagnosis is revised.

INDIA

Acute, short-lived, remitting psychoses after a stressor are a familiar Indian clinical picture and map onto the ICD acute and transient psychotic disorder category used in routine practice; the classic non-affective acute remitting psychosis studied in Indian and other low-and-middle-income-country cohorts sits close to this construct. Treat the episode, then follow up, since a proportion evolve into a chronic illness.

PEARL

Think of brief psychotic disorder as the psychosis that ends before schizophreniform can even begin: same florid positive symptoms, but the clock stops before one month and the person walks out whole. The diagnosis is a promise the follow-up must keep.

GOOD TO REMEMBER

- **Brief psychotic disorder:** sudden onset of one or more positive psychotic symptoms lasting at least a day but less than a month, with full return to premorbid functioning; hold the number: DSM-5-TR F23, one month upper bound.
- **Post-stressor form (with marked stressor(s)):** the psychosis follows an event stressful to almost anyone; the one discriminator is the marked precipitant; the eponymic history is brief reactive psychosis (DSM-III/III-R).
- **With peripartum onset:** onset in pregnancy or within four weeks postpartum; the one number to hold is the four-week postpartum window.
- **Acute and transient psychotic disorder (ICD-11):** the near-equivalent, acute polymorphic rapidly-shifting psychosis; the discriminating feature is the fluctuating polymorphic picture; it subsumes bouffee delirante and cycloid psychosis.
- **Follow-up rule:** good prognosis but diagnostically unstable; the one number is 23% retaining the diagnosis at one year in one series, the rest reclassified to a chronic psychotic or mood illness.

7 · Induced and shared delusional disorder (folie a deux)

A delusion is usually the most private of symptoms, arising and staying inside one mind. The exam-relevant curiosity here is a delusion that **crosses between two people**: a genuinely psychotic, dominant primary transmits a belief to a passive, suggestible secondary who comes to hold it as their own. The entity has collected many names across a century and a half of nosology, and the examiner's favourite hook is precisely that thicket of eponyms and the one management principle that follows from the mechanism.

Why it matters. This is a small, high-yield topic where marks are won on three things: naming the entity correctly across ICD and DSM, attributing the classical folie a deux eponyms without swapping them, and stating the treatment rule (separate the pair) with its diagnostic corollary. The raw descriptive account of delusion as a symptom belongs to the Psychometry, Assessment and Descriptive Psychopathology notes; here the task is the disorder and its discrimination.

7.1 The entity and its many names

One belief, two carriers. The core phenomenon is the transfer of a delusion from a primary, dominant, genuinely psychotic person to a passive, suggestible, usually closely bound and socially isolated secondary who comes to share it (Tasman). Across editions and traditions the same entity is called the **induced delusional** disorder (ICD-10 term), the **shared psychotic** disorder (DSM-IV term), the **shared delusional** disorder, and, in the classical French, the folie a deux (Kaplan & Sadock 2024). Older synonyms include shared paranoid disorder, induced psychosis and double insanity (Kaplan & Sadock 2024).

RULE **Two people, one genuine psychosis.** Only the dominant primary has an authentic, independently generated delusion; the secondary's belief is borrowed and rests on the relationship.

7.2 Nosological placement: ICD-11, DSM-5-TR, ICD-10

Where the codebooks put it. The nosology has drifted, and knowing the current placement is the discriminator. In **ICD-11** the condition no longer has a standalone code: it is folded into

delusional disorder (6A24) as an additional clinical feature, described as "shared or induced delusional disorder", in which one person adopts the delusional belief of another where the two have a strong emotional or situational link (ICD-11 CDDR). In **DSM-5-TR** the discrete category was deleted; the presentation is captured as "delusional symptoms in partner of individual with delusional disorder", coded under other specified schizophrenia spectrum and other psychotic disorder (Kaplan & Sadock 2024). **ICD-10** retained a separate rubric, F24 induced delusional disorder (Shorter Oxford). The wider diagnostic frame for delusional disorder itself is cross-referenced to that topic; the raw phenomenology of the shared delusion is in the Psychometry, Assessment and Descriptive Psychopathology notes.

TRAP **Do not call it a DSM-5 disorder.** DSM-5 and DSM-5-TR deleted the standalone category; ICD-11 also demoted it to a feature of delusional disorder. Only ICD-10 keeps it as a named F24 diagnosis.

7.3 The primary, the secondary and the relationship

An asymmetric pair. The primary (inducer) is typically the older, more intelligent, better educated, dominant partner who carries a genuine psychotic illness, most often schizophrenia or delusional disorder (Tasman). The secondary (induced) is usually more suggestible, more dependent, often less intelligent, more passive or more lacking in self-esteem, and frequently predisposed by personality traits or social circumstance (Tasman). The two are closely associated over a long period, live together, and are relatively or completely isolated from outside social and cultural correction, which removes the reality-testing a wider circle would supply (Tasman). Almost all cases occur within a single family, the commonest dyads being sister-sister, husband-wife and mother-child (Tasman). The content is usually persecutory or hypochondriacal and, being borrowed, tends to stay within the realm of possibility rather than becoming as bizarre as delusions in schizophrenia (Tasman).

7.4 The classical variants

Four named forms, one entity. The historical literature subdivides the syndrome by how the shared belief arose. Hold the two the sources anchor firmly, and recognise the full quartet: **folie imposee**, **folie communiquee**, **folie induite** and **folie simultanee**.

folie imposee

The common, prototypical form: the dominant primary imposes the delusion on a healthy, suggestible secondary, whose belief typically fades on separation (Tasman).

folie communiquee

The delusion is communicated to the secondary after a period of resistance, and the secondary then holds it independently, so that it may persist even after separation (Baillarger's original 1860 description, Tasman).

folie induite

An already-psychotic secondary adds new delusions induced by the dominant partner to their own pre-existing ones.

folie simultanee

Similar delusional systems develop independently and at the same time in two closely associated, often already predisposed people, rather than one being transferred from the other (Tasman).

When the shared system extends beyond a dyad the count is named directly: folie a trois, folie a quatre, folie a cinq, and folie a famille when a whole family is involved; these are rare (Tasman).

MNEMONIC

I C I S: Imposed, Communicated, Induced, Simultaneous

The four classical variants in one string. IMPOSED = healthy secondary, resolves on separation (the exam default); COMMUNICATED = accepted after resistance, may persist; INDUCED = added onto an already psychotic secondary; SIMULTANEOUS = two predisposed people, no true transfer.

7.5 Management: separation, then treat the primary

The pivot is the pair. The single therapeutic and diagnostic manoeuvre is **separation** of the pair. On separating the two, the secondary's delusion frequently resolves, and this resolution both treats the secondary and retrospectively confirms the diagnosis (ICD-11 CDDR; Tasman). The primary does not remit on separation and needs treatment in the usual way for their underlying psychotic illness, the antipsychotic detail of which is cross-referenced to the Schizophrenia: management, antipsychotics and adverse effects notes. Resolution in the secondary on separation is frequent but not invariable, and is least reliable in the communiquee and induite forms where the belief has become independently rooted (Tasman). Supportive work with the secondary during separation, aimed at rebuilding self-esteem and reality contact, supports the recovery.

RULE **Separation is both diagnostic and therapeutic for the secondary.** The delusion that lifts when the pair is parted was never independently generated; the manoeuvre proves and treats the induced case in one move.

TRAP **Do not treat both members as independently and equally psychotic.** Loading the passive secondary with the same long-term antipsychotic plan as the primary misreads a borrowed belief as an autonomous psychosis; separate first, then treat the primary in the usual way.

HOLD THIS

Separate the pair: the secondary's borrowed delusion usually lifts on separation (which both diagnoses and treats them), while the dominant, genuinely psychotic primary is treated in the usual way and does not remit on separation.

INDIA

In Indian family psychiatry the closely bound, co-resident, relatively isolated multigenerational household is exactly the substrate in which induced delusional disorder is described, and the commonest reported dyads, mother-child and husband-wife, map onto that structure. Separation is harder to arrange where the pair share a single home and depend on each other economically, so admission of the secondary, or of the primary, is often the practical route to the therapeutic separation.

PEARL

The bizarreness of the belief is a quiet clue: an induced delusion tends to stay within the realm of the possible (a persecution, an infidelity, a hypochondriacal fear), because a suggestible mind borrows a plausible story more readily than a bizarre one. A frankly bizarre, incoherent delusion in the secondary should push you back toward an independent primary psychosis rather than a shared one.

FEATURE	PRIMARY (INDUCER)	SECONDARY (INDUCED)
Delusion origin	**Authentic, independently generated**	**Borrowed from the primary**
Underlying illness	Genuine psychosis, often schizophrenia or delusional disorder	Usually none; often personality vulnerability
Role in the dyad	Dominant, older, more able	Passive, suggestible, dependent
Effect of separation	Delusion persists; needs treatment	**Delusion frequently resolves**
Management	Treat the underlying psychosis in the usual way	Separate first; support; medicate only if it fails to resolve

The asymmetry of the pair drives the whole management: the discriminator is what happens on separation.

GOOD TO REMEMBER

- **Folie a deux / shared psychotic disorder / induced delusional disorder:** transfer of a delusion from a dominant, genuinely psychotic primary to a passive, suggestible, closely bound and socially isolated secondary. Discriminator: only the primary has an authentic psychosis. Number/eponym to hold: Lasegue and Falret, 1877.
- **folie communiquee:** the delusion is communicated to the secondary after resistance and then held independently, so it may persist after separation. Eponym: Jules Baillarger, 1860.
- **folie imposee:** the common form, a healthy secondary onto whom the belief is imposed; resolves on separation. Hold: this is the exam default variant.
- **folie simultanee:** similar delusions arise independently and simultaneously in two predisposed, closely associated people; no true transfer. Hold: not a genuine induction.
- **folie induite:** an already-psychotic secondary acquires additional delusions induced by the dominant partner. Hold: the secondary is not delusion-free at baseline.
- **Nosology:** ICD-10 F24 induced delusional disorder (standalone); ICD-11 a feature of delusional disorder (no separate code); DSM-5-TR deleted, coded as delusional symptoms in partner of individual with delusional disorder. Hold: only ICD-10 keeps it as a named diagnosis.
- **Management:** separation of the pair is both diagnostic and therapeutic for the secondary; treat the primary's psychosis in the usual way. Hold: the secondary's delusion frequently, though not invariably, resolves on separation.

8 · Postpartum psychosis and the late-onset psychoses

Two psychotic presentations are bound to a life stage rather than to a diagnosis. **Postpartum psychosis** erupts in the days after a delivery and is one of the true psychiatric emergencies of the discipline; the **late-onset** and **very-late-onset** schizophrenia-like psychoses declare themselves in middle and old age, carry the historical burden of paraphrenia, and hide a neurodegenerative or organic cause behind an apparently functional picture. For the exam they are a pair of high-yield edge cases at either end of adult life, each with its own numbers, its own one discriminator, and its own classic trap.

Why they earn a place. Postpartum psychosis is where a psychotic-disorders answer meets emergency psychiatry: get the two-week onset window, the bipolar link, and the protect-mother-and-baby imperative right. The late-onset psychoses are where a psychosis answer meets old-age psychiatry: get the international-consensus split at 40 and 60, the paraphrenia lineage, and the duty to exclude a dementing or organic process. The raw descriptive definitions of the delusions themselves are cross-referenced to the Psychometry, Assessment and Descriptive Psychopathology notes; the disorder schizophrenia itself is owned by the Schizophrenia: aetiology, phenomenology, classification and course notes and is not re-taught here.

8.1 Postpartum psychosis: the acute, delirium-like, often affective psychosis of the early puerperium

The entity. Postpartum psychosis, historically puerperal psychosis, is a new onset of a severe psychotic episode in the immediate puerperium. In around **90%** of cases the onset is within the first **two weeks** of delivery (New Oxford 2020). The picture is characteristically affective, most often a manic or mixed episode with psychotic features, and it is strongly associated with bipolar

I disorder (APA 2022; Kaplan & Sadock 2024). Kaplan & Sadock treat postpartum psychosis as, in effect, a subtype of bipolar disorder.

The delirium-like quality. What sets it apart from an ordinary manic relapse is its fluctuating, delirium-like texture: confusion, perplexity and a rapidly shifting, kaleidoscopic symptom picture that can escalate within hours (New Oxford 2020). The Companion notes that perplexity is seen par excellence in puerperal psychosis (Companion 2010). This is why the classic bedside advice is to exclude an organic cause and treat the presentation as an emergency, not to assume a benign course. The raw phenomenology of the perplexity and the delusions is cross-referenced to the Psychometry, Assessment and Descriptive Psychopathology notes.

RULE **Postpartum psychosis is a psychiatric emergency: protect mother and baby, and admit.** Because of the risk of suicide and of infanticide, urgent assessment and, in the majority of cases, admission (ideally to a mother-and-baby unit) are the default.

HOLD THIS

Postpartum psychosis is an acute, fluctuating, delirium-like, usually bipolar-spectrum psychosis with onset within the first two weeks in 90% of cases; it is a psychiatric emergency because of suicide and infanticide risk, so admit and safeguard the infant.

8.2 Why it is a psychiatric emergency: suicide and infanticide

The danger. Postpartum psychosis is a **psychiatric emergency** because it carries an increased risk of both suicide and infanticide (New Oxford 2020). Suicide is a leading cause of maternal death in the perinatal period, and infanticide is more commonly associated with postpartum psychosis than with postpartum depression (Kaplan & Sadock 2024). Roughly **half** of episodes are the woman's first contact with psychiatric services and so could not have been predicted, which is exactly why prompt assessment matters (New Oxford 2020). The wider perinatal frame (maternity blues, postnatal depression, the perinatal service model) is developed in the Perinatal/women's mental health and emergency psychiatry notes; postpartum psychosis is owned here as a psychotic disorder.

TRAP **Treating it as postnatal blues.** Reassuring a confused, perplexed, psychotic new mother and sending her home misreads a psychiatric emergency; the suicide and infanticide risk demands urgent assessment and usually admission.

8.3 Risk factors: bipolar disorder, a previous postpartum psychosis, primiparity, sleep loss

The high-risk profile. The dominant risk factor is a personal history of a **bipolar disorder**: a woman with bipolar disorder has about a **1 in 5** chance of postpartum psychosis with each delivery, and a previous episode of postpartum psychosis confers a risk in excess of **1 in 2** for the next (New Oxford 2020). A family history of postpartum psychosis roughly doubles the risk in women who already have bipolar disorder. **Primiparity** is the most robust obstetric risk factor identified, and sleep loss around the delivery is a recognised precipitant (Sharma 2003; New Oxford 2020). The risk of a manic episode in the first four weeks postpartum is dramatically raised (New Oxford 2020).

within 2 wk	1 in 5	over 1 in 2	1 to 2 / 1000
ONSET IN 90% OF POSTPARTUM PSYCHOSIS	RISK PER DELIVERY WITH BIPOLAR DISORDER	RISK AFTER A PREVIOUS POSTPARTUM PSYCHOSIS	INCIDENCE PER DELIVERIES

8.4 Management principles: urgent assessment, admission, and safeguarding the infant

The disorder-specific pointers. Management rests on urgent assessment and, in the majority, admission, ideally to a mother-and-baby unit so that mother and infant are kept together and the baby is safeguarded (New Oxford 2020). Pharmacological treatment is the mainstay: antipsychotics and mood stabilisers, with a stepped approach of a benzodiazepine, then a dopamine antagonist or partial agonist, then lithium, achieving remission in the large majority; lithium outperforms an antipsychotic alone for maintenance (New Oxford 2020). Electroconvulsive therapy is used, and can be strikingly rapid, in severe, treatment-resistant or life-threatening episodes; the ECT technique itself is cross-referenced to the ECT and neurostimulation notes, and the antipsychotic and mood-stabiliser drug detail to the Schizophrenia: management, antipsychotics and adverse effects notes. Safeguarding the infant runs in parallel with treating the mother.

WORKED EXAMPLE

A 28-year-old primiparous woman with a prior diagnosis of bipolar I disorder becomes elated, overactive and sleepless four days after delivery, then over 48 hours becomes perplexed, disoriented and convinced the baby has been swapped. The picture fluctuates hour to hour. She is treated as a psychiatric emergency: urgent assessment, exclusion of an organic cause, admission to a mother-and-baby unit with the infant safeguarded, and antipsychotic plus mood-stabiliser cover, with ECT held in reserve for a severe non-response. This is postpartum psychosis on a bipolar substrate, not the maternity blues.

INDIA

Acute, fluctuating postpartum psychoses are a familiar Indian clinical picture and are commonly managed on general psychiatry wards, since dedicated mother-and-baby units are scarce in Indian practice; keeping the mother and infant together and safeguarding the baby therefore has to be arranged within general adult inpatient care. ECT is available and used in Indian psychiatry for severe or treatment-resistant postpartum psychosis where a rapid response is needed.

GOOD TO REMEMBER

- **Postpartum psychosis (puerperal psychosis):** an acute, fluctuating, delirium-like, usually affective (bipolar-spectrum) psychosis of the early puerperium; the one discriminator is the confused, perplexed, rapidly shifting picture; hold the number, onset within two weeks in 90% of cases.
- **Emergency status:** a psychiatric emergency because of suicide and infanticide risk; the one imperative is admit and safeguard the infant; hold that infanticide is more linked to postpartum psychosis than to postpartum depression.
- **Risk factors:** bipolar disorder, a previous postpartum psychosis, primiparity, sleep loss; the biggest single number is a 1 in 5 risk per delivery with bipolar disorder, rising above 1 in 2 after a prior postpartum psychosis.

8.5 The late-onset psychoses: the international-consensus split at 40 and 60

The nosology. An international consensus (Howard, Rabins, Seeman & Jeste 2000) recommended that schizophrenia with onset between ages **40 and 60** be termed late-onset schizophrenia, and psychosis with onset after age **60** be termed very-late-onset schizophrenia-like psychosis, that is, a very late onset variant (Kaplan & Sadock 2024). The **late-onset schizophrenia-like** label thus spans the middle-aged and elderly cases that older classifications handled poorly. The very-late-onset group is the modern home of the old concept of paraphrenia, Kraepelin's term for a paranoid psychosis of middle and later life that was separated from dementia praecox because it lacked the emotional and volitional deterioration, later revived by Roth as late paraphrenia (Shorter Oxford 2018; Companion 2010). ICD-11 keeps a single schizophrenia category rather than a separate age-defined disorder; note that ICD-11 additionally places catatonia transdiagnostically as a cross-cutting entity, a high-yield nosology point developed with the catatonia material.

Epidemiology and the female preponderance. Late-onset schizophrenia is commoner in women, who are affected between **2 and 10** times more often than men, a robust finding often linked to the loss of estrogen-mediated dopaminergic protection at menopause (Kaplan & Sadock 2024). The International Late-Onset Schizophrenia Group judged late-onset schizophrenia to be broadly a neurodevelopmental disorder differing from early-onset disease more in degree than in kind, whereas very-late-onset schizophrenia-like psychosis showed features (more brain abnormalities and neuropsychological deficits) suggesting a neurodegenerative component (Kaplan & Sadock 2024).

RULE **Split by the two ages.** Onset 40 to 60 is late-onset schizophrenia; onset after 60 is very-late-onset schizophrenia-like psychosis (the old paraphrenia); both are commoner in women.

8.6 The clinical picture: persecutory and partition delusions, preserved affect and cognition

The phenomenology. The late-onset picture is dominated by prominent, well-organised **persecutory** delusions and by partition delusions, the conviction that people, objects or radiation can pass through impermeable structures such as walls, ceilings, doors or windows (Kaplan & Sadock 2024). Hallucinations are common and may be multimodal (auditory, visual, tactile, olfactory). By contrast, formal thought disorder and negative symptoms are infrequent, and affect and cognition are relatively preserved, which is precisely why some doubt whether very-late-onset psychosis belongs under schizophrenia at all (Kaplan & Sadock 2024; Tasman 2015). The raw descriptive definition of the partition delusion is cross-referenced to the Psychometry, Assessment and Descriptive Psychopathology notes.

The predisposing background. The late-onset psychoses are classically associated with **sensory impairment** (especially deafness and visual loss), social isolation, and premorbid schizoid or paranoid personality traits; marriage and fertility rates are lower and social isolation greater than in the general population (Kaplan & Sadock 2024). Cognitive dysfunction, where present, affects frontal-subcortical domains, and, importantly, retention of information is usually preserved, in

contrast to the retention (storage) deficit of a cortical dementia like Alzheimer's disease (Kaplan & Sadock 2024).

FEATURE	LATE-ONSET SCHIZOPHRENIA (40 TO 60)	VERY-LATE-ONSET SCHIZOPHRENIA-LIKE PSYCHOSIS (AFTER 60)
Historical label	Late-onset schizophrenia	**Paraphrenia / late paraphrenia**
Sex	Female preponderance	**Female preponderance, more marked**
Delusions	Prominent persecutory	**Persecutory plus partition delusions**
Thought disorder / negative symptoms	Relatively infrequent	**Infrequent (fuels doubt about the diagnosis)**
Affect and cognition	Relatively preserved	Preserved, but organic cause must be excluded
Postulated mechanism	Largely neurodevelopmental (degree, not kind)	**Possible neurodegenerative component**

The international-consensus split: late-onset schizophrenia versus the very-late-onset schizophrenia-like psychosis that inherits the paraphrenia concept.

8.7 The imperative: exclude a neurodegenerative or organic cause

The discriminator that matters most. Late-life psychotic symptoms can arise de novo or be driven by dementia, delirium, medical illness, polypharmacy, substance misuse or sensory deficit; secondary psychosis (attributable to another medical or neurological disorder) accounts for a large share of late-life cases (Kaplan & Sadock 2024). A very-late-onset presentation in particular must be worked up as potentially the herald of a dementing process: a proportion convert to dementia on follow-up, so a confident diagnosis of very-late-onset schizophrenia-like psychosis can be made only after a sufficient observation period (Kaplan & Sadock 2024). Disorder-specific treatment is low-dose antipsychotic cover (the starting dose is a fraction of the usual adult dose, with careful attention to older-age adverse effects), plus correcting sensory deficits and social isolation; the antipsychotic drug detail is cross-referenced to the Schizophrenia: management, antipsychotics and adverse effects notes.

TRAP **Missing an emerging dementia.** Labelling a first psychosis after 60 as very-late-onset schizophrenia-like psychosis without a neurocognitive workup and follow-up misses the organic or neurodegenerative cause hiding behind it.

PEARL

Preserved retention is the tell: in very-late-onset psychosis the patient can still lay down and hold new memories, so a rapidly failing memory should push you towards a dementia, not towards paraphrenia. Deafness, isolation and a suspicious premorbid personality complete the classic late-onset triad.

GOOD TO REMEMBER

- **Late-onset schizophrenia:** schizophrenia with onset between 40 and 60; the discriminator is the age band with prominent persecutory delusions and relatively preserved affect and cognition; hold the international-consensus split at 40 (Howard et al. 2000).
- **Very-late-onset schizophrenia-like psychosis:** psychosis with onset after 60, the old paraphrenia; the discriminator is partition delusions with a possible neurodegenerative component; hold the number, onset after 60.
- **Paraphrenia:** Kraepelin's paranoid psychosis of later life spared the emotional and volitional decline of dementia praecox, revived by Roth as late paraphrenia; the one feature is well-organised persecutory delusions with preserved personality.
- **Partition delusion:** the belief that people, objects or radiation pass through walls, doors or windows; the discriminating context is very-late-onset schizophrenia-like psychosis; the one association is late-life onset.
- **Associations:** female sex (2 to 10 times commoner), sensory impairment, social isolation, paranoid or schizoid premorbid traits; the imperative is to exclude a neurodegenerative or organic cause.

9 · Attenuated psychosis syndrome

The **attenuated psychosis syndrome** is DSM-5-TR's attempt to give the clinical high-risk state a name inside the manual: sub-threshold positive symptoms, present often enough and distressing enough to bring a young person to attention, but with reality testing still relatively intact and the person still able to doubt the experience. It is not a full psychotic disorder and, importantly, it is not a formal diagnosis: it sits in Section III among the conditions for further study. For the exam it is a small, high-yield item precisely because it is the point where the **at-risk mental state** becomes a candidate category, and where the false-positive problem is the reason it stops short of the main manual.

Why it earns a place. It formalises the prodrome or at-risk mental state that the schizophrenia course chapter describes, and it forces the discrimination examiners want: an at-risk state named for study is not established schizophrenia. The prodrome and the wider at-risk-mental-state material, along with the schizophrenia course it belongs to, are owned by the Schizophrenia: aetiology, phenomenology, classification and course notes and are not re-derived here; this section is deliberately short.

9.1 The DSM-5-TR condition for further study: attenuated, sub-threshold, insight relatively intact

Core definition. Attenuated psychosis syndrome is a DSM-5-TR **condition for further study** (Section III), characterised by **attenuated psychosis**: delusion-like ideas, unusual perceptual experiences or disorganised speech that are psychosis-like but below the threshold of a full psychotic symptom, with reality testing still relatively intact and the person generally still able to appreciate that the perceptions are altered (APA 2022). The symptoms are less severe and more transient than in a psychotic disorder, and skepticism about them can be induced. In effect it is the at-risk mental state formalised into a candidate category (Kaplan & Sadock 2024).

The operational criteria. Criterion A requires at least one of three attenuated symptoms in relatively intact reality testing: attenuated delusions, attenuated hallucinations, or attenuated disorganized speech. The symptom must have been present **at least once per week for the past**

month (Criterion B) and must have **begun or worsened in the past year** (Criterion C). It must be sufficiently distressing or disabling to warrant clinical attention (Criterion D), not better explained by another mental disorder or a substance or medical cause (Criterion E), and the criteria for any psychotic disorder must never have been met (Criterion F) (APA 2022).

RULE **It names the at-risk mental state, but it is not a formal diagnosis.** Attenuated psychosis syndrome is a DSM-5-TR condition for further study, a candidate label for the clinical high-risk state, not an established Section II diagnosis.

HOLD THIS

Attenuated psychosis syndrome = sub-threshold delusion-like ideas, unusual perceptions or disorganised speech with reality testing relatively intact, at least once a week for the past month and worsening in the past year, distressing enough to warrant clinical attention; it is the at-risk mental state as a DSM-5-TR condition for further study, not a formal diagnosis.

9.2 Why it stays "for further study": only a minority transition, so false-positive and stigma risks dominate

The transition problem. The syndrome remains a condition for further study rather than a formal diagnosis because only a minority of those identified go on to a full psychotic disorder. Modern meta-analytic clinical-high-risk data put transition to a full psychotic disorder at roughly **11% at 6 months**, **15% at 1 year** and **20% at 2 years**, with the older ultra-high-risk cohorts reporting higher rates of around a third at one year; across all of them most people meeting criteria never convert, the non-converting group generally improving in symptoms over time (Kaplan & Sadock 2024, citing Fusar-Poli et al.). The at-risk mental state is detected with structured measures: the Comprehensive Assessment of At-Risk Mental State (CAARMS) and the Structured Interview for Psychosis-Risk Syndromes (SIPS) (New Oxford 2020; Kaplan & Sadock 2024).

The cost of a false positive. Because most identified individuals will not become psychotic, formalising the label risks over-diagnosis: labelling large numbers of help-seeking adolescents as pre-psychotic, exposing them to stigma and to potentially unnecessary antipsychotic treatment. That false-positive and stigma calculus is exactly why DSM-5-TR held the syndrome back in Section III for study rather than promoting it to a full diagnosis. Management is therefore watchful clinical attention and monitoring, not routine antipsychotic prescription; where drug treatment is considered, the antipsychotic detail is cross-referenced to the Schizophrenia: management, antipsychotics and adverse effects notes.

TRAP **Labelling an at-risk state as established schizophrenia.** Calling a person with attenuated, sub-threshold symptoms "schizophrenic" mislabels a candidate at-risk state as a lifelong illness that most of them will never develop.

~15% at 1 yr	Section III	most
TRANSITION TO PSYCHOSIS (ABOUT 20% AT 2 YEARS)	DSM-5-TR CONDITION FOR FURTHER STUDY, NOT A FORMAL DIAGNOSIS	DO NOT CONVERT; NON-CONVERTERS GENERALLY IMPROVE

WORKED EXAMPLE

A distressed 17-year-old is brought in with a few months of intermittent suspiciousness that people are talking about him, fleeting shadow-like visual experiences, and speech that his teachers find vague and off-track. He can, when gently challenged, entertain that there might be another explanation and does not act on the ideas. Symptoms are present several times a week, began this year, and are impairing his schooling. Reality testing is relatively intact and full psychotic-disorder criteria have never been met. This fits attenuated psychosis syndrome as a condition for further study: he is monitored and supported, not diagnosed with schizophrenia and not started on an antipsychotic by reflex.

INDIA

Dedicated clinical-high-risk and early-intervention services are still uncommon in routine Indian practice, so young people with attenuated, sub-threshold symptoms are usually seen once frankly psychotic rather than in a formal at-risk clinic. The practical lesson is the same as the syndrome's own caution: hold the at-risk label lightly, monitor over time, and avoid committing an adolescent to a schizophrenia diagnosis, and to long-term antipsychotics, on sub-threshold symptoms alone.

GOOD TO REMEMBER

- **Attenuated psychosis syndrome:** sub-threshold delusion-like ideas, unusual perceptual experiences or disorganised speech with reality testing relatively intact, distressing enough to warrant clinical attention; the one discriminator is preserved insight with symptoms below full psychotic threshold; hold the number: DSM-5-TR Section III condition for further study, symptoms at least once a week for the past month.
- **At-risk mental state / clinical high-risk:** the construct the syndrome formalises; the discriminating feature is help-seeking with sub-threshold symptoms not yet meeting psychotic-disorder criteria; the number to hold is a minority transition (about 15% at one year, 20% at two years), with most never converting.
- **Why "for further study":** only a minority transition, so the false-positive and stigma risks of formalising the label keep it out of the main diagnostic manual; the one point is that it is not a formal diagnosis.

10 · Differentiate: the schizophrenia-spectrum differential

Why this matters. This is the payoff of the whole primary-psychosis band: the examiner does not hand you a label, they hand you a presentation and ask which spectrum diagnosis it is. Every one of these disorders can look identical on a single afternoon of cross-section, a patient who is deluded, hallucinating and functioning poorly. What separates them is almost never the snapshot; it is the **longitudinal architecture**, chiefly two axes: how long the psychosis has run, and how much mood has been loaded onto it. Hold those two axes and the spectrum falls into an orderly grid; forget them and you will misclassify on the strength of a single interview.

This topic does not re-teach any of these disorders. Schizophrenia itself, its phenomenology, aetiology, criteria and course, is owned by the Schizophrenia: aetiology, phenomenology, classification and course notes; the raw descriptive definitions of the delusions live in the Psychometry, Assessment and Descriptive Psychopathology notes; the mood-episode criteria that

schizoaffective leans on are cross-referenced to the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; and the drug detail belongs to the Schizophrenia: management, antipsychotics and adverse effects notes. Here we do one thing: we **distinguish** the members of the spectrum from one another, keyed on the single discriminator for each pairing.

10.1 The two master axes: duration and mood-loading

Almost the entire spectrum resolves onto two continuous variables. The first is **duration of psychosis**: the DSM-5 ladder runs less than **one month** (brief psychotic disorder), **one to six months** (schizophreniform disorder), and more than **six months** (schizophrenia), with the ICD-11 threshold for schizophrenia set at **one month** or longer (Kaplan & Sadock 2024, DSM-5-TR 2022, ICD-11 CDDR). The second is **mood-loading**: how much of the illness is occupied by a full mood episode, and whether any psychosis ever runs in the absence of a prominent mood episode. Schizoaffective disorder, mood-with-psychosis and schizophrenia are separated almost entirely on this second axis. Learn to place a case on both axes before you reach for a name.

RULE **Duration and mood-loading are the master discriminators across the spectrum.** Duration sorts the short-course psychoses (brief, schizophreniform, schizophrenia); mood-loading sorts schizoaffective from mood-with-psychosis and from schizophrenia. Place the case on both axes first, then name it.

TRAP **Letting a cross-sectional snapshot of psychosis override the longitudinal criteria.** A single florid interview cannot distinguish brief psychotic disorder, schizophreniform disorder, schizophrenia or an acute mood episode with psychosis; only the course over time can. Diagnosing from the snapshot is the classic spectrum error.

10.2 Schizoaffective vs mood-with-psychosis vs schizophrenia: the mood-loading axis

This is the three-way discrimination that turns entirely on mood-loading, and it is the single highest-yield pairing on the spectrum. Two rules do the work (DSM-5-TR 2022, Kaplan & Sadock 2024). First, schizoaffective disorder requires a period of at least **two weeks** of delusions or hallucinations in the absence of a prominent mood episode over the lifetime of the illness (Criterion B): this is the two-weeks-of-psychosis-without-a-prominent-mood-episode rule, and it is what separates schizoaffective disorder from mood-with-psychosis, where psychosis is confined to the mood episode and never runs alone. Second, schizoaffective disorder requires that mood symptoms are present for the majority of the total duration of the active and residual illness (Criterion C): this mood-for-the-majority requirement is what separates it from schizophrenia, which carries no prominent mood for the majority of its course.

So the logic is a fork. If psychosis *only ever* appears inside a mood episode, it is **mood-with-psychosis** (a mood disorder with psychotic features), and its mood-episode criteria are set out in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes. If psychosis runs for at least two weeks *without* a prominent mood episode, and mood nonetheless occupies the majority of the illness, it is schizoaffective disorder. If psychosis dominates and mood is a minor, non-majority accompaniment, it is schizophrenia. The two-week gate excludes

mood-with-psychosis below; the majority rule excludes schizophrenia above; schizoaffective disorder lives only in the narrow band where both hold.

10.3 Delusional disorder vs paranoid schizophrenia: the breadth axis

Here duration is not the discriminator (both are chronic); breadth of the psychosis is. Delusional disorder is defined by one or more well-systematised, long-lasting delusions on a single circumscribed theme, classically *non-bizarre*, with the rest of mental life and personality relatively intact and social and occupational functioning largely preserved (New Oxford Textbook 2020, Kaplan & Sadock 2024, ICD-11 CDDR). What is *absent* is what makes the diagnosis: no persistent prominent hallucinations, no formal thought disorder or disorganisation, no negative symptoms, no experiences of influence or passivity, no general functional decline. The contrast with **paranoid schizophrenia** is exactly this breadth. Schizophrenia brings the wider syndrome, prominent hallucinations, disorganised thinking and behaviour, negative symptoms and functional deterioration, on top of the delusions; if those broader features are present, the ICD-11 boundary text is explicit that a diagnosis of schizophrenia is made instead of delusional disorder (ICD-11 CDDR). Two further pointers: delusional disorder comes to attention at a later age (onset generally in the mid- to late-30s) and shows far less deterioration over time than schizophrenia (Kaplan & Sadock 2024). Hallucinations may occur in delusional disorder if they are congruent with the delusional theme and not persistent; persistent, prominent hallucinations push the diagnosis over into schizophrenia.

RULE **One theme, preserved function equals delusional disorder; the wider syndrome equals schizophrenia.** A single-theme non-bizarre delusion with otherwise intact personality and function is delusional disorder; add prominent hallucinations, disorganisation, negative symptoms or functional decline and it becomes paranoid schizophrenia.

10.4 ATPD vs brief psychotic disorder vs schizophreniform: the duration ladder

These three are the short-course psychoses, and the discrimination is chiefly the duration ladder plus, for ATPD, a distinctive onset and course. In DSM-5-TR the ladder is clean: brief psychotic disorder lasts at least one day but less than **one month** with eventual full return to premorbid functioning; schizophreniform disorder lasts at least **one month but less than six months** and is otherwise identical to schizophrenia (a diagnosis of exclusion in time, made provisional if recovery has not yet occurred); and beyond six months with functional impairment the diagnosis becomes schizophrenia (DSM-5-TR 2022, Kaplan & Sadock 2024). The ICD-11 counterpart to the acute end is acute and transient psychotic disorder (ATPD, 6A23), and its signature is not merely brevity but the *quality* of the episode: an abrupt onset progressing from a non-psychotic to a clearly psychotic state within **two weeks**, with symptoms that fluctuate rapidly in intensity and type (the content of delusions and hallucinations shifting even on a daily basis), an absence of negative symptoms, a duration that does not exceed **three months** (most often lasting from a few days to one month), and a characteristically good outcome with full recovery (ICD-11 CDDR). This rapidly-shifting, abrupt-onset, short-and-remitting picture is the ATPD fingerprint; the older Scandinavian reactive-psychosis and cycloid-psychosis concepts feed into it.

Two boundary points the examiner likes. A case that meets all schizophrenia requirements but has lasted less than the one-month threshold, with no previous history of schizophrenia, is *not* ATPD in ICD-11: it is assigned to other specified primary psychotic disorder, because ATPD is defined by its fluctuating acute quality, not merely by being schizophrenia-cut-short (ICD-11 CDDR). And because ICD-11 sets the schizophrenia duration at one month, many cases that DSM-5 would call schizophreniform disorder are simply classified as schizophrenia in ICD-11; there is no schizophreniform category in ICD-11 (Kaplan & Sadock 2024).

under 1 mo	1 to 6 mo	up to 3 mo	within 2 wk
BRIEF PSYCHOTIC DISORDER (DSM-5-TR)	SCHIZOPHRENIFORM DISORDER (DSM-5-TR)	ATPD, DOES NOT EXCEED THREE MONTHS (ICD-11)	ATPD ONSET, NON-PSYCHOTIC TO PSYCHOTIC (ICD-11)

10.5 The interleaved spectrum differential

The grid below interleaves all the spectrum members onto the two master axes and names, for each, the one discriminator that settles the pairing. Read it as a single decision tool: place the case by presentation, duration and mood-loading, then read off the discriminator column.

DISORDER	PRESENTATION	DURATION	MOOD-LOADING	THE ONE DISCRIMINATOR
Brief psychotic disorder	Acute florid psychosis, often with a stressor; full return to premorbid function	1 day to under 1 month	None required	**Resolves in under one month with full recovery**
ATPD (ICD-11 6A23)	Abrupt onset, rapidly fluctuating delusions or hallucinations shifting even daily; no negative symptoms; good outcome	Onset within 2 weeks; does not exceed 3 months (often days to 1 month)	Variable, mood may colour the picture but is not the axis	**Abrupt onset plus rapidly shifting content plus short remitting course**
Schizophreniform disorder	Identical to schizophrenia in symptoms; functional decline not required	1 to 6 months	Mood-with-psychosis and schizoaffective ruled out	**Duration one to six months (provisional if recovery not yet occurred)**
Schizophrenia	Full syndrome: delusions, hallucinations, disorganisation, negative symptoms, functional decline	6 months or more (ICD-11: 1 month or more)	No prominent mood for the majority of the illness	**Duration over 6 months (owned by the schizophrenia notes)**

DISORDER	PRESENTATION	DURATION	MOOD-LOADING	THE ONE DISCRIMINATOR
Schizoaffective disorder	Full mood episode concurrent with schizophrenia-criterion psychosis	Chronic	Mood for the majority; at least 2 weeks of psychosis without a prominent mood episode	**Two weeks of psychosis without a prominent mood episode, and mood for the majority**
Mood-with-psychosis	Delusions or hallucinations occurring only within a mood episode	Duration of the mood episode	Maximal, psychosis is confined to the mood episode	**Psychosis only within mood episodes, never for two weeks alone**
Delusional disorder	Single-theme, well-systematised, non-bizarre delusion; personality and function preserved	Chronic, at least 3 months (ICD-11); later onset (mid to late 30s)	Low, mood not the axis	**One non-bizarre theme with preserved function and no wider syndrome**
Paranoid schizophrenia	Persecutory delusions plus prominent hallucinations, disorganisation, negative symptoms, decline	6 months or more	No prominent mood for the majority	**The wider syndrome and functional decline on top of the delusions**

The spectrum keyed on two axes: duration sorts the short-course psychoses; mood-loading sorts schizoaffective from mood-with-psychosis and schizophrenia; breadth sorts delusional disorder from paranoid schizophrenia.

10.6 Working the differential in practice

The examiner rewards a method, not a lucky guess. Take any psychotic presentation and ask, in order: has an organic or substance cause been excluded (a detailed history, examination and, where indicated, toxicology and imaging, since a rapid onset in someone with a substance history should raise substance-induced psychosis)? How long has the psychosis run in its full form (the duration ladder)? Does any psychosis run in the absence of a prominent mood episode, and does mood occupy the majority of the illness (the mood-loading fork)? Is the psychosis a single circumscribed non-bizarre theme with preserved function, or the wider syndrome with decline (the breadth axis)? Is the onset abrupt with rapidly shifting content and a short remitting course (the ATPD fingerprint)? Answer those five in sequence and the spectrum diagnosis is usually forced. The one thing that must never drive the answer is the cross-sectional snapshot alone.

COMMON TRAP

Do not diagnose schizoaffective disorder simply because psychosis and a mood episode co-occur. Concurrent psychotic and mood symptoms are equally compatible with a mood disorder with psychotic features; the diagnosis turns on the two-weeks-of-psychosis-without-a-prominent-mood-episode rule (Criterion B) and the mood-for-the-majority rule (Criterion C). Without the two-week window this is mood-with-psychosis, not schizoaffective disorder.

INDIA

Acute, abrupt-onset, short-course psychoses of the ATPD type are a well-recognised and relatively common presentation in Indian practice, frequently precipitated by an acute stressor and carrying a good outcome, which fits the classic acute-and-transient picture; longitudinal follow-up remains essential because a proportion later declare themselves as schizophrenia or a mood disorder. Delusional disorder is similarly encountered as a late-onset, single-theme, function-preserving condition. Verify any local prevalence claim against a primary source before quoting a figure.

MNEMONIC

DURATION, MOOD, BREADTH

Three questions settle almost the whole spectrum. DURATION sorts the short-course psychoses (under one month brief, one to six months schizophreniform, over six months schizophrenia). MOOD-loading sorts schizoaffective (two weeks of psychosis without a prominent mood episode, plus mood for the majority) from mood-with-psychosis (psychosis only within mood episodes) and schizophrenia (no prominent mood for the majority). BREADTH sorts delusional disorder (one theme, preserved function) from paranoid schizophrenia (the wider syndrome, decline).

HOLD THIS

Never diagnose the spectrum from a cross-sectional snapshot: duration (the under-one-month / one-to-six-month / over-six-month ladder) and mood-loading (two weeks of psychosis without a prominent mood episode, plus mood for the majority) are the master discriminators, and breadth (one non-bizarre theme with preserved function versus the wider syndrome) settles delusional disorder against paranoid schizophrenia.

GOOD TO REMEMBER

- **Schizoaffective vs mood-with-psychosis vs schizophrenia:** schizoaffective needs at least **two weeks** of psychosis without a prominent mood episode (Criterion B, separates it from mood-with-psychosis) and mood for the majority of the illness (Criterion C, separates it from schizophrenia); mood-with-psychosis has psychosis only within mood episodes; the number to hold is two weeks.
- **Delusional disorder vs paranoid schizophrenia:** a single-theme, non-bizarre delusion with preserved personality and function is delusional disorder; add prominent hallucinations, disorganisation, negative symptoms or decline and it is paranoid schizophrenia; delusional disorder onset is later (mid to late 30s).
- **Brief psychotic disorder:** acute psychosis lasting at least one day but less than **one month** with full recovery; the one number is under one month (DSM-5-TR).
- **Schizophreniform disorder:** schizophrenia-identical symptoms lasting **one to six months**; the one eponym is Langfeldt (1939), who introduced the concept; no equivalent in ICD-11.
- **ATPD (ICD-11 6A23):** abrupt onset from non-psychotic to psychotic within **two weeks**, rapidly shifting content, no negative symptoms, duration not exceeding **three months** (often days to one month), good outcome; the discriminator is the abrupt-onset, fluctuating, short-remitting quality, not brevity alone.
- **The two master axes:** duration sorts the short-course psychoses; mood-loading sorts schizoaffective from mood-with-psychosis and schizophrenia; the trap is diagnosing from the cross-sectional snapshot instead of the longitudinal course.

Delusional misidentification and named delusional syndromes

11 · Delusional misidentification syndromes

Why this matters. The **delusional misidentification syndromes** are a small keyed grid the examiner loves precisely because the eponyms are so easy to swap: four named beliefs, each turning on *who* is misidentified and in *which direction*. They are not a disorder in their own right but a recurring content pattern that rides on top of schizophrenia, mood disorder with psychosis, dementia and focal brain disease. The whole topic can be held on two axes: the familiar-versus-stranger axis (Capgras is the familiar seen as an impostor; Fregoli is the stranger seen as a familiar-in-disguise), and the appearance-versus-identity axis (whether the physical face changes, the psychological identity changes, or both).

Because these beliefs so often sit on organic pathology, the topic doubles as a trap about aetiology: a striking eponymous delusion is an invitation to look for a right-hemisphere lesion, a dementia or a metabolic cause, not to reflexively call it functional psychosis. The raw descriptive phenomenology of each belief (how it is elicited and graded at the mental-state level) is cross-referenced to the Psychometry, Assessment and Descriptive Psychopathology notes; here the job is the diagnosis and the discrimination. The **misidentification syndromes** are grouped in ICD-11 and DSM-5-TR under other specified schizophrenia-spectrum and other psychotic disorders when they stand alone (Kaplan & Sadock 2024).

Syndrome	The core misidentification	Typical association
Capgras	A familiar person seen as an identical impostor	commonest; up to 40% organic; right hemisphere
Fregoli	One persecutor disguised inside strangers	persecutory psychosis; rarer; Courbon & Fail 1927
Inter-metamorphosis	Appearance AND identity both swapped	total transformation; rare
Subjective doubles	Doubles of the self, acting on their own	not heautoscopy (self seen)
Reduplicative paramnesia	A place or object believed duplicated	right-hemisphere / frontal lesion

Fig 10.5 The delusional misidentification syndromes, keyed on the core misidentification. Capgras and Fregoli are direction-reversed (the familiar seen as an impostor versus one persecutor seen disguised inside strangers); intermetamorphosis changes both appearance and identity; subjective doubles turns the belief on the self; reduplicative paramnesia is the organic marker of the group. A face-processing disconnection and a high rate of right-hemisphere or organic pathology make excluding an organic cause mandatory.

11.1 Capgras syndrome: the familiar seen as an impostor

Capgras syndrome. The belief that a familiar person, to whom the sufferer is emotionally close (typically a spouse or relative, and sometimes a pet or an object), has been replaced by an identical impostor or double who has assumed that person's role and behaves like them (Companion 2010, Kaplan & Sadock 2024). It is the most common of the group. The conviction can be dangerous: some patients threaten, harm or even kill the supposed impostor, so the recognition of Capgras

carries a real risk assessment. It was described by the French psychiatrist Joseph Capgras (Capgras & Reboul-Lachaux 1923), who called it the "illusion of doubles" (Tasman 2015). The core is a paradox the patient rationalises: the person looks exactly the same but does not *feel* the same, and so must be a substitute.

RULE **Capgras is the familiar seen as an impostor; Fregoli is the stranger seen as a familiar-in-disguise.** Fix the direction first (who is real, who is disguised) and the two eponyms stop swapping.

11.2 Fregoli syndrome: the persecutor disguised as everyone

Fregoli syndrome. The belief that a persecutor is present but disguised as different people the patient meets: a single familiar figure (usually the persecutor) is repeatedly identified inside strangers who bear no physical resemblance, so the doctor is "really" the persecutor, then the nurse is, and so on, yet the persecutor is always one and the same (New Oxford 2020, Shorter Oxford 2018). The direction is the mirror-image of Capgras: the outward appearance is a stranger's, but the inner identity is claimed to be a known person. It was originally described by Courbon and Fail in 1927 and is even rarer than the Capgras delusion (Shorter Oxford 2018), named after Leopoldo Fregoli, the Italian stage actor famous for rapid changes of appearance and character.

TRAP **Attributing misidentification to a functional psychosis without excluding neurological or organic disease.** Up to 40% of Capgras cases carry an organic disorder (head injury, dementia); a striking misidentification delusion mandates an organic screen before it is called schizophrenia.

11.3 Intermetamorphosis and the syndrome of subjective doubles

Intermetamorphosis. The belief that others have been swapped in *both* physical appearance and identity, so that people (and sometimes animals or objects) are transformed into one another; unlike Fregoli, where only the hidden identity changes, here the delusion is of a total transformation, psychological as well as physical (New Oxford 2020, AIIMS Clinical Methods 2024). **Syndrome of subjective doubles.** The belief in doubles of oneself: the patient is convinced that exact physical copies of themselves exist and carry out independent actions, each with a separate psychological identity (New Oxford 2020, AIIMS Clinical Methods 2024). It is distinct from heautoscopy, in which the self is *seen* as a visual double rather than believed to exist as an independent agent.

11.4 The proposed mechanism: a face processing disconnection

The influential cognitive account of Capgras, and by extension the group, is the two-route model of face processing (Ellis & Young 1990). It proposes two routes to facial recognition: one for the identification of the face itself and one that gives the face its affective sense of familiarity. Prosopagnosia is a loss of the first route; Capgras is proposed as its "mirror image", an intact primary recognition route with a disconnection of the second, so the face is recognised as identical but is stripped of the emotional feeling that should accompany a loved one (Ellis & Young 1990). Ellis et al (1997) supported this by showing that Capgras patients fail to show the normal autonomic (skin-conductance) discrimination between familiar and unfamiliar faces. This

account is consistent with the high rate of right-hemisphere and organic pathology in these patients (Companion 2010). The patient mistakes a change in themselves (the missing sense of familiarity) for a change in the other person, and rationalises it as substitution.

The imperative to exclude an organic cause. Because delusional misidentification is so strongly associated with right-hemisphere dysfunction and structural or metabolic brain disease, an organic cause must be actively excluded in every case. Capgras has been reported in central nervous system lesions, dementia (especially dementia with Lewy bodies), vitamin B12 deficiency, hepatic encephalopathy, diabetes and hypothyroidism (AIIMS Clinical Methods 2024, Companion 2010). Right-sided cerebral pathology also produces reduplicative paramnesia, the belief that a familiar place, object or body part has been duplicated, which sits alongside the misidentification group and points the same way, toward the right hemisphere.

up to 40%
OF CAPGRAS CASES CARRY AN ORGANIC DISORDER (COMPANION 2010)

1927
COURBON AND FAIL DESCRIBE FREGOLI SYNDROME

right hemisphere
THE PATHOLOGY TO SUSPECT ACROSS THE GROUP

11.5 The differentiation grid

The grid is the deliverable: each syndrome is defined by *who* is misidentified and in *which* direction (familiar to stranger, or the reverse), and by whether appearance, identity, or both are held to have changed. Read down the "core misidentification" column and the eponyms separate cleanly.

SYNDROME	THE CORE MISIDENTIFICATION	TYPICAL ASSOCIATION
Capgras syndrome	**A familiar person (or pet or object) is believed replaced by an identical impostor or double; looks the same, does not feel the same**	Most common of the group; schizophrenia; organic disease in up to 40%; right-hemisphere pathology
Fregoli syndrome	**A persecutor is present but disguised as different strangers the patient meets; one persecutor inside many faces**	Persecutory psychosis; rarer than Capgras; Courbon and Fail 1927
Intermetamorphosis	**Others are swapped in both physical appearance and identity; a total transformation, not merely a disguise**	Psychotic disorders; rare; may include animals and objects
Syndrome of subjective doubles	**Belief in exact doubles of oneself, acting independently with separate identities**	Psychotic disorders; distinguish from heautoscopy (the self seen, not believed independent)
Reduplicative paramnesia	A familiar place, object or body part is believed duplicated (e.g. the real home moved and replaced by an identical one)	Right-hemisphere and frontal lesions; organic marker rather than functional psychosis

The misidentification grid: Capgras and Fregoli are direction-reversed (familiar-as-impostor versus stranger-as-familiar); intermetamorphosis changes both appearance and identity; subjective doubles turns the belief on the self.

HOLD THIS

Capgras = the familiar person seen as an identical impostor (looks the same, does not feel the same); Fregoli = the one persecutor seen disguised inside many strangers; a face processing disconnection between recognition and the affective sense of familiarity, plus right-hemisphere or organic pathology in a large minority, makes excluding an organic cause mandatory.

PEARL

The two-route face-recognition model gives the group a tidy logic: prosopagnosia loses the recognition route but keeps the feeling of familiarity; Capgras is the mirror image, keeping recognition but losing the feeling, so the face is known yet feels wrong and is rationalised as a substitute (Ellis & Young 1990). One model, two opposite clinical pictures.

INDIA

In Indian practice these syndromes are seen most often as content within schizophrenia and, increasingly, within dementia in older patients; the clinical discipline is identical, an organic and metabolic screen (including B12, thyroid and neuroimaging where a focal or right-hemisphere picture is suspected) before the belief is attributed to a functional psychosis. The named beliefs are managed by treating the underlying disorder, not as entities in themselves.

GOOD TO REMEMBER

- **Capgras syndrome:** a familiar person replaced by an identical impostor; the discriminator is "looks the same, does not feel the same"; the number to hold is up to 40% organic, and the eponym is Joseph Capgras (1923).
- **Fregoli syndrome:** a persecutor disguised as different strangers the patient meets; the discriminator is one identity inside many unfamiliar faces (mirror image of Capgras); the eponym and date are Courbon and Fail, 1927.
- **Intermetamorphosis:** others swapped in both physical appearance and identity; the discriminator is a total transformation (not just a disguise); hold that both appearance and identity change.
- **Syndrome of subjective doubles:** the belief in doubles of oneself acting independently; the discriminator is that the double is of the self and is believed to exist as an agent; distinguish it from heautoscopy.
- **The mechanism:** a face processing disconnection between visual recognition and the affective sense of familiarity; the fact to hold is the high rate of right-hemisphere and organic pathology and the imperative to exclude an organic cause (Ellis & Young 1990, Companion 2010).

12 · Named monothematic delusional syndromes

A handful of delusions carry their own eponyms, and the examiner loves them precisely because they are quick to name and easy to misattribute. Each is a **monothematic** delusion: a single, circumscribed belief that dominates the clinical picture while the rest of thought stays comparatively intact (Coltheart, Langdon & McKay 2007; New Oxford Textbook). The trap built into the whole set is a category error, treating the eponym as a diagnosis in its own right, when almost all of these are **delusional content** that can appear across schizophrenia, delusional disorder, a mood episode or an organic state.

Why it matters. Marks here are won on three moves: attaching each eponym to the correct content without swapping them, stating that the content is transdiagnostic rather than a disorder in itself, and, for one of them, recognising a real forensic danger. The raw descriptive definitions of the delusions themselves belong to the Psychometry, Assessment and Descriptive Psychopathology notes; the task here is to name the content precisely and then find the illness underneath it.

Eponym	Core delusional content	Association or risk
De Clerambault (erotomania)	A higher-status person is secretly in love	solitary women; stalking
Othello (delusional jealousy)	A partner is being unfaithful	violence and homicide risk; commoner in men
Cotard (nihilistic)	Being dead, not existing, or the organs lost	severe psychotic depression; severity marker
Ekbom (infestation)	Infested with parasites under the skin	matchbox sign; presents to dermatology

Fig 10.6 The named monothematic delusional syndromes, keyed on the core delusional content. Each is delusional content that can occur across schizophrenia, delusional disorder, a mood episode or an organic state, not a disorder in itself: name the content, then find the illness. Othello syndrome (delusional jealousy) is the one that carries a genuine forensic risk of violence to the partner.

12.1 Content, not disorder: the governing principle

Name the content, then find the illness. These eponymous syndromes are defined by *what* the patient believes, not by any distinct aetiology or course. Each can occur as the somatocentric, erotocentric or securocentric expression of an underlying condition, so the same named delusion is seen in schizophrenia, in delusional disorder as a monothematic somatic or persecutory belief, in a severe mood episode, and in organic and drug-induced states (New Oxford Textbook; Tasman; Kaplan & Sadock 2024). The clinical move is therefore two-step: recognise the content, attach the eponym, then work out the diagnostic bed it sits in. Only where the belief stands alone, monosymptomatic and monothematic with otherwise preserved functioning, does it become a delusional disorder in its own right (New Oxford Textbook).

RULE **Eponym first, diagnosis second.** Erotomania, morbid jealousy, nihilism and infestation are *content* that occurs across schizophrenia, delusional disorder, mood disorder and organic states; name the content, then find the illness that carries it.

12.2 De Clerambault syndrome: erotomania

The delusion of being loved. In de clerambault syndrome, also written **clerambault**'s syndrome and synonymous with erotomania (psychose passionelle), the patient is imperturbably convinced that another person, usually of higher social status and often a celebrity or a distant figure, is secretly in love with them (New Oxford Textbook; Kaplan & Sadock 2024; AIIMS Clinical Methods 2024). The supposed lover is typically unaware, unattainable and out of reach, and the belief persists even when that person ignores or rejects the patient's contacts (Kaplan & Sadock

2024). It is classically described in solitary, withdrawn, dependent women, though it is not confined to them, and it can drive persistent stalking and unwanted approach behaviour (Kaplan & Sadock 2024; New Oxford Textbook). Erotomanic content is not unique to delusional disorder and occurs as a symptom of other psychotic conditions (Tasman).

GOOD TO REMEMBER

- **De Clerambault syndrome (erotomania):** the delusion that another person, usually of higher status, is secretly in love with the patient. Discriminator: the object is typically unaware and unattainable, yet the conviction is unshakeable. Eponym to hold: de Clerambault (psychose passionelle); classically described in women.

12.3 Othello syndrome: delusional jealousy

The dangerous one. In othello syndrome, also called morbid jealousy, conjugal paranoia or delusional jealousy, the patient holds the fixed, false belief that their sexual partner is being unfaithful, pursued with relentless checking, interrogation and demands for confession (New Oxford Textbook). The name is drawn directly from Shakespeare's Othello, who killed his wife on a false belief of infidelity (New Oxford Textbook). This is the syndrome that carries a genuine forensic risk: the jealous delusion is associated with a real danger of violence, including homicide, toward the accused partner, and morbid jealousy is over-represented in spousal violence and killing (New Oxford Textbook; Companion to Psychiatric Studies). It is reported as more common in men, and, like the others, delusional jealousy is transdiagnostic and can be a symptom of schizophrenia, an organic or alcohol-related state, or delusional disorder (Companion to Psychiatric Studies; New Oxford Textbook).

TRAP **Treating Othello syndrome as benign.** Delusional jealousy is not a quirk of a suspicious marriage; it carries a real risk of harm to the partner, up to homicide, and mandates a formal risk assessment and, often, separation for the partner's safety.

GOOD TO REMEMBER

- **Othello syndrome (delusional jealousy):** the delusion that a partner is unfaithful. Discriminator: fixed, false conviction of infidelity, pursued with checking and demands for confession, distinct from ordinary jealousy. Forensic caveat to hold: genuine risk of violence and homicide toward the partner; reported commoner in men.

12.4 Cotard syndrome: the nihilistic delusion

The delusion of non-existence. Cotard syndrome, the delusion of negation, is the nihilistic delusion that the self, others or the world no longer exist, have been destroyed, or have lost their meaning and value (Shorter Oxford Textbook; AIIMS Clinical Methods 2024). In its fuller forms the patient believes they are dead, do not exist, or have lost or rotted away their internal organs, that the bowels are blocked or the blood has stopped, and it may extend to a delusion that the whole world is about to end (Shorter Oxford Textbook). It is classically seen in severe, often psychotic, depression, and also occurs in schizophrenia and in organic states (Shorter Oxford Textbook; Kaplan & Sadock 2024). It is a marker of severity: the nihilistic-somatic core signals a profound depressive or organic illness rather than a benign hypochondriacal worry.

GOOD TO REMEMBER

- **Cotard syndrome (nihilistic delusion):** the delusion of being dead, not existing, or having lost the internal organs. Discriminator: nihilistic-somatic negation, classically bowels or blood, not merely fear of illness. Association to hold: severe, often psychotic, depression (Jules Cotard, delire de negation).

12.5 Ekbom syndrome: delusional infestation

The delusion of parasites under the skin. Ekbom syndrome, or delusional infestation (delusional parasitosis, acarophobia, monosymptomatic hypochondriacal psychosis), is the fixed belief that one is infested with parasites, insects or small organisms crawling on or under the skin (Kaplan & Sadock 2024; Shorter Oxford Textbook). Thibierge first described the condition in the 1890s; it is now widely termed Ekbom syndrome after Karl-Axel Ekbom (Kaplan & Sadock 2024). The hallmark clinical sign is the **matchbox sign**: the patient brings collected specimens, usually skin, hair or lint, in a small container as proof of the infestation (Kaplan & Sadock 2024). These patients present most often to dermatology rather than psychiatry, and self-treat by washing, excoriating and using insecticides (Kaplan & Sadock 2024). It is the commonest presentation of monosymptomatic hypochondriacal psychosis and, again, can be primary (delusional disorder, somatic type) or secondary to schizophrenia, depression, dementia or drug intoxication such as cocaine (Tasman; Kaplan & Sadock 2024).

PEARL

The matchbox sign (the patient arriving with a container of "specimens", now sometimes the "specimen-jar" or smartphone-photo sign) is near-pathognomonic for delusional infestation and a fast bedside pointer. Before settling on Ekbom syndrome, exclude a real infestation, and screen for the organic and drug causes, cocaine and amphetamine "formication", B12 deficiency, hypothyroidism and dementia, that mimic it.

GOOD TO REMEMBER

- **Ekbom syndrome (delusional infestation):** the delusion of being infested with parasites or small organisms. Discriminator: the matchbox sign, specimens brought as proof; presents to dermatology. Eponym to hold: Karl-Axel Ekbom (first described by Thibierge, 1890s); commonest monosymptomatic hypochondriacal psychosis.

12.6 The keyed grid: eponym, content, association

The four in one table. The classic way to hold these is by their arche-theme: erotocentric (erotomania, jealousy), securocentric (persecutory, misidentification) and somatocentric (infestation, nihilistic-somatic) content (New Oxford Textbook). The grid below pins each eponym to its core delusional content and its single most exam-relevant association or risk. Every row is content that can occur across diagnoses; the eponym names the belief, not the illness.

EPONYM	CORE DELUSIONAL CONTENT	CLASSIC ASSOCIATION OR RISK
De Clerambault syndrome	**Another person, often of higher status, is secretly in love with the patient (erotomania)**	Classically solitary women; stalking and unwanted approach
Othello syndrome	**A partner is being unfaithful (delusional jealousy)**	**Real forensic risk of violence and homicide to the partner; commoner in men**
Cotard syndrome	**One is dead, does not exist, or has lost the internal organs (nihilistic delusion)**	Severe, often psychotic, depression; a severity marker
Ekbom syndrome	**One is infested with parasites or small organisms (delusional infestation)**	Matchbox sign; presents to dermatology; exclude cocaine, B12, dementia

Fig 10.6: the named monothematic delusional syndromes, eponym to content to association; each is delusional content that occurs across diagnoses, not a disorder in itself.

MNEMONIC

JELLI: Jealousy, Erotomania, Loss-of-existence, Little-organisms Infest

Othello = Jealousy; de Clerambault = Erotomania; Cotard = Loss of existence (nihilism); Ekbom = Little organisms Infest the skin. Four eponyms, four contents, in one string.

INDIA

In Indian delusional-disorder series the somatic and jealous subtypes are well represented, and delusional infestation and morbid jealousy are recognisable presentations in general and old-age psychiatry practice; the jealous subtype in particular carries the same domestic-violence risk that mandates a routine risk assessment and, where alcohol is the driver, attention to the alcohol-related state underlying the jealousy. The onset in delusional disorder is typically later than in schizophrenia (Companion to Psychiatric Studies), which fits the older, married patient in whom Othello and Ekbom syndromes are so often seen.

HOLD THIS

De Clerambault (loved by another), Othello (partner unfaithful), Cotard (dead or without organs) and Ekbom (infested) are monothematic delusional *contents* that occur across schizophrenia, delusional disorder, mood disorder and organic states; name the content, find the illness, and never call Othello syndrome benign, it can kill the partner.

Catatonia

13 · Catatonia: the signs and their examination

Catatonia is elicited, not merely observed. **Catatonia** is a syndrome of disturbed motility, volition and reactivity that sits *transdiagnostically* across mood, psychotic, autistic, developmental and organic disease. The examiner who only watches a still patient will miss it; the one who

reaches out, resists, mirrors and instructs will find it. This topic teaches the **catatonic signs** in their classical and modern groupings, the bedside **examination** that elicits them, and the Bush-Francis rating scale that standardises the search. The raw descriptive definitions of the movement and volition disorders are cross-referenced to the Psychometry, Assessment and Descriptive Psychopathology notes; here the target is recognition and the discrimination from the mimics that get catatonia missed.

The high-yield frame for a viva: catatonia is a *clinical* diagnosis made on a defined constellation of signs, most of which must be actively drawn out. Naming the sign, describing how you elicit it, and placing it on the Bush-Francis map is the examiner-pleasing sequence.

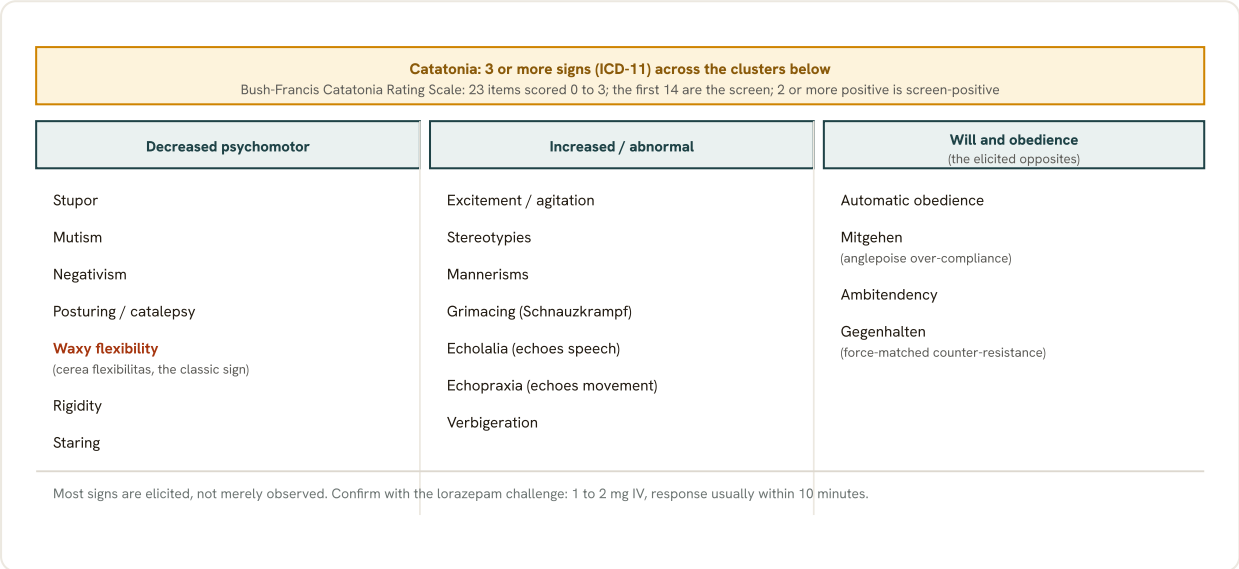


Fig 10.7 The catatonic signs grouped across the three ICD-11 psychomotor clusters, mapped onto the Bush-Francis rating scale. Waxy flexibility (cerea flexibilitas) is the classic elicited sign. Most signs must be actively drawn out at the bedside, which is why stuporous catatonia is so easily missed as depression or non-cooperation; three or more signs meet the ICD-11 threshold, and a lorazepam challenge confirms.

13.1 The Kahlbaum picture and the transdiagnostic modern concept

Origin. Kahlbaum described catatonia (*Die Katatonie oder das Spannungsirresein*, the "tension insanity") in 1874 as a cyclical illness passing through melancholic, manic, stuporous and, ultimately, dementing phases (Kahlbaum 1874). Kraepelin then folded it into dementia praecox as a subtype, and catatonia was for a century treated as a form of schizophrenia. That is the error the modern concept corrects.

The modern reframe. Catatonia is a *transdiagnostic* motor syndrome: in a New York cohort of catatonic inpatients only a small minority met criteria for schizophrenia, while more than two-thirds had affective disorders, usually mania (Kaplan & Sadock 2024). The disorder schizophrenia itself is not re-taught here; its phenomenology and criteria are owned by the Schizophrenia: aetiology, phenomenology, classification and course notes. What matters for catatonia is that a mood episode, an organic or medical illness, autism or substances are each more likely than schizophrenia to be the substrate.

RULE **Catatonia is a syndrome, not a subtype.** Diagnose the sign-constellation first, then hunt the cause; mood and medical illness outrank schizophrenia as the substrate.

13.2 The ICD-11 placement and diagnostic requirement

Nosology (ICD-11 primary). ICD-11 makes catatonia a free-standing, cross-cutting entity with its own grouping: 6A40 Catatonia associated with another mental disorder, 6A41 Catatonia induced by substances or medications, 6E69 Secondary catatonia syndrome, and 6A4Z Catatonia, unspecified (ICD-11 CDDR 2024). This is a genuinely high-yield point: catatonia is coded independently of the underlying disorder.

The threshold. ICD-11 requires **three or more** symptoms of decreased, increased or abnormal psychomotor activity, drawn from any combination of three clusters, typically lasting **at least several hours** (ICD-11 CDDR 2024). DSM-5-TR cross-maps closely, requiring **three or more** of twelve psychomotor features (DSM-5-TR 2022). ICD-10 flagged the older habit of counting catatonia as a schizophrenia subtype, which both current systems abandon.

three or more	3 clusters	6A40
SIGNS FOR ICD-11 / DSM-5-TR CATATONIA	DECREASED, INCREASED, ABNORMAL PSYCHOMOTOR	ICD-11 CODE, ASSOCIATED WITH ANOTHER DISORDER

13.3 The motor and volitional signs

The core cluster. These are the hypokinetic and postural signs that dominate the classical stuporous picture.

Stupor

Immobility with no or markedly reduced psychomotor activity, minimally responsive to external stimuli, yet the patient is often aware of the surroundings (Fish, in Companion 2010).

Mutism

No or very little verbal response; speech hushed to unintelligibility. Do not count if due to a nervous-system disease or a speech/language disorder (ICD-11 CDDR 2024).

Negativism

Motiveless resistance to instruction; the patient does the reverse of what is asked, holding the breath when told to breathe deeply, resisting being stood up then refusing to lie down (Sims 2015).

Posturing / catalepsy

Posturing: spontaneous active maintenance of a posture against gravity. Catalepsy: passive induction of a posture by the examiner that is then held against gravity (ICD-11 CDDR 2024).

Waxy flexibility (cerea flexibilitas)

Slight, even, plastic resistance to positioning, "like bending a warm candle wax," the limb then held in the new position (ICD-11 CDDR 2024).

Rigidity

Resistance from increased tone, from mild to severe "lead-pipe" rigidity; requires examination (ICD-11 CDDR 2024).

Staring

Fixed gaze with decreased blinking, often with widely opened eyes (ICD-11 CDDR 2024).

Waxy flexibility is the classic teaching sign of catatonia: the examiner moulds the limb and it stays. It is distinct from *catalepsy* (posture held without the plastic resistance) and from lead-pipe *rigidity* (uniform resistance with no "give").

> **HOLD THIS**
> Waxy flexibility (cerea flexibilitas) is the classic catatonic sign: mould the limb like warm wax and it holds the new position against gravity.

13.4 The echo phenomena, and the disorders of will and obedience

Echo phenomena. Echolalia is the automatic mirroring of the examiner's speech; echopraxia is the mirroring of the examiner's movements. ICD-11 groups them as "echophenomena" under abnormal psychomotor activity (ICD-11 CDDR 2024).

Disorders of will and obedience. These paired opposites are the volitional heart of catatonia and are pure elicited signs.

Automatic obedience

Every instruction is carried out regardless of consequence. Kraepelin's demonstration was to ask the patient to put out the tongue and prick it with a pin; the patient kept protruding it despite the repeated prick.

Mitgehen

Extreme cooperation: the limb moves in the direction of the examiner's slightest pressure ("anglepoise-lamp" sign), then returns, despite instruction to resist (Companion 2010).

Ambitendency

The patient appears "motorically stuck," alternating between starting and stopping a movement in indecisive, hesitant fashion (ICD-11 CDDR 2024).

Gegenhalten (opposition)

The opposite of mitgehen: springy resistance to passive movement that increases in proportion to the force the examiner applies (Sims 2015; Companion 2010).

The clinical elegance is the pairing: mitgehen is over-compliance with passive movement, gegenhalten is motiveless counter-resistance to it, and both can appear in the same patient.

13.5 Mannerisms, stereotypies, grimacing and verbigeration

The abnormal-activity cluster. These are the "odd but active" signs.

Mannerisms

Odd, purposeful, goal-directed acts executed in an idiosyncratic, exaggerated-caricature way (e.g. a bizarre style of washing or dressing) (ICD-11 CDDR 2024; Companion 2010).

Stereotypies

Repetitive, non-goal-directed motor activity (finger-play, patting or rubbing the self). The abnormality is not in the action but in its frequency (ICD-11 CDDR 2024).

Grimacing

Odd or distorted facial expressions inappropriate to the situation. The exaggerated forward-thrusting of the lips (Schnauzkrampf, snout spasm) is a related facial sign that the AIIMS Clinical Methods text

files as a facial stereotypy (ICD-11 CDDR 2024; AIIMS 2024).

Verbigeration

Continuous, directionless repetition of words, phrases or sentences; the senseless repetition of sounds or words (ICD-11 CDDR 2024; Shorter Oxford 2018).

In practice mannerisms and stereotypies shade into one another; both are motor behaviour rendered abnormal by repetitiveness, purposelessness or stiltedness. Mannerisms retain a goal (a distorted *way* of doing a normal act); stereotypies have no goal at all.

13.6 The bedside examination: catatonia is elicited

Observe, then elicit. A structured examination splits the signs into those you simply observe (staring, mutism, grimacing, stupor, excitement, stereotypy, mannerism, verbigeration, spontaneous echo) and those you must actively draw out (rigidity, negativism, posturing, waxy flexibility, catalepsy, automatic obedience, mitgehen, mitmachen, ambitendency, echo phenomena) (AIIMS 2024). The whole discipline of the topic is that most catatonic signs live in the second column.

How to elicit the key signs.

- **Posturing and waxy flexibility.** Passively move the neck, jaw, shoulders, elbows, fingers and lower limbs; feel for the even, plastic "candle-wax" resistance and watch whether the induced posture is held against gravity (catalepsy). Look for the "psychological pillow," where the head is held elevated as if on a pillow (AIIMS 2024).
- **Negativism and gegenhalten.** Instruct simple actions and note reversal; then move a limb passively and feel for springy resistance that grows to match your force (AIIMS 2024).
- **Automatic obedience and mitgehen.** Instruct the patient to resist your manipulation; if the limb nonetheless rises with the slightest upward pressure of your finger and returns when released, that is mitgehen. The pin-prick tongue test is the historical automatic-obedience manoeuvre; instructing the patient to "resist" is the humane modern substitute (AIIMS 2024).
- **Echo phenomena.** Perform a gesture or a phrase and watch for spontaneous mirroring (echopraxia, echolalia).

WORKED EXAMPLE

A mute, immobile young man on the ward is written up as "uncooperative." You lift his arm; it offers the even plastic resistance of warm wax and stays raised (waxy flexibility, catalepsy). You press a finger under his forearm having told him to keep still; the arm floats upward and returns when you let go (mitgehen). You now have two elicited signs, staring and mutism observed: four signs, well over the three-sign threshold. This is catatonia, not obstinacy.

13.7 The Bush-Francis rating scale: domains and screen

The standard instrument. The Bush-Francis Catatonia Rating Scale (BFCRS) is the preferred scale for routine use for its validity, reliability and ease of administration (Bush 1996; AIIMS 2024). It has 23 items rated 0 to 3 for severity, and the presence or absence of the first 14 items

forms the standardised *screening* examination (the Bush-Francis Catatonia Screening Instrument, BFCSI) (Bush 1996; AIIMS 2024).

The screen. By convention, **two or more** of the first 14 signs identify probable catatonia and warrant the full 23-item rating and a lorazepam challenge (a widely-applied screening cutoff; the standardised instrument is Bush 1996). The 14 screening items span the same clusters covered above: the standardised examination proceeds observe-then-elicit, exactly as in 13.6. FIG 10.7 maps the catatonic signs onto the Bush-Francis domains.

23 items	first 14	two or more
FULL BFCRS, SCORED 0 TO 3	SCREENING INSTRUMENT (BFCSI)	SCREEN-POSITIVE THRESHOLD (BUSH 1996)

Confirmation, cross-referenced. Once catatonia is identified, the intravenous lorazepam challenge helps confirm it and predicts treatment response: 1 to 2 mg IV over two minutes, with reassessment at 5 minutes and a favourable response usually within 10 minutes (AIIMS 2024). The therapeutics of benzodiazepines and ECT are disorder-specific pointers here; ECT technique points to the ECT and neurostimulation notes.

TRAP **Do not read hypokinetic catatonia as "just severe depression."** A mute, staring, immobile, food-refusing patient is easily filed as retarded depression or an "uncooperative" patient; examine for the elicited signs before you settle for either.

COMMON TRAP

Stuporous (hypokinetic) catatonia hides in plain sight. Because the patient looks passively unwell, teams label it depression, negativistic personality, or non-cooperation and never lay hands on the limbs. Several catatonic signs (waxy flexibility, gegenhalten, mitgehen, catalepsy) exist only when elicited, so a purely observational assessment will systematically miss them.

INDIA

Catatonia remains common in Indian inpatient practice and is a mainstay indication for ECT, which is widely available in government and teaching hospitals; the AIIMS Clinical Methods text codifies the observe-and-elicit examination taught in Indian residencies (AIIMS 2024). Benzodiazepine (lorazepam) first-line and ECT for lorazepam-non-response are the standard Indian pathway, with the lorazepam challenge used at the bedside to confirm.

PEARL

Malignant catatonia (hyperthermia, autonomic instability, rigidity, raised creatine phosphokinase) is clinically near-identical to neuroleptic malignant syndrome and shares management logic; NMS as an entity is owned by the Schizophrenia: management, antipsychotics and adverse effects notes and appears here only as this differential. If antipsychotics were recently given, you cannot separate the two on signs alone: stop the antipsychotic, support in intensive care, and treat as malignant catatonia.

MNEMONIC

WRENCHES-M: Waxy flexibility, Rigidity, Echophenomena, Negativism, Catalepsy/posturing, Automatic obedience/Mitgehen, Stupor/mutism, Stereotypy/mannerism

A rough hook for the sign families across the three ICD-11 clusters; the "M" tail flags mitgehen and mannerisms as the easily forgotten elicited/abnormal-activity signs.

GOOD TO REMEMBER

- **Catatonia:** transdiagnostic psychomotor syndrome; ICD-11 needs three or more signs from decreased, increased and abnormal-activity clusters; coded free-standing (6A40 associated with another disorder).
- **Kahlbaum:** first described catatonia (the "tension insanity") in 1874; hold the eponym and the year.
- **Waxy flexibility (cerea flexibilitas):** even plastic "warm-wax" resistance with the limb held in the new position; the classic catatonic sign, an elicited sign.
- **Catalepsy vs rigidity:** catalepsy is a passively induced posture held against gravity; rigidity is uniform increased tone ("lead-pipe"), no plastic give; waxy flexibility sits between as plastic resistance.
- **Negativism:** motiveless doing-the-reverse of instruction; discriminator from apathy is the active opposition.
- **Mitgehen vs gegenhalten:** mitgehen is anglepoise-lamp over-compliance to slight passive pressure; gegenhalten is force-matched springy counter-resistance; the elicited opposites of obedience/opposition.
- **Echophenomena:** echolalia (mirrors speech), echopraxia (mirrors movement); the discriminator is automatic, uninstructed mimicry.
- **Verbigeration:** senseless directionless repetition of words or phrases; a speech-level stereotypy.
- **Bush-Francis (BFCRS):** 23 items scored 0 to 3; first 14 form the screen; two or more screen-positive signs identify probable catatonia (Bush 1996).
- **Lorazepam challenge:** 1 to 2 mg IV over two minutes, reassess at 5 minutes, favourable response usually within 10 minutes; confirms and predicts response (AIIMS 2024).

14 · Aetiology and the transdiagnostic placement of catatonia

Catatonia is the syndrome that most reliably catches the resident out, because its causes are wide and its nosology has just moved under the examiner's feet. The old teaching, that catatonia is a subtype of schizophrenia, is wrong on both counts: the commonest psychiatric cause is a mood disorder, not schizophrenia, and a large minority of cases are driven by a neurological or medical process that will be missed unless it is actively sought. This is a clinical-disorder topic, so it teaches the two things a viva tests: the *causes* (which three groups, and which is commonest), and the *discrimination* (why an organic cause must be excluded, and where ICD-11 now places the syndrome). The descriptive definitions of the individual catatonic signs (stupor, mutism, negativism, waxy flexibility, echophenomena) are owned by the Psychometry, Assessment and Descriptive Psychopathology notes and are not re-derived here; the schizophrenia disorder itself is owned by the Schizophrenia: aetiology, phenomenology, classification and course notes.

How to use this page. Hold three cause groups (**psychiatric**, **neurological**, **medical**), then one principle (**organic catatonia** must always be excluded in new or atypical presentations), then one nosological shift (**ICD-11** codes catatonia as a **transdiagnostic** syndrome that many conditions can share, not a schizophrenia subtype). The single fact most often swapped is which psychiatric disorder is the commonest cause: it is the *mood* disorders, not schizophrenia (DSM-5-TR 2022).

14.1 The aetiology of catatonia in three groups

Causes are widespread and cut across the whole of medicine. The **aetiology of catatonia** is best carried as three groups (Kaplan & Sadock 2024, ICD-11 CDDR). The first is **psychiatric**: the **mood disorders** (both depressive and bipolar episodes) and schizophrenia and other primary psychotic disorders, with neurodevelopmental disorders (especially autism spectrum disorder) a third psychiatric home. The crucial exam point is that, taken together, the mood disorders are a *commoner psychiatric cause than schizophrenia*: catatonia occurs in up to **35%** of individuals with schizophrenia, but the majority of catatonia cases arise in depressive or bipolar disorders (DSM-5-TR 2022). The second group is a **neurological cause**: encephalitis, in particular anti-NMDA-receptor encephalitis, seizures and the postictal state, stroke (cerebrovascular disease), and space-occupying lesions (neoplasms), alongside head trauma. The third group is **medical causes**: metabolic and electrolyte derangement (diabetic ketoacidosis, hypercalcaemia, hyponatraemia, hepatic encephalopathy, homocystinuria), infection (typhoid, HIV, infectious mononucleosis), autoimmune disease (systemic lupus erythematosus, Hashimoto/autoimmune encephalopathy, PANDAS), and drug-related or withdrawal states (antipsychotics, benzodiazepines, steroids, disulfiram, ciprofloxacin; and intoxication with or withdrawal from PCP, cannabis, cocaine, MDMA, LSD or opioids) (ICD-11 CDDR).

GROUP	REPRESENTATIVE CAUSES	THE ONE POINT TO HOLD
Psychiatric	Mood disorders (depressive and bipolar); schizophrenia and other primary psychotic disorders; autism spectrum disorder	**The *mood disorders are the commonest psychiatric cause*, not schizophrenia; catatonia in up to 35% of schizophrenia but most cases are mood**
Neurological cause	Encephalitis (esp. anti-NMDA-receptor encephalitis); seizures and the postictal state; stroke; space-occupying lesions; head trauma	**Anti-NMDA-receptor encephalitis is the archetypal treatable neurological cause and classically presents with psychiatric features then catatonia**
Medical causes	Metabolic and electrolyte derangement; infection; autoimmune disease; drug-related or withdrawal states	**Any of these produces catatonia indistinguishable from the psychiatric form; screening bloods, imaging and (if encephalitis is suspected) lumbar puncture are how they are found**

The three cause groups of catatonia; the trap is to stop at the psychiatric group and, within it, to assume schizophrenia rather than a mood disorder (Kaplan & Sadock 2024, DSM-5-TR 2022, ICD-11 CDDR).

RULE **Always exclude a medical or neurological cause of new catatonia, and remember the mood disorders are the commonest psychiatric cause.** New or atypical catatonia is organic until proven otherwise; among psychiatric causes, think mood first, not schizophrenia.

TRAP **Assuming catatonia means schizophrenia.** Catatonic signs are nonspecific: the commonest psychiatric cause is a mood disorder, and a substantial share of cases are neurological or medical; treating catatonia as pathognomonic of schizophrenia misses both the mood disorder and the organic cause.

14.2 The organic catatonia principle

Exclude a physical cause before you land on a psychiatric one. The **organic catatonia** principle is the governing safety rule of the syndrome: a medical or neurological cause must *always* be excluded, especially in new or atypical catatonia, because so many treatable physical illnesses reproduce it exactly and because an untreated organic driver can be lethal (Kaplan & Sadock 2024). Several pointers raise the prior for an organic cause: a first-ever episode, an atypical picture, older age (secondary catatonia syndrome becomes more likely after 40 and rises sharply after 65), very early onset (before 20, which associates with nervous-system disease or a neurodevelopmental disorder), co-occurring delirium or fluctuating consciousness, focal neurological signs, or autonomic instability. Catatonia itself has *no* specific laboratory, imaging or EEG signature, so the workup is aimed at the underlying cause and at complications: complete blood count, a complete metabolic panel, serum creatine kinase (markedly raised in malignant catatonia and NMS, and a warning of rhabdomyolysis), neuroimaging for a lesion, EEG (diffuse slowing points to delirium or encephalitis rather than a primary psychiatric cause), and, where encephalitis is suspected, a lumbar puncture with an autoimmune-encephalitis panel (Kaplan & Sadock 2024). If the entire workup is unremarkable, a psychiatric cause becomes the likely explanation, but only by exclusion.

WORKED EXAMPLE

A previously well 24-year-old woman is admitted with two weeks of mutism, negativism and waxy flexibility, preceded by headache, low-grade fever and a few days of odd, fearful behaviour. She meets the criteria for catatonia. Because this is a *first, atypical* episode in a young person with a prodrome suggesting encephalitis, the organic catatonia principle applies: alongside a lorazepam challenge for the catatonia, she needs neuroimaging and a lumbar puncture with an autoimmune-encephalitis panel. Anti-NMDA-receptor encephalitis is the target diagnosis (it classically opens with psychiatric symptoms and catatonia before seizures and dysautonomia appear), and immunotherapy, not an antipsychotic, is the definitive treatment. Reaching for schizophrenia here would be the cardinal error.

14.3 The ICD-11 transdiagnostic placement

Catatonia is now a syndrome of its own, not a schizophrenia subtype. The important nosological shift is that **ICD-11** treats catatonia as a **transdiagnostic** syndrome of primarily psychomotor disturbance that **catatonia can accompany** many conditions (ICD-11 CDDR). It is given its own grouping with four categories rather than being buried as a form of schizophrenia:

6A40 catatonia associated with another mental disorder (the associated disorder, whether a mood disorder, schizophrenia or another primary psychotic disorder, or autism spectrum disorder, is coded separately), 6A41 catatonia induced by substances or medications, 6E69 secondary catatonia syndrome (the direct pathophysiological consequence of a medical or neurological condition), and 6A4Z catatonia, unspecified. So the syndrome sits above any single diagnosis: it is defined once (three or more psychomotor symptoms from decreased, increased or abnormal activity) and then attributed to whatever underlies it. This is the important shift from the older ICD-10 view, in which catatonic schizophrenia (F20.2) was a named schizophrenia subtype and the organic form (F06.1, organic catatonic disorder, which maps onto ICD-11's 6E69) sat apart; ICD-11 dissolves the subtype and makes the transdiagnostic syndrome primary (ICD-11 CDDR).

PEARL

The examiner's favourite one-liner is the direction of the shift. ICD-10 had a *catatonic subtype of schizophrenia* (F20.2); ICD-11 abolishes the schizophrenia subtypes and codes catatonia as a standalone transdiagnostic syndrome (6A40 / 6A41 / 6E69 / 6A4Z) that can accompany many disorders. If asked "where does catatonia live in ICD-11?", the answer is "its own grouping, coded to whatever it accompanies", not "under schizophrenia".

14.4 DSM-5-TR: a specifier and an associated syndrome, not a subtype

DSM-5-TR reaches the same destination by a different route. DSM-5 similarly stopped treating catatonia as a subtype of schizophrenia and instead handles it three ways: as a *specifier* ("with catatonia") that can be appended to a neurodevelopmental, psychotic, bipolar or depressive disorder; as **catatonia associated with another mental disorder** (the catatonia specifier, coded F06.1) when it is the focus; and as two standalone entities, *catatonic disorder due to another medical condition* and *unspecified catatonia* (DSM-5-TR 2022). Catatonia is defined by three or more of twelve psychomotor features. DSM-5-TR is explicit that although catatonia has historically been associated with schizophrenia, the symptoms are *nonspecific* and occur across mood, neurodevelopmental and medical conditions, and that "the majority of catatonia cases involve individuals with depressive or bipolar disorders" (DSM-5-TR 2022). So both major systems now converge: catatonia is a transdiagnostic syndrome, commonest in mood disorders, and never assumed to mean schizophrenia.

SYSTEM	HOW CATATONIA IS PLACED	THE DISCRIMINATING POINT
ICD-10 (older view)	Catatonic schizophrenia F20.2 as a named subtype; organic catatonic disorder F06.1 separate	**Catatonia partly owned *within* schizophrenia as a subtype: the view now superseded**
ICD-11	Own grouping: 6A40 associated with another mental disorder, 6A41 substance/medication-induced, 6E69 secondary catatonia syndrome, 6A4Z unspecified	**A transdiagnostic syndrome coded to whatever it accompanies; schizophrenia subtypes abolished**
DSM-5-TR	"With catatonia" specifier; catatonia associated with another mental disorder (F06.1); catatonic disorder due to another medical condition; unspecified catatonia	**A specifier or an associated syndrome, *not* a subtype; 3 of 12 features; explicitly nonspecific**

The nosological placement of catatonia across systems: the shared modern message is transdiagnostic, and the ICD-10 catatonic-schizophrenia subtype is the view being left behind (ICD-11 CDDR, DSM-5-TR 2022).

14.5 Why the placement changed: the aetiological and neurobiological logic

The nosology followed the biology and the epidemiology. Two lines of evidence made the schizophrenia subtype untenable. The first is epidemiological: once catatonia was rated systematically (with scales such as the Bush-Francis Catatonia Rating Scale, Bush 1996), it was found across mood, neurodevelopmental, medical and neurological conditions, with mood disorders supplying more cases than schizophrenia (a clinical-sample meta-analysis put the pooled prevalence of catatonia at about **9%**) (Bush 1996, DSM-5-TR 2022). The second is neurobiological: catatonic features arise from disruption of cortical-subcortical (basal-ganglia-thalamocortical) circuits regulating cortical function, producing reduced dopaminergic and GABA-A activity and upregulation of NMDA activity, and this final common pathway can be reached from many upstream causes (Kaplan & Sadock 2024). The same model explains the diagnostic-and-therapeutic lorazepam challenge (a GABA-A agonist disinhibits striatal dopamine and reverses the state) and why glutamate antagonists such as amantadine help in refractory cases. A shared circuit-level endpoint entered from many directions is exactly a transdiagnostic syndrome, which is what the classifications now encode.

up to 35%	**~9%**	**3 of 12**
OF SCHIZOPHRENIA SHOWS CATATONIA	POOLED PREVALENCE IN CLINICAL SAMPLES	FEATURES REQUIRED (DSM-5-TR)

14.6 The one differential the placement forces: malignant catatonia versus NMS

The organic-exclusion rule has one high-stakes special case. When catatonia is accompanied by autonomic instability and hyperthermia it becomes malignant catatonia, a life-threatening state that carried a **75% to 100%** mortality before modern treatment and around **10%** with benzodiazepines and ECT (Kaplan & Sadock 2024). Its near-twin is neuroleptic malignant syndrome, which shares rigidity, hyperthermia, dysautonomia and a raised creatine kinase; the practical distinction is the drug history (NMS follows dopamine-antagonist exposure or

dopamine-agonist withdrawal), and the two are increasingly regarded as variants of the same catatonic spectrum. NMS as an entity is owned by the Schizophrenia: management, antipsychotics and adverse effects notes and is taught in full there; it appears here only as the differential that malignant catatonia forces. The management principle is that antipsychotics can worsen catatonia and precipitate the malignant form, so in a catatonic patient they are used cautiously and covered by lorazepam; the ECT technique itself points to the ECT and neurostimulation notes.

TRAP **Mistaking malignant catatonia for NMS (or giving a catatonic patient an antipsychotic).** The two overlap almost completely; the drug history separates them, and reaching for an antipsychotic in catatonia can tip a patient into the malignant, potentially fatal form.

INDIA

Catatonia is a familiar presentation in Indian practice, where a substantial share is mood-related and organic causes (post-infective, metabolic, autoimmune encephalitis) are actively sought given the local infectious and nutritional burden; the ICD-11 (and long-familiar ICD-10) framework is standard in Indian teaching texts. A practical advantage is that ECT, the definitive treatment for severe and malignant catatonia, is comparatively well established and available across Indian psychiatric units, and lorazepam is cheap and generic, so the diagnostic-therapeutic lorazepam challenge is readily performed at the bedside. The system-wide rule that no diagnosis is named to the patient sits comfortably here: the family is engaged around the psychomotor state and its urgency rather than a label. Where a brand is needed, lead with the Indian generic and verify locally rather than inventing one.

GOOD TO REMEMBER

- **Aetiology of catatonia (three groups):** psychiatric (mood disorders and schizophrenia/primary psychotic disorders, plus autism), neurological (encephalitis including anti-NMDA-receptor encephalitis, seizures/postictal state, stroke, space-occupying lesions) and medical (metabolic/electrolyte, infective, autoimmune, drug/withdrawal); the number to hold is that the mood disorders are the commonest psychiatric cause, with catatonia in up to 35% of schizophrenia but most cases being mood.
- **Organic catatonia principle:** a medical or neurological cause must always be excluded, especially in new or atypical catatonia; the discriminator prompting a physical workup is a first, atypical, older-onset or delirium-associated episode; the workup to hold is CBC, CMP, creatine kinase, neuroimaging, EEG and (if encephalitis suspected) lumbar puncture.
- **Anti-NMDA-receptor encephalitis:** the archetypal treatable neurological cause; the discriminator is a young patient with a psychiatric-then-catatonic prodrome progressing to seizures and dysautonomia; the point to hold is that CSF autoimmune-encephalitis testing and immunotherapy, not an antipsychotic, are definitive.
- **ICD-11 transdiagnostic placement:** catatonia is its own grouping, coded to whatever it accompanies; the discriminator from the old view is that the schizophrenia subtype (ICD-10 F20.2) is abolished; the codes to hold are 6A40 (associated with another mental disorder), 6A41 (substance/medication-induced), 6E69 (secondary catatonia syndrome) and 6A4Z (unspecified).
- **DSM-5-TR placement:** a "with catatonia" specifier, catatonia associated with another mental disorder (F06.1), catatonic disorder due to another medical condition and unspecified catatonia; the discriminator is that it is a specifier or associated syndrome, not a subtype; the number to hold is 3 of 12 psychomotor features.
- **Malignant catatonia:** catatonia plus hyperthermia and autonomic instability; the discriminator from NMS is the drug history (NMS follows dopamine antagonism); the number to hold is the mortality, 75% to 100% untreated, about 10% with benzodiazepines and ECT.

HOLD THIS

Catatonia is a transdiagnostic psychomotor syndrome, not a schizophrenia subtype: its commonest psychiatric cause is a mood disorder (catatonia occurs in up to 35% of schizophrenia but most cases are mood), it is frequently neurological or medical (encephalitis, especially anti-NMDA-receptor encephalitis, seizures, stroke, metabolic, autoimmune, drug or withdrawal), so a medical or neurological cause must always be excluded in new or atypical catatonia; ICD-11 codes it in its own grouping (6A40 / 6A41 / 6E69 / 6A4Z) that catatonia can accompany many conditions, and DSM-5-TR treats it as a specifier or associated syndrome rather than a subtype.

15 · Catatonia management: the lorazepam challenge and benzodiazepines

Why this matters. Catatonia is the one psychiatric syndrome where a single bedside manoeuvre is at once the diagnostic test and the first dose of treatment. The **lorazepam challenge** lets you confirm the syndrome and start reversing it in the same act, and the examiner rewards the resident who states the drug, the route, the dose and the response window, and who knows why a high-potency antipsychotic is the wrong reflex. The raw descriptive signs of catatonia (stupor, mutism, negativism, waxy flexibility, echophenomena) are set out in the Psychometry, Assessment and Descriptive Psychopathology notes and are not re-derived here; this topic teaches what you *do* once the syndrome is recognised.

The governing principle is simple and heavily tested: benzodiazepines, opened by the lorazepam challenge and then titrated, are first-line for catatonia regardless of the underlying cause, and

antipsychotics are the classic trap. Two facts anchor the topic: a **benzodiazepine** reduces catatonic symptoms in roughly 70% to 80% of patients (Kaplan & Sadock 2024), and the antipsychotic reflex can worsen catatonia or tip it into malignant catatonia. Supportive care and treatment of the cause run alongside from the outset; ECT is the definitive treatment where the drug fails or the picture is malignant, with its technique cross-referenced to the ECT and neurostimulation notes.

15.1 The lorazepam challenge: one act, two purposes

The lorazepam challenge is a test dose of **lorazepam** given to a patient with suspected catatonia, watched for a rapid reduction in catatonic features. It is diagnostic and therapeutic in the same stroke: a positive response (increased interaction, reduction of catatonic features) is confirmatory of catatonia and is simultaneously the first therapeutic dose (Kaplan & Sadock 2024). The standard adult dose is **2 mg**, commonly quoted as **1 to 2 mg** given intravenously or intramuscularly; IV is preferred for its quick onset and because a stuporous patient may be unable to swallow the oral formulation, with IM or sublingual used when IV access is unavailable (Kaplan & Sadock 2024). Lower doses suit paediatric and frail older adults, but 2 mg is recommended for most adults to avoid an equivocal result.

The **response** is often dramatic and quick: a robust "Lazarus" response may be seen within 30 minutes of a 2 mg dose, though some patients need up to 120 minutes; the AIIMS bedside protocol gives 1 to 2 mg IV over two minutes and reassesses at 5 to 10 minutes (Kaplan & Sadock 2024, Clinical Methods in Psychiatry AIIMS 2024). Interpretation has a trap of its own: a positive response confirms catatonia, but a negative response (sedation, or no change) does *not* rule it out, so if suspicion stays high a further dose is given and the patient reassessed. Monitor the change with a standardised scale rather than by impression.

RULE **The lorazepam challenge is diagnosis and treatment in one.** A test dose of lorazepam (2 mg, IV preferred) that produces a marked partial or full response both confirms catatonia and begins reversing it.

TRAP **Reading a negative challenge as excluding catatonia.** A positive response confirms the syndrome; sedation or no change does not rule it out. If clinical suspicion remains, repeat the dose rather than abandon the diagnosis.

15.2 Benzodiazepines as first-line: titration after the challenge

Once the challenge is positive, **lorazepam** is the first-line treatment irrespective of aetiology, carrying an efficacy of **70% to 80%** for symptom reduction (Kaplan & Sadock 2024). The drug is then scheduled and titrated upward, often to higher-than-usual doses. A typical regimen schedules lorazepam (for example 2 mg every 6 hours) for at least the first 24 to 48 hours, moving to a titrated total daily dose that controls symptoms without excessive sedation; some patients need a total of 30 mg or more per day (Kaplan & Sadock 2024). Nursing must give scheduled doses even when the patient appears to sleep, because that "sleep" may be stupor returning as the last dose wears off. Once symptoms have clearly improved, taper slowly (a 10% to 25% reduction per day).

Second-line and adjunctive agents exist but are not the answer to hold first: zolpidem 5 to 10 mg, GABA-active antiepileptics (valproate, topiramate, carbamazepine), and NMDA-receptor antagonists (amantadine 100 to 600 mg/day, memantine 10 to 20 mg/day) are used as adjuncts to lorazepam rather than as monotherapy, chiefly in lorazepam-refractory cases (Kaplan & Sadock 2024).

RULE **Benzodiazepines, not antipsychotics, are first-line for catatonia.** Lorazepam challenge, then titration (often to higher-than-usual doses), reverses roughly 70% to 80% of cases whatever the cause.

15.3 Supportive care and treating the cause

Catatonia is a medical emergency of immobility, so supportive care runs in parallel from the outset and is examinable in its own right. The stuporous, immobile patient is at risk of dehydration, malnutrition, aspiration pneumonia, pressure sores, contractures, urinary retention, rhabdomyolysis with renal failure, and venous thromboembolism. Management therefore includes hydration and nutrition (IV fluids, nasogastric or PEG feeding), VTE prophylaxis (anticoagulation), pressure care and mobilisation, aspiration precautions, urinary catheterisation, and monitoring of vitals and fluid balance (Kaplan & Sadock 2024, Clinical Methods AIIMS 2024). In parallel, the underlying cause is investigated and treated: catatonia is transdiagnostic, so a medical, neurological or substance cause must be actively excluded and managed, and treatment must not be delayed merely because the cause has not yet been found. Serum creatine kinase is tracked because marked elevation flags malignant catatonia or neuroleptic malignant syndrome and predicts renal risk.

WORKED EXAMPLE

A young man with schizophrenia is mute, immobile and holds imposed postures; he is not eating or drinking. You give 2 mg lorazepam IV over two minutes and at 8 minutes he begins to speak and move: the challenge is positive, confirming catatonia and starting treatment. You schedule lorazepam 2 mg every 6 hours, start IV fluids, VTE prophylaxis and pressure care, send CK and a metabolic screen, and arrange ECT cover in case of incomplete response. You do *not* reach for haloperidol to treat his psychosis while he is catatonic.

15.4 The antipsychotic caution: precipitating malignant catatonia

Antipsychotics were historically used to treat catatonia, but in many cases this worsened symptoms and increased mortality (Kaplan & Sadock 2024). The mechanistic reason fits the model: catatonia involves reduced GABA-A activity and a resulting hypodopaminergic state, so a dopamine-blocking drug can deepen the very dysfunction that drives the syndrome. The clinical danger is that a high-potency antipsychotic given to a catatonic patient can precipitate malignant catatonia or neuroleptic malignant syndrome (NMS), both life-threatening. Where the underlying psychosis genuinely needs antipsychotic cover, it should be given alongside lorazepam, and a second-generation agent, especially one with partial D2-agonist activity such as aripiprazole, is preferred over a high-potency D2 blocker (Kaplan & Sadock 2024). The drug detail and NMS as

an entity are owned by the Schizophrenia: management, antipsychotics and adverse effects notes; here NMS appears only as the malignant-catatonia differential.

TRAP **Giving a high-potency antipsychotic to a catatonic patient.** It can fail to help, worsen the catatonia, and precipitate malignant catatonia or neuroleptic malignant syndrome. Treat the catatonia with a benzodiazepine first; if antipsychotic cover is needed, add it to lorazepam and prefer a partial D2 agonist.

15.5 Malignant catatonia versus NMS, and where ECT comes in

Malignant catatonia (the older terms lethal catatonia and malignant catatonia are used interchangeably) is catatonic stupor accompanied by hyperthermia and autonomic instability, a potentially fatal state that on presentation may be clinically and biochemically indistinguishable from neuroleptic malignant syndrome, which has led to the view that NMS is best regarded as a drug-induced variant of malignant catatonia (Maudsley Prescribing Guidelines 2021, DSM-5-TR 2022). The practical consequence for management is that antipsychotics must be stopped and benzodiazepines and ECT brought forward urgently. **ECT** is the definitive treatment: it is the gold-standard for catatonia with about 90% efficacy, has no absolute contraindications, and is the treatment of choice when the patient is lorazepam-refractory or has progressed to malignant catatonia (Kaplan & Sadock 2024). Because malignant catatonia can evolve fast, ECT should be considered and consent sought early rather than after the drug has clearly failed. The ECT technique itself is set out in the ECT and neurostimulation notes and is not taught here.

FEATURE	MALIGNANT CATATONIA	NEUROLEPTIC MALIGNANT SYNDROME
Nature	Severe catatonia with hyperthermia and autonomic instability	**Antipsychotic-induced; often viewed as a drug-triggered variant of malignant catatonia**
Trigger	**Any cause of catatonia; may arise without any drug**	Onset after starting or increasing a dopamine antagonist
Core signs	Stupor, rigidity, fever, autonomic instability; raised CK	Fever, "lead-pipe" rigidity, altered mental state, autonomic instability; raised CK
The offending drug	**Antipsychotics can precipitate or worsen it: stop them**	Antipsychotic is the cause: withdraw immediately
Treatment overlap	Benzodiazepines and ECT; stop antipsychotics	**Supportive care, dantrolene/bromocriptine, benzodiazepines; ECT if refractory (see the Schizophrenia: management, antipsychotics and adverse effects notes)**

Malignant catatonia and NMS may be indistinguishable at the bedside; the discriminator is the drug history, and either way antipsychotics come off and benzodiazepines plus ECT go on. NMS as an entity is owned by the Schizophrenia: management, antipsychotics and adverse effects notes.

TRAP **Chasing the malignant-catatonia versus NMS label instead of acting.** The two overlap so closely they may be one spectrum; do not delay stopping the antipsychotic and starting benzodiazepines and ECT while you argue the name.

15.6 ICD-11 and the transdiagnostic frame

The nosology carries a high-yield point that shapes management. ICD-11 treats catatonia as a separate transdiagnostic grouping of primarily psychomotor disturbance that can arise across schizophrenia and other primary psychotic disorders, mood disorders, neurodevelopmental disorders (especially autism), with substances or medications, and with medical conditions (ICD-11 CDDR). The grouping codes are 6A40 catatonia associated with another mental disorder, 6A41 catatonia induced by substances or medications, and 6E69 secondary catatonia syndrome (a medical cause), with 6A4Z unspecified. Because catatonia crosses diagnoses, the management principle is the same whatever the parent: treat the syndrome with a benzodiazepine and, where needed, ECT, while identifying and treating the cause. DSM-5-TR maps the same idea through a "with catatonia" specifier and catatonia due to another medical condition; ICD-10 lacked the clean transdiagnostic grouping and is flagged as the older frame.

INDIA

Indian practice fits the standard picture and adds two local realities. The lorazepam-based approach is the AIIMS bedside standard: 1 to 2 mg IV over two minutes, reassessed at 5 to 10 minutes, with at least 6 mg given over 24 hours if the response is partial, then scheduled IV lorazepam continued (Clinical Methods in Psychiatry AIIMS 2024). Lorazepam is widely available and inexpensive, so benzodiazepine-first is fully practicable. ECT is comparatively accessible and heavily used in Indian tertiary centres, and modified ECT (mECT) is explicitly recommended as an urgent, life-saving intervention in malignant catatonia, with consent sought as early as possible; maintenance mECT is used in some cases (Clinical Methods AIIMS 2024). The 2025 Indian Psychiatric Society schizophrenia Clinical Practice Guideline (IPS 2025, Sahoo et al., Indian J Psychiatry 2026;68:94-121) is concordant: it endorses the lorazepam challenge test as serving both a diagnostic and a therapeutic purpose in catatonia, with benzodiazepines (lorazepam or clonazepam) used short-term and their long-term use avoided because of tolerance and dependence risk. This material is largely generic; lorazepam is prescribed by generic name.

70% to 80% RESPOND TO A BENZODIAZEPINE (LORAZEPAM), FIRST-LINE	**2 mg** STANDARD ADULT LORAZEPAM CHALLENGE DOSE (1 TO 2 MG IV/IM)	**~90%** ECT EFFICACY, THE DEFINITIVE TREATMENT

MNEMONIC

CHALLENGE, THEN CLIMB, NEVER BLOCK

CHALLENGE: 2 mg lorazepam confirms and starts treatment. CLIMB: titrate the benzodiazepine up, often to higher-than-usual doses, with supportive care and cause-treatment alongside. NEVER BLOCK: avoid a high-potency antipsychotic, which can precipitate malignant catatonia or NMS; ECT is the definitive step.

HOLD THIS

Catatonia is treated benzodiazepine-first: a 2 mg lorazepam challenge that produces a rapid response both confirms the diagnosis and begins treatment, then lorazepam is titrated up (roughly 70% to 80% respond); antipsychotics are the trap because they can precipitate malignant catatonia or NMS, and ECT is the definitive treatment.

GOOD TO REMEMBER

- **Lorazepam challenge:** a test dose of lorazepam, **2 mg** (1 to 2 mg IV or IM), watched for a rapid partial or full response that both confirms catatonia and starts treatment; a negative response does not exclude catatonia; response often within 30 minutes ("Lazarus" response).
- **Benzodiazepine, first-line:** lorazepam titrated up after a positive challenge, often to higher-than-usual doses (up to 30 mg/day or more); efficacy for symptom reduction is **70% to 80%** (Kaplan & Sadock 2024).
- **Antipsychotic caution:** may not help and can worsen catatonia or precipitate malignant catatonia or NMS; if antipsychotic cover is needed, add it to lorazepam and prefer a partial D2 agonist (aripiprazole).
- **Malignant catatonia:** catatonic stupor with hyperthermia and autonomic instability, potentially fatal; may be indistinguishable from NMS (which is viewed as a drug-induced variant); stop antipsychotics, give benzodiazepines and urgent ECT.
- **ECT:** the definitive, gold-standard treatment, about **90%** efficacy, for lorazepam-refractory or malignant catatonia; technique in the ECT and neurostimulation notes.
- **Bush-Francis Catatonia Rating Scale:** the 23-item scale (Bush 1996) used to identify and monitor catatonic signs and to track response to the challenge and titration.
- **ICD-11 placement:** catatonia is a separate transdiagnostic grouping, 6A40 (with another mental disorder), 6A41 (substance/medication-induced), 6E69 (secondary), so the management principle holds across every parent diagnosis.

16 · Malignant catatonia and the differential from NMS

Why this matters. This is the emergency at the far end of the catatonia spectrum and the closer of the schizophrenia-spectrum block: an untreated catatonia that tips into fever, autonomic storm and rigidity is a life-threatening state with a high mortality if it is missed. The examiner's favourite pivot here is the differential from neuroleptic malignant syndrome, because the two are so alike that many authors treat them as one entity, and the resident who can say what leans the picture one way or the other, and who knows that the first move is the same for both, scores. The raw descriptive catatonic signs (stupor, mutism, negativism, waxy flexibility) are set out in the Psychometry, Assessment and Descriptive Psychopathology notes and are not re-derived here.

Two things carry the topic. First, malignant catatonia is a diagnosis of the whole picture, not of a single sign: it is catatonia complicated by hyperthermia and autonomic instability, and it can grow out of an ordinary catatonia that was not treated in time. Second, the differential from NMS is more a matter of leaning than of a clean line, and the safe reflex is identical whichever way you lean: stop any antipsychotic, give benzodiazepines, and move to ECT early. The neuroleptic malignant syndrome as an entity is owned by the Schizophrenia: management, antipsychotics and adverse effects notes and appears here only for this differential; the ECT technique points to the ECT and neurostimulation notes.

	Malignant catatonia (lethal catatonia, Stauder 1934)	Neuroleptic malignant syndrome (owned by the management notes)
Preceding picture	catatonic signs come first	on recent antipsychotic
Drug trigger	none needed (may follow catatonia)	antipsychotic start or dose rise
Rigidity	rigidity within a catatonic picture	lead-pipe rigidity
Fever, autonomic	present (shared)	present (shared)
Creatine kinase	raised (shared)	raised, often marked (shared)
First move (same)	stop the drug, benzodiazepines, ECT	identical: stop, benzodiazepines, ECT

Almost every cross-sectional feature is shared; the preceding picture and drug history lean the label, and NMS is often held to be a drug-induced malignant catatonia.

Fig 10.8 Malignant catatonia versus neuroleptic malignant syndrome. Fever, rigidity, autonomic instability and a raised creatine kinase are shared, so the discriminators are the preceding picture (catatonic signs first, without a necessary antipsychotic trigger, lean to malignant catatonia; a recent antipsychotic with lead-pipe rigidity leans to NMS). The first management move is the same for both: stop the antipsychotic, give benzodiazepines, and move to ECT early.

16.1 The entity: catatonia plus fever, dysautonomia and rigidity

Malignant catatonia, historically named lethal catatonia, is catatonic stupor accompanied by hyperthermia and autonomic instability, a potentially fatal emergency (Maudsley Prescribing Guidelines 2021). The fatal, hyperpyrexial form of catatonia was described by Karl Heinz Stauder at the Munich University Psychiatric Hospital in 1934 (Kaplan & Sadock 2024), which is why the older literature speaks of Stauder's lethal catatonia. The defining move from ordinary to malignant catatonia is the arrival of autonomic and thermoregulatory failure: on top of the immobility, mutism, posturing and rigidity of catatonia the patient develops fever, labile blood pressure and pulse, tachycardia, sweating, and often a raised serum creatine kinase. It is important that this is not always a fresh presentation: a catatonia that was recognised late or treated inadequately can progress into malignant catatonia, so the state is partly a failure to intervene in time (Kaplan & Sadock 2024).

The **stakes** are what make it examinable as an emergency. Untreated, malignant catatonia carries a high mortality, historically very high before benzodiazepines and ECT, driven by the complications of prolonged immobility and dysautonomia: hyperthermia, dehydration and malnutrition, rhabdomyolysis with acute renal failure, aspiration pneumonia, venous thromboembolism and cardiovascular collapse (Kaplan & Sadock 2024). The practical reading is that malignant catatonia is treated as a medical emergency in parallel with, not after, the diagnostic workup, and that recognising the febrile, rigid, autonomically unstable catatonic patient early is the whole game.

RULE **Fever plus autonomic instability turns catatonia into an emergency.** Malignant catatonia is catatonic stupor complicated by hyperthermia and dysautonomia, it can evolve from an untreated catatonia, and it kills if it is not treated.

16.2 The overlap with NMS: two names, arguably one entity

The heart of the topic is the differential from neuroleptic malignant syndrome, the NMS. The problem is that the two states share almost every feature: fever, rigidity, altered consciousness, autonomic instability and a raised creatine kinase all appear in both, and malignant catatonia may not be distinguishable from NMS either clinically or by laboratory testing (Maudsley Prescribing Guidelines 2021). This near-identity has produced a genuine nosological view rather than a mere resemblance: a symposium at the American Psychiatric Association meeting in 2000 concluded that NMS and malignant catatonia were the same (Kaplan & Sadock 2024), and the widely quoted formulation is that NMS is best regarded as a drug-induced form of malignant catatonia, that is, a malignant catatonia whose trigger happens to be a dopamine antagonist (Maudsley Prescribing Guidelines 2021). DSM-5-TR lists the two together precisely as a differential-diagnostic pair, malignant catatonia versus neuroleptic malignant syndrome (DSM-5-TR 2022).

Because the states overlap, the **discriminating leaning** rests less on the cross-sectional signs than on the preceding picture and the drug history. Catatonic signs that came first, in a patient who has not necessarily had an antipsychotic trigger, favour malignant catatonia: NMS can probably be ruled out in the absence of any prior or recent administration of a dopamine antagonist (Maudsley Prescribing Guidelines 2021). Conversely, a recent antipsychotic start or a dose rise, with lead-pipe rigidity emerging in that context, favours NMS. The leaning is a lean, not a proof, and the examiner wants it framed that way: the drug history tilts the label, the two may in truth be one spectrum, and the management does not wait on the argument.

TRAP **Treating malignant catatonia and NMS as cleanly separable.** They overlap so closely that they may be one entity; the drug history only leans the label, and chasing the name must never delay stopping the antipsychotic and starting benzodiazepines and ECT.

16.3 The differential grid: what leans which way

The discriminators are best held as a grid, because the point is exactly which features overlap (almost all of them) and which few lean the diagnosis. The shared column is large: fever, rigidity, autonomic instability and raised creatine kinase sit in both. The leaning column is small and is dominated by the temporal and pharmacological sequence.

FEATURE	LEANS TO MALIGNANT CATATONIA	LEANS TO NMS
Preceding picture	**Catatonic signs first (stupor, mutism, negativism, posturing), then fever and dysautonomia**	Rigidity and fever emerge on a background of recent antipsychotic exposure
Drug trigger	**No antipsychotic trigger necessary; may arise from an untreated catatonia**	**Recent antipsychotic start or dose rise (a dopamine antagonist)**
Character of the rigidity	Rigidity within a catatonic picture (posturing, waxy flexibility may be present)	**"Lead-pipe" rigidity is the classic descriptor**
Fever and autonomic instability	Present (defining the malignant state)	Present (shared, non-discriminating)
Creatine kinase	Often raised	Often markedly raised (shared, non-discriminating)
Nosological view	The primary entity	**Often regarded as a drug-induced form of malignant catatonia**
First management move	Stop any antipsychotic, benzodiazepines, ECT early	**Identical: stop the antipsychotic, benzodiazepines, ECT early (see the Schizophrenia: management, antipsychotics and adverse effects notes)**

The malignant catatonia versus NMS differential: almost every cross-sectional feature is shared, so the discriminators are the preceding picture and the antipsychotic history, and the first management move is the same either way.

16.4 Management: stop the drug, benzodiazepines, ECT early, intensive care

The management is deliberately the same whichever side of the differential the patient sits, because the states overlap and delay is what kills. The steps are: **stop any antipsychotic** (it can precipitate or worsen the state, and if the diagnosis is NMS the antipsychotic is the cause); **give benzodiazepines**, with lorazepam first-line and the lorazepam challenge as the entry point; **move to ECT early**, which treats both malignant catatonia and NMS and is the definitive step; and run **intensive supportive care** in parallel (hydration and nutrition, cooling, VTE prophylaxis, monitoring of vitals, creatine kinase and renal function, aspiration precautions) (Maudsley Prescribing Guidelines 2021, Kaplan & Sadock 2024). ECT is the gold-standard treatment for catatonia with about **90%** efficacy (Kaplan & Sadock 2024), and because malignant catatonia can evolve fast it should be considered and consent sought early rather than after benzodiazepines have clearly failed. In malignant catatonia every effort is made to maximise the effect of ECT by using liberal stimulus dosing to induce well-generalised seizures (Maudsley Prescribing Guidelines 2021); the technique itself is set out in the ECT and neurostimulation notes and is not taught here.

WORKED EXAMPLE

A woman with a two-week history of progressive mutism, immobility and imposed posturing, never started on an antipsychotic, now spikes a temperature of 39.5 C with labile blood pressure, tachycardia and a creatine kinase of several thousand. The catatonic picture came first and there is no dopamine antagonist in the history, so this leans to malignant catatonia rather than NMS, but the management does not depend on settling the label: no antipsychotic is given, lorazepam is started (2 mg challenge, then scheduled), ECT is arranged urgently, and she is managed with IV fluids, cooling, VTE prophylaxis and renal monitoring on a medical or high-dependency setting. Had she instead deteriorated days after a haloperidol start with lead-pipe rigidity, the lean would be to NMS, and the first moves would be identical.

RULE **Malignant catatonia and NMS overlap, so treat both the same.** Stop the antipsychotic, reach for benzodiazepines, and move to ECT early, with intensive supportive care alongside.

TRAP **Giving more antipsychotic to a febrile, rigid, catatonic patient.** Adding or increasing a dopamine antagonist here can precipitate or deepen malignant catatonia or NMS; the antipsychotic comes off, it does not go up.

~90%	2 mg	1934
ECT EFFICACY IN CATATONIA, THE DEFINITIVE STEP, TREATS BOTH	LORAZEPAM CHALLENGE, BENZODIAZEPINE ENTRY POINT	STAUDER DESCRIBES FATAL HYPERPYREXIAL (LETHAL) CATATONIA

INDIA

Indian tertiary practice fits the standard picture and is well placed for it. Lorazepam is widely available and inexpensive, so a benzodiazepine-first approach is fully practicable, and ECT is comparatively accessible and heavily used in Indian centres. Modified ECT is explicitly recommended as an urgent, life-saving intervention in malignant catatonia, with consent sought as early as possible (Clinical Methods in Psychiatry AIIMS 2024). The message for the Indian resident is that the definitive treatment for this emergency is within reach and should not be delayed while the malignant catatonia versus NMS label is debated.

MNEMONIC

STOP, SEDATE, SHOCK

STOP the antipsychotic (it can precipitate or is the cause). SEDATE with benzodiazepines (lorazepam, the challenge then titration). SHOCK, that is ECT, early, because it treats both malignant catatonia and NMS. All three run with intensive supportive care.

HOLD THIS

Malignant (lethal) catatonia is catatonic stupor plus fever and autonomic instability, and it overlaps so completely with NMS that they may be one entity; the drug history only leans the label (catatonia-first without a drug leans malignant catatonia, a recent antipsychotic with lead-pipe rigidity leans NMS), so for both you stop the antipsychotic, give benzodiazepines, and move to ECT early.

GOOD TO REMEMBER

- **Malignant catatonia (lethal catatonia):** catatonic stupor complicated by hyperthermia and autonomic instability, a life-threatening emergency that can evolve from an untreated catatonia; the fatal hyperpyrexial form was described by Stauder in 1934.
- **The one discriminator from NMS:** the preceding picture and drug history, catatonic signs first without a necessary antipsychotic trigger lean to malignant catatonia, a recent antipsychotic start or dose rise with lead-pipe rigidity leans to NMS; the states otherwise overlap and NMS is often regarded as a drug-induced form of malignant catatonia.
- **The first step, the same for both:** stop any antipsychotic, give benzodiazepines (lorazepam), move to ECT early (which treats both), with intensive supportive care; ECT is the gold-standard with about **90%** efficacy (Kaplan & Sadock 2024).

17 · ECT in catatonia and the Bush-Francis rating scale

Catatonia is treatable, and knowing the ladder is the exam point. Once catatonia is recognised as a syndrome (its signs and the raw descriptive definitions are cross-referenced to **the Psychometry, Assessment and Descriptive Psychopathology notes**, and its transdiagnostic nosology sits under ICD-11 6A40), management runs a short, fixed sequence: withdraw dopamine antagonists, correct the medical substrate, give a lorazepam challenge, and move to **electroconvulsive** therapy where the benzodiazepine fails or the picture turns malignant. This topic covers the place of **ect in catatonia** and the one instrument every resident is asked to name, the Bush-Francis Catatonia Rating Scale.

The examiner will not ask you to re-teach schizophrenia (that is owned by **the Schizophrenia: aetiology, phenomenology, classification and course notes**); the discriminator here is knowing *when not to delay* ECT, and knowing what the scale is for. The technique of ECT (electrode placement, stimulus dosing, the course itself) belongs to **the ECT and neurostimulation notes** and is cross-referenced, not taught here.

17.1 The place of ECT: gold standard, and definitive

Highly effective. Electroconvulsive therapy is the gold standard treatment for catatonia, with an efficacy around **90%** in catatonia (Kaplan & Sadock 2024). It has no absolute contraindications and is well tolerated even in medically ill patients, which matters because catatonia often presents in the acutely unwell. Most patients improve within the first **two to three** treatments (Kaplan & Sadock 2024). Lorazepam is best framed as a temporary bridge to definitive ECT: in many cases the benzodiazepine buys time while ECT is arranged, and some patients need monthly maintenance ECT to prevent relapse into catatonia.

RULE **Do not delay it.** ECT is the treatment for malignant or benzodiazepine-non-responsive catatonia; arrange it early, do not wait out a deteriorating patient.

17.2 When ECT is definitive: malignant and benzodiazepine-non-responsive catatonia

Two clear indications. ECT is specifically the treatment for **benzodiazepine-non-responsive** catatonia and for malignant catatonia. In malignant catatonia (also called lethal or pernicious catatonia, described by Stauder), the syndrome is acute in onset with stupor or excitement,

hyperthermia, autonomic instability, rigidity and altered consciousness; it is potentially fatal and, clinically and by laboratory testing, may be indistinguishable from neuroleptic malignant syndrome (Maudsley 2021). Here ECT can be lifesaving and should not be delayed. The same holds for **benzodiazepine non-responsive** catatonia: once an adequate lorazepam challenge has failed, escalating benzodiazepines or, worse, antipsychotics wastes time. Anticipating this, the clinician arranges ECT while the challenge is still running, so that a non-response or a slide into malignant catatonia does not cost days (Kaplan & Sadock 2024).

TRAP **Persisting with benzodiazepines alone.** In a deteriorating malignant catatonia, staying on lorazepam and hoping is the classic error; the patient needs urgent ECT.

WORKED EXAMPLE

A young man in catatonic stupor, mute and rigid, spikes a fever to 39.5 with labile blood pressure and a rising creatine kinase. He had a partial response to 2 mg lorazepam yesterday but is now worse. This is malignant catatonia (indistinguishable from NMS if he was recently on an antipsychotic). The move is not more lorazepam: it is to stop any dopamine antagonist, support vitals, and arrange urgent ECT the same day.

17.3 The lorazepam challenge: the step before ECT

Diagnostic and therapeutic. The lorazepam challenge is given both to confirm catatonia and to treat it. An initial dose of **2 mg** lorazepam is standard for most adults (lower in frail older adults and children), used for both diagnostic and treatment purposes (Kaplan & Sadock 2024). Indian practice describes an intravenous injection of **1 to 2 mg** lorazepam given over two minutes, with catatonic features reassessed shortly after (AIIMS Clinical Methods 2024). The patient is observed over roughly **30 to 60** minutes to see whether the catatonia dissipates; a positive response supports both diagnosis and the plan, and daily lorazepam then holds the symptoms at bay (Practical Psychopharmacology). A positive challenge is also a signal to arrange ECT, because a good lorazepam response predicts a good ECT response.

2 mg	30 to 60 min	around 90%
INITIAL LORAZEPAM CHALLENGE DOSE	OBSERVATION WINDOW	ECT EFFICACY IN CATATONIA

17.4 The Bush-Francis Catatonia Rating Scale: structure

The standard instrument. The **bush-francis catatonia rating scale** (Bush, Fink, Petrides, Dowling & Francis 1996) is the most used scale to identify catatonia and to grade its severity. It is a 23-item scale with two parts that map onto its two jobs (AIIMS Clinical Methods 2024):

The 14-item screen

The presence or absence of items 1 to 14 (the screening subscale) is scored to flag whether catatonia is present.

The severity scale

All 23 items are then rated 0 to 3 to grade severity, based on behaviour observed during a standardised examination.

Ratings are made on observed behaviour during the examination, with withdrawal and autonomic abnormality drawing on chart and collateral. The scale earns its keep at the bedside because catatonia often goes undetected or is mistaken for psychotic agitation, and it does not respond to escalating antipsychotic dosing (Practical Psychopharmacology).

17.5 Using the scale to monitor response

Serial scoring tracks treatment. Beyond identifying catatonia, the Bush-Francis Catatonia Rating Scale is used to monitor the response to the lorazepam challenge and to ECT: the same 0 to 3 severity ratings, repeated before and after the challenge and across the ECT course, give an objective measure of whether the catatonic burden is falling. This is why the instrument is paired with the challenge and the course rather than used once. It is not diagnostic on its own; the ICD-11 or DSM-5-TR criteria (with the anchoring signs cross-referenced to **the Psychometry, Assessment and Descriptive Psychopathology notes**) make the diagnosis, and the scale quantifies it.

MNEMONIC

SCREEN 14, SCORE 23

Items 1 to 14 are the presence/absence screen; all 23 items rated 0 to 3 give the severity score you track across the lorazepam challenge and ECT.

17.6 Malignant catatonia versus NMS: the differential that decides urgency

The one that changes management. The reason malignant catatonia is worth a row of its own is that it can be indistinguishable from neuroleptic malignant syndrome, and mistaking one for the other is a classic error. Both show hyperthermia, autonomic instability and rigidity; a 2000 American Psychiatric Association meeting concluded the two are the same entity, and NMS is often viewed as a drug-induced (antipsychotic-precipitated) variant of malignant catatonia (Kaplan & Sadock 2024; Maudsley 2021). NMS as a discrete entity is owned by **the Schizophrenia: management, antipsychotics and adverse effects notes**; it appears here only as the mirror of malignant catatonia. The practical convergence is the same: stop the offending dopamine antagonist, support the patient, and both respond to ECT.

FEATURE	MALIGNANT CATATONIA	NMS
Core state	Stupor or excitement, then hyperthermia, autonomic instability, rigidity	Hyperthermia, lead-pipe rigidity, autonomic instability, altered consciousness
Trigger	**May arise primary to the psychiatric illness, without a drug trigger**	**Drug-induced: follows a dopamine antagonist, often after a dose increase**
Relationship	Often clinically and biochemically indistinguishable; widely held to be the same syndrome, with NMS a drug-induced variant	
Decisive treatment	**Urgent ECT; do not delay**	Stop the antipsychotic, support, dantrolene/bromocriptine; ECT if refractory

Malignant catatonia and NMS overlap so completely that the same first moves apply; the discriminator is the drug history, not the presentation.

INDIA

ECT for catatonia is well established in Indian practice. AIIMS Clinical Methods in Psychiatry (2024) sets out that urgent modified ECT (mECT) is administered in malignant catatonia, so consent for mECT is taken as soon as possible; mECT may be given daily in severe cases and then reduced to alternate days or twice weekly as the catatonic syndrome declines, with maintenance mECT where needed. The lorazepam challenge (intravenous 1 to 2 mg over two minutes) is the standard first step, and the Bush-Francis Catatonia Rating Scale is named as the most used instrument to monitor catatonic signs. Overall prognosis is good, but delay in diagnosis and treatment raises morbidity, which is exactly why ECT is not delayed.

PEARL

ICD-11 places catatonia as a transdiagnostic grouping, not a subtype of schizophrenia: 6A40 catatonia associated with another mental disorder, 6A41 catatonia induced by substances or medications, and 6E69 secondary catatonia syndrome. This nosological move (catatonia can accompany mood disorders, psychoses or medical illness) is the high-yield point and reinforces the search for a treatable substrate before ECT.

HOLD THIS

ECT is the gold standard for catatonia (around 90% efficacy) and the definitive treatment for malignant or benzodiazepine-non-responsive catatonia; do not delay it, and track the response with the Bush-Francis scale.

GOOD TO REMEMBER

- **ECT in catatonia:** gold standard, efficacy around 90%; definitive for malignant and benzodiazepine-non-responsive catatonia; most improve within two to three treatments; maintenance ECT prevents relapse. Technique lives in the ECT and neurostimulation notes.
- **Lorazepam challenge:** initial 2 mg (IV 1 to 2 mg over two minutes in Indian practice), diagnostic and therapeutic; observe over 30 to 60 minutes; a positive response is the cue to arrange ECT.
- **Bush-Francis Catatonia Rating Scale (Bush 1996):** 23-item scale; items 1 to 14 = presence/absence screen; all 23 rated 0 to 3 for severity; used to identify catatonia and monitor response to the lorazepam challenge and ECT.
- **Malignant (lethal/pernicious) catatonia:** acute stupor or excitement with hyperthermia, autonomic instability, rigidity; often indistinguishable from NMS; urgent ECT can be lifesaving. Stauder eponym.
- **ICD-11 placement:** transdiagnostic grouping, 6A40 (with another mental disorder), 6A41 (substance/medication-induced), 6E69 (secondary catatonia syndrome).

Apply and consolidate

18 · Apply: four worked vignettes

Why this matters. Everything in these notes converges on a small number of bedside decisions, and the examiner tests them through short cases, not through recall of a criterion list. This closing topic runs four **worked vignettes**, each a brief scenario, the reasoning, then the resolution, that reassemble the moves already taught: the schizoaffective versus mood-with-psychosis gate, the delusional-disorder read, the catatonia lorazepam challenge, and the malignant-catatonia versus NMS emergency. Nothing new is introduced; the raw criteria live in the sibling topics and are cross-referenced, not re-derived.

Each vignette is deliberately compact and ends on the single discriminator that settles it, because that is how the case discussion is scored: not on how many features you can list, but on whether you name the one rule that decides the diagnosis. Treat every "Case:" below as a **worked example** to rehearse aloud, and hold the four clinical moves in the good-to-remember box at the close.

18.1 Vignette one: schizoaffective disorder versus mood with psychotic features

The commonest trap in the whole spectrum is to see psychosis and a mood episode together and reach for the schizoaffective label. The decision turns entirely on the two DSM-5-TR rules (Kaplan & Sadock 2024, DSM-5-TR 2022): Criterion B, a period of at least **two weeks** of delusions or hallucinations in the absence of a prominent mood episode; and Criterion C, mood-episode symptoms present for the majority of the total duration of the illness. The full mood-episode criteria are cross-referenced to the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes, and the disorder architecture to the Schizophrenia: aetiology, phenomenology, classification and course notes.

WORKED EXAMPLE

Case: A 34-year-old woman is admitted with a four-month history. For the first two months she had persecutory delusions and third-person auditory hallucinations with no disturbance of mood; over the following two months a full major depressive episode developed, and psychosis and depression have run together since.

Reasoning. Both phenomena are present, so the question is not "is there psychosis and mood" but "is the temporal architecture met". Criterion B asks for at least two weeks of psychosis without a prominent mood episode: here there were a full two months of pure psychosis before any mood change, so the gate is cleared. Criterion C asks that mood dominate the majority of the illness: depression has been present for two of the four months and continues, which is at least half. Both rules hold.

Resolution. Schizoaffective disorder, depressive type. Contrast the counter-case that resolves the other way: if psychosis had appeared only once the depressive episode began and had cleared when mood lifted, Criterion B would fail, there would be no two-week window of pure psychosis, and the diagnosis would be a major depressive episode with psychotic features, where psychosis is confined to the mood episode.

RULE **Two weeks of psychosis without a prominent mood episode is the gate.** Criterion B excludes mood-with-psychosis; Criterion C (mood for the majority of the illness) excludes schizophrenia; both must hold.

TRAP **Diagnosing schizoaffective from concurrent psychosis and mood alone.** Without the two-week pure-psychosis window, co-occurring psychosis and a mood episode are a mood disorder with psychotic features, not schizoaffective disorder.

18.2 Vignette two: delusional disorder versus paranoid schizophrenia

The delusional-disorder read is a discrimination against schizophrenia by what is *absent*. The positive requirement is a single-theme, characteristically non-bizarre delusional system present for at least one month in DSM-5-TR (three months in ICD-11 6A24), with functioning outside the delusion largely preserved and none of the other schizophrenia features (Kaplan & Sadock 2024, DSM-5-TR 2022, ICD-11 CDDR). The raw descriptive definition of a non-bizarre delusion is cross-referenced to the Psychometry, Assessment and Descriptive Psychopathology notes.

WORKED EXAMPLE

Case: A 42-year-old accountant is brought by his wife. For eight months he has been convinced that a colleague is quietly sabotaging his promotion, checking his emails and turning management against him. He is otherwise working, dressing and relating normally; there are no hallucinations, no thought disorder, no negative symptoms, and outside this one theme his conversation and affect are unremarkable.

Reasoning. The belief is plausible-in-kind, a non-bizarre persecutory theme, single and encapsulated, and it has run well beyond the one-month DSM-5-TR (and three-month ICD-11) threshold. The decisive point against paranoid schizophrenia is the preserved picture around the delusion: no prominent hallucinations, no formal thought disorder, no functional collapse. Schizophrenia would show a broader psychotic syndrome and deterioration; here there is a walled garden of one belief in an otherwise intact person.

Resolution. Delusional disorder, persecutory type. Had there been prominent ongoing auditory hallucinations, disorganisation or global functional decline, the diagnosis would shift to schizophrenia, whose criteria and course are owned by the Schizophrenia: aetiology, phenomenology, classification and course notes.

TRAP **Escalating an encapsulated non-bizarre delusion to schizophrenia.** A single-theme delusion with preserved function and no other psychotic features is delusional disorder; do not upgrade it on the strength of the delusion's conviction alone.

INDIA

Indian teaching leans on the ICD tradition, so the three-month non-bizarre-delusion template is the one most residents carry, and morbid jealousy and persecutory presentations are the delusional-disorder subtypes seen most often in clinic. The discrimination against schizophrenia is made the same way: preserved function outside the delusion and the absence of a wider psychotic syndrome.

18.3 Vignette three: catatonia and the lorazepam challenge

Catatonia is the syndrome the examiner most wants worked as a sequence: examine for the signs, exclude an organic cause, give the challenge, then treat with benzodiazepines. ICD-11 codes catatonia as a free-standing, transdiagnostic entity (6A40) requiring three or more signs from the decreased, increased and abnormal-activity clusters; DSM-5-TR cross-maps as three or more of twelve signs (ICD-11 CDDR, DSM-5-TR 2022). The individual signs, catalepsy, waxy flexibility (cerea flexibilitas), mutism, negativism, posturing, echophenomena, are set out with the Bush-Francis scale in the catatonia signs notes and not re-listed here.

WORKED EXAMPLE

Case: A 27-year-old man with known bipolar disorder is admitted mute and immobile. On examination he holds an arm where it is placed (catalepsy), resists passive movement, and shows waxy flexibility. Vitals are stable and he is afebrile.

Reasoning. Three or more signs are present, so the syndromic threshold is met. Before treating, exclude an organic or medical cause, the sign cluster is transdiagnostic and can be driven by encephalitis, metabolic derangement or medication, so a focused work-up (temperature, bloods including creatine kinase, and a screen for an organic driver) comes first. With no red flag for an organic emergency, proceed to the lorazepam challenge: 2 mg IV (1 to 2 mg in adults, lower in children and frail older adults) as a single diagnostic-and-therapeutic dose (Kaplan & Sadock 2024, Clinical Methods in Psychiatry 2024).

Resolution. A marked partial or full response, increased interaction and reduction of catatonic signs, typically within **30 minutes**, both confirms catatonia and opens treatment. Benzodiazepines are then first-line: scheduled lorazepam, titrated from the challenge dose. A positive response confirms the syndrome; sedation or no change does not rule it out. If catatonia is lorazepam-refractory or malignant, ECT is the definitive step, its technique cross-referenced to the ECT and neurostimulation notes.

2 mg IV	within 30 min	70 to 80%
STANDARD ADULT LORAZEPAM CHALLENGE DOSE	RESPONSE WINDOW THAT CONFIRMS CATATONIA	BENZODIAZEPINE EFFICACY FOR SYMPTOM REDUCTION

RULE **The lorazepam challenge is diagnosis and treatment in one.** A 2 mg IV dose that produces a marked response confirms catatonia and starts the benzodiazepine; response is judged by at least 50% improvement.

TRAP **Missing the organic cause.** Catatonia is transdiagnostic: exclude encephalitis, a metabolic cause and medication effects before assuming a primary psychiatric driver, and never reflexively reach for a high-potency antipsychotic.

18.4 Vignette four: malignant catatonia versus neuroleptic malignant syndrome

The emergency vignette is a febrile, rigid, dysautonomic patient, and the task is to reason to the discriminator and the first steps. Malignant catatonia is catatonic stupor with hyperthermia and autonomic instability, a potentially fatal state that at the bedside may be clinically indistinguishable from neuroleptic malignant syndrome, itself best regarded as a drug-induced variant of malignant catatonia (Maudsley Prescribing Guidelines 2021, DSM-5-TR 2022). NMS as an entity is owned by the Schizophrenia: management, antipsychotics and adverse effects notes and appears here only inside this differential.

WORKED EXAMPLE

Case: A 30-year-old man, three days into haloperidol for an acute psychosis, becomes febrile at 39.5 C with lead-pipe rigidity, fluctuating consciousness, labile blood pressure and a creatine kinase of several thousand units.

Reasoning. Fever, rigidity, autonomic instability and a raised CK are common to both malignant catatonia and NMS, so the syndromes overlap at the bedside. The discriminator that settles this presentation is the drug history: recent exposure to a dopamine-blocking antipsychotic points to NMS as the mechanism. Either way the immediate management is the same, because the two are on one continuum.

Resolution. Stop the antipsychotic first. Give a benzodiazepine (lorazepam) and start supportive care, IV fluids, cooling, monitoring, with ECT brought forward urgently if there is incomplete response or progression, since malignant catatonia can evolve fast. Where the underlying psychosis genuinely needs cover once the crisis settles, avoid a high-potency agent; the drug choice belongs to the management notes and the ECT technique to the ECT and neurostimulation notes.

RULE **The discriminator is the drug history; the first three moves are shared.** Recent antipsychotic exposure points to NMS, but for both: stop the antipsychotic, give a benzodiazepine, and move to urgent ECT if it does not settle.

TRAP **Treating malignant catatonia as if it were something other than NMS.** They sit on one continuum; do not withhold benzodiazepines or delay ECT while arguing the label, and never add a high-potency antipsychotic.

HOLD THIS

Four moves, four one-liners: schizoaffective needs at least two weeks of psychosis without a prominent mood episode plus mood for the majority; delusional disorder is a non-bizarre single-theme delusion with preserved function and nothing else; catatonia is three or more signs, exclude an organic cause, then a 2 mg IV lorazepam challenge; malignant catatonia and NMS are one continuum, so stop the antipsychotic, give a benzodiazepine and use urgent ECT.

GOOD TO REMEMBER

- **Schizoaffective versus mood-with-psychosis:** the move is the two-week gate. Diagnose schizoaffective only with at least **two weeks** of psychosis in the absence of a prominent mood episode (Criterion B) plus mood for the majority of the illness (Criterion C); otherwise it is a mood disorder with psychotic features.
- **Delusional disorder versus paranoid schizophrenia:** the move is to diagnose by what is absent. A non-bizarre single-theme delusion, at least one month (DSM-5-TR; three months in ICD-11), with preserved function and no other psychotic features; the number to hold is the one-month (ICD-11 three-month) threshold.
- **Catatonia and the lorazepam challenge:** the move is examine, exclude organic, challenge, treat. Three or more signs (ICD-11 6A40, transdiagnostic; DSM-5-TR three of twelve), then a **2 mg IV** lorazepam challenge with response in about **30 minutes**; benzodiazepines are first-line, symptom reduction **70 to 80%** (Kaplan & Sadock 2024).
- **Malignant catatonia versus NMS:** the move is treat the continuum, not the label. The discriminator is the antipsychotic drug history (NMS is the drug-induced variant); for both, stop the antipsychotic, give a benzodiazepine and use urgent ECT.

19 · Consolidate: mnemonic, retrieval and synthesis

Why this matters. A chapter this long is only as useful as the single thread the reader can pull at midnight before the case discussion. This closing topic does three things and nothing else: it hands over one **master mnemonic** that chains every entity in the block into an ordered walk, it sets a bank of answer-free recall prompts to force **active recall** rather than re-reading, and it names the whole-chapter **synthesis map** to be redrawn from memory. Nothing new is taught here. Every criterion, eponym and number below is owned and verified in the sibling topics and is compressed, not re-derived; the raw descriptive definitions of the delusions and misidentification phenomena stay cross-referenced to the Psychometry, Assessment and Descriptive Psychopathology notes, and the illness schizophrenia itself to the Schizophrenia: aetiology, phenomenology, classification and course notes.

Consolidation is a study step with an evidence base, not a decoration: chaining discrete facts onto one retrieval structure, then testing that structure against blank paper, is how a large block moves from recognition to recall. Treat the mnemonic as the spine, the retrieval prompts as the test, and the synthesis map as the object you must be able to draw with the book shut.

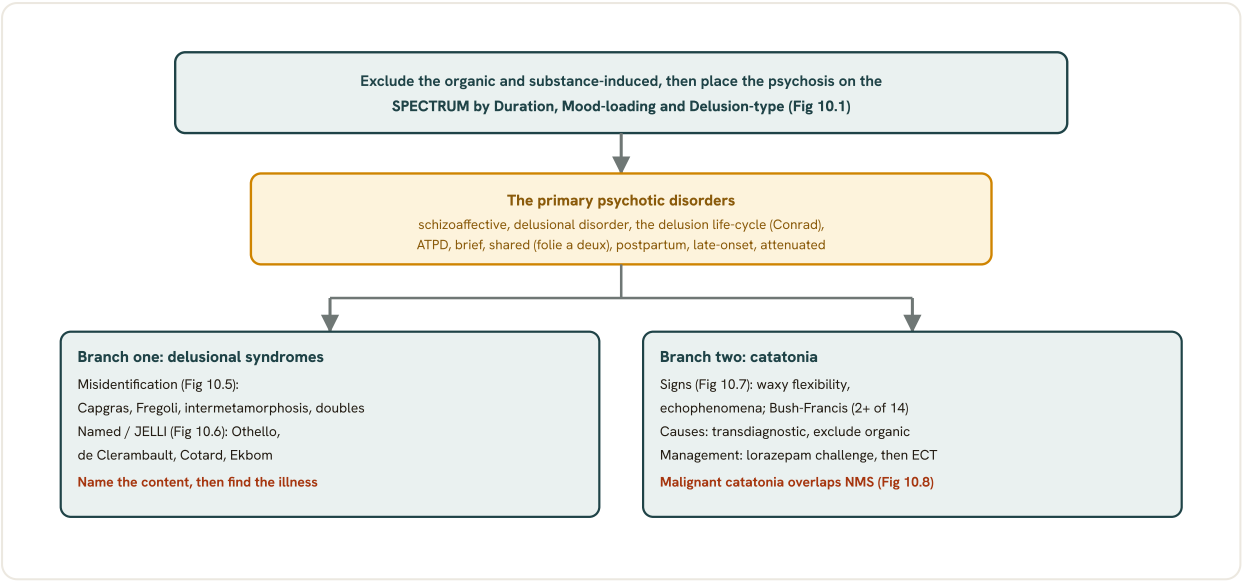


Fig 10.9 The whole-chapter synthesis map, to redraw from memory. The trunk is the spectrum decision tree and its primary psychotic disorders; branch one is the delusional syndromes (misidentification and the named monothematic set), held by content and direction; branch two is catatonia, run as a sequence from signs to causes to management to its malignant form. The gaps you cannot draw are the topics to revisit.

19.1 The master mnemonic: the whole chapter on one chain

The chain seeded at the orientation step now carries its full payload. The three-axis core (place the psychosis on the SPECTRUM by **D**uration, **M**ood-loading and **D**elusion-type) is the trunk; the named delusional syndromes and catatonia hang off it as the two branches the examiner tests by eponym and by sequence. Unpack every letter and the block is recoverable from a single string.

MNEMONIC

"SPECTRUM by D-M-D; then the DELUSIONAL syndromes; then CATATONIA MOVES."

SPECTRUM (the decision tree). First exclude the organic and substance-induced (the silent branch), then place the psychosis on the spectrum by the three axes. **D = Duration:** brief psychotic disorder under one month, schizophreniform one to six months (DSM-5-TR), schizophrenia six months or more (DSM-5-TR) or one month or more (ICD-11), with the acute and transient psychotic disorder (ATPD, ICD-11 6A23) off to the side, acute onset to the full picture within two weeks. **M = Mood-loading:** schizoaffective disorder needs a mood episode for the majority of the illness plus at least two weeks of psychosis without a prominent mood episode; psychosis only within mood episodes is a mood disorder with psychotic features. **D = Delusion-type:** a single non-bizarre delusion in an otherwise preserved person is delusional disorder.

The LIFE-CYCLE and the OTHER PRESENTATIONS. A primary delusion forms through Conrad's stages (Trema to Apophany to Apocalypse) and resolves by integration or sealing-over; and the block carries the compact presentations, the ACUTE and transient psychosis (bouffee delirante, cycloid psychosis), the BRIEF psychotic disorder, the SHARED delusion (folie a deux and its variants: imposee, communiquee, induite, simultanee), the PERINATAL (postpartum) psychosis, the LATE-onset and very-late-onset pictures (paraphrenia, partition delusions), and the ATTENUATED psychosis syndrome.

DELUSIONAL syndromes (branch two). The MISIDENTIFICATION set (Capgras, Fregoli, intermetamorphosis, subjective doubles, reduplicative paramnesia) and the named MONOTHEMATIC set (JELLI: Jealousy = Othello, Erotomania = de Clerambault, Loss-of-existence = Cotard, Little-organisms-Infest = Ekbom). Name the content, then find the illness.

CATATONIA MOVES (branch three). Read it as a sequence: the SIGNS (Kahlbaum's stupor, catalepsy, waxy flexibility, mutism, negativism, echophenomena; scored on the Bush-Francis Catatonia Rating Scale), the CAUSES (transdiagnostic, ICD-11 6A40/6A41/6E69, so exclude an organic driver), the MANAGEMENT (the lorazepam challenge, then benzodiazepines first-line, ECT if refractory), and the MALIGNANT form (Stauder's lethal catatonia, sitting on one continuum with NMS). "Moves" is the reminder that catatonia is the psychomotor syndrome that cuts across the whole grouping. D-M-D is the three-axis core the examiner rewards.

RULE **Walk the mnemonic in order, never by feature-count.** Exclude organic first, place on the spectrum by the three axes, then decide whether the picture is a named delusional content or catatonia; the order is the answer.

TRAP **Memorising the eponyms without the discriminators.** The chain is worthless if Capgras and Fregoli swap, or if Othello is called benign, or if a lorazepam-responsive catatonia is missed for want of the challenge; the letter must recall the rule, not just the name.

HOLD THIS

One string carries the block: place every psychosis on the SPECTRUM by Duration, Mood-loading and Delusion-type after excluding the organic; then work the named DELUSIONAL syndromes (misidentification and JELLI monothematic); then run CATATONIA as signs to causes to management to malignant form.

19.2 Retrieval practice: test the chain against blank paper

Reading the notes again feels like learning and mostly is not. The step that moves this block into recall is **retrieval practice**: shut the book, answer from memory, then check. The prompts below span the three bands (the spectrum and the primary psychotic disorders; the named delusional syndromes; catatonia). Work them as **active recall**, out loud or on paper, before you look anything up. The answers are deliberately absent; every prompt is answered somewhere in the sibling topics of this block.

RETRIEVAL PRACTICE, COVER THE ANSWERS

1. Name the three axes that separate every member of the schizophrenia spectrum, and state the silent branch you apply before you enter the tree.
2. Give the two DSM-5-TR criteria (B and C) that make a diagnosis schizoaffective disorder, and the one that separates it from a mood disorder with psychotic features.
3. State the duration thresholds that split brief psychotic disorder, schizophreniform disorder and schizophrenia in DSM-5-TR, and say where ICD-11 draws the schizophrenia line differently.
4. Define the acute and transient psychotic disorder (ICD-11 6A23) by its onset speed and course, and name its two classical antecedents in the European literature.
5. Give the positive criteria for delusional disorder and the single discriminator that separates it from paranoid schizophrenia.
6. Trace the life-cycle of a primary delusion through its named formation stages, and name the two ways it can resolve.
7. Name the shared-delusion syndrome and its four subtypes by the Latin terms, and state which member is the inducer versus the recipient.
8. State the incidence of postpartum psychosis per deliveries, the window in which most cases begin, and the single highest recurrence-risk factor.
9. Distinguish late-onset schizophrenia from very-late-onset schizophrenia-like psychosis, and name the delusion type classically tied to the older presentation.
10. Match each misidentification eponym (Capgras, Fregoli, intermetamorphosis, subjective doubles) to its core belief and its direction, and state the proportion of Capgras cases with an organic cause.
11. Unpack the JELLI monothematic set: give each eponym, its delusional content, and the one association or forensic risk to hold for each.
12. List the core catatonic signs and name the scale used to score them; then define waxy flexibility and negativism precisely.
13. Describe the lorazepam challenge: drug, dose, route, the response window that confirms catatonia, and the first-line treatment it opens.
14. State where catatonia lives in ICD-11 and why that placement is high-yield, and name the three organic categories you must exclude before calling it primary.
15. Give the one bedside discriminator between malignant catatonia and neuroleptic malignant syndrome, and the first three management moves the two share.

RULE **Answer before you check.** A prompt you can answer from memory is learned; a prompt you can only recognise once you read the answer is not, and re-reading will not fix it, only retrieval will.

19.3 The synthesis map: redraw the block from memory

The final consolidation object is a whole-chapter **synthesis map** to **redraw from memory**: a single figure with one trunk and three branches that you should be able to reproduce on blank paper without the notes open. The **trunk** is the spectrum decision tree, the primary psychotic disorders placed by the three axes (duration, mood-loading, delusion-type) after the organic and substance-induced are excluded, with schizoaffective, delusional disorder and the compact

presentations (acute and transient, brief, shared, perinatal, late-onset, attenuated) as its leaves. **Branch one** is the misidentification and named monothematic delusional syndromes, held by content and direction (Capgras versus Fregoli; the JELLI four). **Branch two** is catatonia as a sequence, signs to causes to management to the malignant form, drawn as the psychomotor syndrome that cuts transdiagnostically across the whole trunk. Redraw it, then check each branch against the good-to-remember boxes of the sibling topics; the gaps you find are the topics to revisit. This is the concept figure for the whole block (Fig 10.9), and drawing it from memory, not reading it, is the last consolidation step.

GOOD TO REMEMBER

- **The master mnemonic:** "SPECTRUM by D-M-D; then the DELUSIONAL syndromes; then CATATONIA MOVES." The whole-chapter chain: exclude organic, place on the spectrum by Duration, Mood-loading and Delusion-type, then the named delusional syndromes, then catatonia signs to causes to management to malignant form. The three-axis core (D-M-D) is the load-bearing part.
- **Retrieval practice:** the study move is to answer the 15 prompts from memory with the answers covered, out loud or on paper, then check; retrieval, not re-reading, is what fixes a large block. Every prompt is answered within the sibling topics of this block.
- **The synthesis map (Fig 10.9):** the object to redraw from memory is one trunk (the spectrum tree and primary disorders, by the three axes) and two branches (the misidentification and JELLI delusional syndromes; catatonia as signs to causes to management to malignant form). The gaps you cannot draw are the topics to revisit.
- **The high-yield anchors to carry:** schizoaffective needs at least **two weeks** of psychosis without prominent mood plus mood for the majority; delusional disorder is at least **one month** (ICD-11 **three months**) of a single non-bizarre delusion; up to **40%** of Capgras cases are organic; catatonia is the **2 mg IV** lorazepam challenge with response in about **30 minutes** and **70% to 80%** benzodiazepine efficacy; malignant catatonia and NMS are one continuum.

Previously asked

This territory is examined less as the long diagnose-and-manage essay of the schizophrenia papers and more as a steady stream of short notes and definitions: a named syndrome, a single disorder, or a discriminating feature. Catatonia is the most examinable single topic and has come as a full short note on its clinical features and, in the neuropsychiatry papers, as organic catatonia; postpartum psychosis and bouffee delirante have each been asked as named short notes. The delusional and misidentification syndromes recur as the definition-and-differentiate items the descriptive-psychopathology and clinical papers favour. The genuine questions on record are grouped by band below.

? HOW THIS TERRITORY IS ACTUALLY TESTED

Q **Other primary psychotic disorders.** These come as named short notes rather than long essays. "Postpartum psychosis" has been asked as a 10-mark note, and "bouffee delirante" as a 10-mark note that rewards knowing the acute-and-transient category it seeds. Schizoaffective disorder, delusional disorder and its subtypes, the acute and transient psychotic disorders, and the late-onset psychoses are the definition-and-criteria short notes this band yields; prepare each to be defined, placed on the spectrum by duration and mood-loading, and separated from schizophrenia. short note, recurring

Q **Delusional misidentification and named syndromes.** The eponymous syndromes are classic short-note and viva territory: define Capgras, Fregoli, De Clerambault (erotomania), Othello (delusional jealousy), Cotard and Ekbom, and name the core delusional content and the one association or risk. Othello syndrome carries a forensic angle worth stating. Prepare these as crisp keyed definitions, not essays. short note and viva, recurring

Q **Catatonia.** The most examinable single topic of the block. "Clinical features of catatonia" has been asked as a 10-mark note, and "organic catatonia" as a 10-mark note in the neuropsychiatry papers, rewarding the aetiological work-up and the exclusion of a medical or neurological cause. Build the signs, the lorazepam challenge, the transdiagnostic placement, and malignant catatonia and its differential from neuroleptic malignant syndrome to answer the note and the emergency question. short note, recurring

Prepare this material to be defined and discriminated, not just recited: the spectrum decision tree, the misidentification and named-syndrome grids, and the malignant-catatonia-versus-NMS differential in these notes are built to the way the topic is examined. The phenomenology, aetiology, criteria and course of schizophrenia itself are developed in the Schizophrenia: aetiology, phenomenology, classification and course notes; the antipsychotic drugs and neuroleptic malignant syndrome as an entity live in the Schizophrenia: management, antipsychotics and adverse effects notes; these notes own the other psychotic disorders, the delusional syndromes, and catatonia.

Quick review

The whole subject in one table	
THE SPECTRUM DECISION TREE	Exclude the organic and substance-induced first, then place the psychosis on three axes: duration, mood-loading and delusion-type. A prominent mood episode pulls out schizoaffective and mood-with-psychosis; a single non-bizarre delusion with preserved function is delusional disorder; the rest go on the duration ruler.
SCHIZOAFFECTIVE DISORDER	At least two weeks of psychosis in the absence of a prominent mood episode (DSM-5-TR Criterion B, excludes mood-with-psychosis) plus mood symptoms for the majority of the total illness (Criterion C, excludes schizophrenia). Bipolar type (F25.0) or depressive type (F25.1). A diagnosis of debated validity: at two years only about a third keep the label. Coined by Kasanin (1933).
DELUSIONAL DISORDER	One or more non-bizarre delusions for at least one month (DSM-5-TR; three months in ICD-11 6A24), function otherwise preserved, no other schizophrenia symptoms. Subtypes by theme: persecutory (commonest), grandiose, jealous (male-predominant, Othello), erotomanic (De Clerambault), somatic (Ekbom), mixed. Onset mid- to late-30s. Kendler's original five dimensions (conviction, extension, bizarreness, disorganisation, pressure) rate form, not content.
THE LIFE-CYCLE OF A DELUSION	Formation through Conrad's phases: trema (delusional mood, an uncanny atmosphere) precedes apophany (the crystallised delusional perception), then anastrophe and apocalypse. Jaspers' primary forms: delusional mood, delusional perception (Schneider's two-memberedness), the sudden autochthonous idea, delusional memory. Dissolution: Bleuler's double bookkeeping and double orientation, encapsulation, and the recovery styles sealing-over versus integration.
ACUTE AND TRANSIENT PSYCHOTIC DISORDER	ICD-11 6A23: abrupt onset to full psychosis within two weeks, no prodrome, a rapidly shifting polymorphic picture, no negative symptoms, duration not over three months, with a characteristically good outcome. The acute polymorphic form (with or without schizophrenia symptoms) is the prototype; bouffee delirante (the Magnan and Legrain tradition) is the French antecedent. Follow up: a minority become schizophrenia or a mood disorder.
BRIEF PSYCHOTIC AND SHARED DISORDERS	Brief psychotic disorder (DSM-5-TR): at least a day, under one month, full recovery, often post-stressor. Induced or shared delusional disorder (folie a deux): a delusion transferred from a dominant psychotic primary to a passive, isolated secondary; separation of the pair usually resolves the secondary's delusion.
PERINATAL AND LATE-ONSET PSYCHOSES	Postpartum psychosis: an acute, often bipolar-spectrum, fluctuating psychosis of the first two weeks, a psychiatric emergency for suicide and

The whole subject in one table	
	infanticide risk; admit, ideally with the baby. Late-onset schizophrenia-like psychosis (onset after 40) and very-late-onset (after 60, the old paraphrenia): commoner in women, with sensory impairment, isolation and partition delusions; exclude a neurodegenerative cause.
ATTENUATED PSYCHOSIS SYNDROME	A DSM-5-TR condition for further study (Section III), not a formal diagnosis: sub-threshold delusion-like ideas, unusual perceptions or disorganised speech with reality testing relatively intact. It names the at-risk mental state (detected by CAARMS or SIPS); only a minority transition (about 15% at one year, 20% at two years), so false-positive and stigma risks keep it out of the main manual.
THE SPECTRUM DIFFERENTIAL	Duration and mood-loading are the master discriminators: brief psychotic under one month, schizophreniform one to six months, schizophrenia six months or more (one month in ICD-11). Schizoaffective needs the two-week pure-psychosis window and mood for the majority; delusional disorder is a single non-bizarre delusion in an otherwise preserved person, against the broader decline of paranoid schizophrenia.
MISIDENTIFICATION SYNDROMES	Keyed on the core misidentification: Capgras (a familiar person seen as an identical impostor), Fregoli (one persecutor disguised inside strangers, the mirror image), intermetamorphosis (both appearance and identity swapped), subjective doubles (doubles of the self). A face-processing disconnection between recognition and the affective sense of familiarity, with a high rate of right-hemisphere or organic pathology (up to 40% of Capgras cases), so exclude an organic cause.
NAMED MONOTHEMATIC SYNDROMES	Content, not disorder (JELLI): De Clerambault (erotomania, another of higher status secretly in love), Othello (delusional jealousy, a real forensic risk of violence, commoner in men), Cotard (nihilistic, being dead or the organs lost, in severe psychotic depression), Ekbom (delusional infestation, the matchbox sign, presents to dermatology). Name the content, then find the illness.
CATATONIA: THE SIGNS	A transdiagnostic psychomotor syndrome, elicited not just observed. The classical Kahlbaum and modern signs across three clusters: decreased (stupor, mutism, negativism, posturing, catalepsy, waxy flexibility), increased or abnormal (stereotypies, mannerisms, echolalia, echopraxia, verbigeration), and the elicited opposites of will (automatic obedience, mitgehen, gegenhalten, ambitendency). Waxy flexibility is the classic sign. Bush-Francis scale: 23 items, first 14 the screen, two or more positive.
CATATONIA: AETIOLOGY	Psychiatric (mood disorders are commoner than schizophrenia), neurological (encephalitis, especially anti-NMDA-receptor encephalitis, seizures, stroke) and medical (metabolic, autoimmune, drug-related) causes; always exclude an organic cause. ICD-11 codes it transdiagnostically (6A40 with another

The whole subject in one table	
	disorder, 6A41 substance-induced, 6E69 secondary), not as a subtype of schizophrenia, which is the shift from ICD-10.
CATATONIA: MANAGEMENT	The lorazepam challenge (1 to 2 mg IV, response usually within 10 minutes) is diagnostic and therapeutic; benzodiazepines are first-line (70 to 80% efficacy), with supportive care and treatment of the cause. Antipsychotics can worsen catatonia and precipitate the malignant form, so they are used with caution. ECT for lorazepam-refractory or malignant catatonia (about 90% efficacy).
MALIGNANT CATATONIA AND NMS	Malignant (lethal) catatonia is catatonic stupor plus fever and autonomic instability, a fatal emergency described by Stauder (1934). It overlaps so completely with neuroleptic malignant syndrome that many treat them as one entity (NMS a drug-induced malignant catatonia). The drug history only leans the label: catatonic signs first, no antipsychotic needed, lean to malignant catatonia; a recent antipsychotic with lead-pipe rigidity leans to NMS. For both: stop the antipsychotic, benzodiazepines, ECT early.
ECT AND THE RATING SCALE	ECT is the treatment for malignant and benzodiazepine-non-responsive catatonia and should not be delayed; the technique is developed in the ECT and neurostimulation notes. The Bush-Francis Catatonia Rating Scale (a 14-item screen and a fuller severity scale) identifies catatonia and monitors the response to the lorazepam challenge and to ECT.
APPLY AND CONSOLIDATE	Four worked cases: schizoaffective versus mood-with-psychosis by the two-week gate, a delusional-disorder read against paranoid schizophrenia, a catatonia worked through the lorazepam challenge, and a malignant-catatonia versus NMS emergency. Then the master mnemonic (place on the spectrum by duration, mood-loading and delusion-type, then the delusional syndromes, then catatonia), answer-free retrieval prompts, and the three-branch synthesis map to redraw from memory.

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The content is grounded in the standard clinical and descriptive-psychopathology texts (the source hierarchy below), which anchor the settled facts on the criteria, the delusional syndromes and catatonia; the nosology follows ICD-11 (CDDR) primary with DSM-5-TR cross-mapped. Every diagnostic criterion, duration threshold, eponym attribution and named figure has been checked against these sources. Each journal PMID here was verified against PubMed this build, matched on title, first author and year; any identifier that did not resolve to the cited paper was corrected or dropped rather than guessed. The classic historical papers that seed the syndromes (Kasanin, Langfeldt, Conrad, Kahlbaum, Capgras and Reboul-Lachaux, Courbon and Fail, Stauder, Schneider) have no stable PubMed record and are cited by author, title and year.

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