APORIA · ISSUE FOUR

04

The Quiet That Speaks.

A 78-year-old widower hears children playing in the courtyard. There are no children. His hearing aids are six years old and out of battery. The first instinct is schizophrenia. The first answer is the audiologist.

CLINICAL DILEMMAS IN PSYCHIATRY

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01 / APORIA

Hallucination is not psychosis

A 78-year-old widower is brought to OPD by his daughter-in-law. For four months he has heard children playing and singing in the courtyard, sometimes at night. He goes to the window to look. There are no children. He has cataracts in both eyes, awaiting surgery; his hearing aids are six years old and the batteries have been dead since his wife died eighteen months ago. He sleeps poorly. He has not eaten with the family for months. He insists what he hears is real. The intern writes "schizophrenia, late onset" in the case sheet. The senior writes "audiology, ophthalmology, sleep hygiene, social reintegration, review at six weeks before any antipsychotic." Both heard the same history.

This is the aporia of the fourth issue. Hallucinations are a symptom class, not a diagnosis. The patient with a hallucination is not the patient with psychosis. The patient with a hallucination in a deprived sensory channel is not the patient with primary psychosis at all. The clinical work is to identify the mechanism, deafferentation, predictive-processing imbalance, source-monitoring failure, sleep disruption, isolation, organic insult, substance effect, or primary psychotic illness, and to treat by mechanism rather than by reflex. Antipsychotic prescription to a Charles Bonnet patient with intact insight is iatrogenic harm. Failure to assess hearing in a deaf elderly patient with voices is the inverse failure.

This issue walks the sensory-deprivation differential from definitions through mechanisms through the four classical syndromes (Charles Bonnet, hearing-loss auditory hallucinations, musical hallucinosis, peduncular hallucinosis), through the recent reframe of minor hallucinations as prodromal markers of Lewy body disease, through sleep deprivation as the related model, through ICU psychosis, through solitary confinement, into the bedside differential and the Indian disability frame. The reference list anchors every clinical claim to the 2023 to 2026 literature, with India-specific data where it exists.

> WHAT THIS ISSUE COVERS
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> Aporia 04 is built on a 998-line research handoff prepared by Luna inside Claudex, supplemented by a 2024 to 2026 literature deepening pass and a verifier crosscheck against primary sources. The structure follows mechanism first, syndromes second, contemporary frontier third, related deprivation models fourth, bedside reasoning fifth, treatment by mechanism sixth, and Indian legal and disability practice seventh. The reference list is twenty-three citations long. The piece is fourteen chapters.

Why this differential matters in Indian practice

Two demographic facts shape this differential. The first is the late-life prevalence of treatable sensory impairment. Indian cataract burden is substantial; hearing-aid access is patchy; the prototypical Indian elderly OPD patient with hallucinations is also the prototypical patient with untreated sensory loss. Marmamula and colleagues' population-based screen in Khammam and Warangal documented this clearly.[1] The second is the cultural construction of perceptual experience: "I heard my husband's voice last night" is reported with

conviction in a population for whom contact with the deceased is a culturally available frame. Distinguishing release hallucinations, normative spiritual experience, prodromal Lewy body disease, and primary psychosis in this register is a real clinical skill.

02 / DEFINITIONS

The vocabulary of misperception

The vocabulary matters because the management diverges sharply by category. A hypnagogic experience, an illusion, a flashback, a release hallucination, a delusional misperception, and a primary psychotic hallucination produce different treatment plans. Wrong label, wrong plan.

HALLUCINATION

A perception-like experience occurring **without a corresponding external stimulus**. Can occur in any modality: auditory, visual, tactile, olfactory, gustatory, somatic, vestibular, multimodal. Reality testing may or may not be preserved.

ILLUSION

Misperception of a real external stimulus. The stimulus exists; the brain misinterprets it. Common in poor lighting, ambiguity, fatigue, anxiety, and intoxication. Not pathognomonic of any disorder.

RELEASE HALLUCINATION

A hallucination that emerges after loss or degradation of sensory input in the same modality. The classical examples are Charles Bonnet syndrome (visual loss, visual hallucinations) and hearing-loss auditory hallucinations. Insight is often retained or recoverable with education. The mechanism is deafferentation plus predictive-processing imbalance.

PSEUDOHALLUCINATION

An older and inconsistently used term, often applied to internally located, insight-retained perceptual experiences. The conceptual boundary against true hallucinations is unstable; many contemporary texts recommend describing the experience phenomenologically rather than reaching for the label.

INTRUSIVE IMAGERY OR THOUGHT

An image or thought with variable sensory vividness but usually recognised by the patient as a mental event. Common in OCD, PTSD, depression. Not a hallucination.

HYPNAGOGIC AND HYPNOPOMPIC PHENOMENA

Sleep-wake transition experiences. Often vivid, sometimes multimodal, usually brief, usually non-pathological. Reported by approximately 30 to 50 percent of the general population at some point. Diagnosis only when frequent, distressing, or associated with narcolepsy.

FLASHBACK

A trauma-linked re-experiencing event, often with dissociative features, often modality-mixed. Tied phenomenologically to the original traumatic memory. Belongs in the PTSD frame, not the psychosis frame.

MINOR HALLUCINATIONS (MHS)

An umbrella for **passage hallucinations** (the sense of something moving past in the peripheral visual field), **presence hallucinations** (the sense of someone being there when no one is), and **visual illusions including face pareidolia**. Once treated as benign incidental findings, now recognised as the single most important early marker of synucleinopathy in Parkinson's disease and Lewy body dementia. [2]

PAREIDOLIA

The perception of meaningful patterns (faces, figures, words) in ambiguous stimuli (clouds, shadows, wood grain). Non-pathological in mild forms. When marked, persistent, or accompanied by behavioural conviction, raises suspicion for DLB or PD.

What counts as sensory deprivation

Sensory deprivation is not one thing. The differential treats it as six overlapping categories. A blind elderly patient living alone, sleeping badly, on anticholinergics, is in five of them simultaneously.

EXPERIMENTAL

Sensory isolation

Laboratory paradigms: ganzfeld, dark adaptation, REST chambers, audiovisual restriction. Produces perceptual distortions, simple hallucinations, anxiety, depersonalisation in healthy subjects.

ENVIRONMENTAL

Setting deprivation

Solitary confinement, ICU isolation, dark rooms, low-stimulation wards, institutional neglect, lockdown conditions. Sensory plus social deprivation overlap.

ORGAN LOSS

Sensory disability

Blindness, low vision, deafness, hearing impairment. The classical substrate for release hallucinations. Severity matters: more loss, more release.

ORGAN DEGRADATION

Sensory degradation

Cataract, macular degeneration, glaucoma, tinnitus, conductive hearing loss, poor lighting, poor acoustics. Often correctable. Often missed.

SOCIAL

Functional deprivation

Bereavement, isolation, occupational inactivity, reduced conversation, family fragmentation, lockdown after retirement. Often the trigger that converts asymptomatic sensory loss into clinical release hallucinations.

CIRCADIAN

Sleep deprivation

Sleep loss, night shifts, mania, stimulant use, delirium risk. Produces a recognisable progression from perceptual distortions through hallucinations to delusion-like states.

03 / MECHANISM

Why the cortex fills the silence

The mechanistic frame for sensory-deprivation hallucinations in 2026 is no longer "the bored cortex makes things up." It is more precise. Four mechanisms operate, usually in combination, and the dominant mechanism determines the clinical signature and the treatment.

01 · THE CLASSICAL ACCOUNT

Perceptual release and deafferentation

CONTINUOUS INPUT NORMALLY CONSTRAINS SPONTANEOUS CORTICAL ACTIVITY

Sensory cortex is not silent in the absence of input. It is active, with stored templates, learned associations, intrinsic oscillations, and population-level firing patterns. Continuous input from the periphery normally constrains this activity by carrying corrective error signals. When peripheral input is reduced or absent, the cortex's spontaneous activity is no longer constrained, and stored content can be "released" into conscious perception.

CLINICAL SIGNATURE

The hallucination modality matches the impaired sensory system. Visual loss produces visual hallucinations (faces, figures, geometric patterns, landscapes). Auditory loss produces auditory hallucinations (voices, music, environmental sounds, doorbells). Insight is often preserved or recoverable with education. Symptoms fluctuate with sensory context: darkness, silence, fatigue, isolation worsen them; engagement, light, sound, social contact relieve them.

> THE RELEASE RULE
>
> *If the hallucination modality matches the deprived sensory system, and there is no other psychotic syndrome, and insight is preserved, the working diagnosis is a release phenomenon. Treat the sensory loss first. Antipsychotic only if distress, danger, or refractory disability remains after sensory restoration.*

02 · THE CONTEMPORARY ACCOUNT

Predictive processing and overweighted priors

BAYESIAN BRAIN UNDER DEGRADED EVIDENCE

The brain continuously predicts sensory input. Perception is the brain's best guess given its priors and the incoming evidence, weighted by their relative precision. When sensory evidence is weak, because the receptor is damaged, the medium is degraded, or the environment is ambiguous, the top-down priors dominate perception. A deprived visual or auditory system gives the brain less corrective feedback against its predictions. The brain perceives what it expects more than what is there.

THE 2024 SYNTHESIS

Keller and Sterzer's 2024 Annual Review of Neuroscience synthesis maps predictive processing onto cortical microcircuitry. Pyramidal cells in specific cortical layers carry prediction-error signals; other layers carry prediction signals. Interneuron-driven gain control sets the precision weighting. Antipsychotics, on this account, work by re-tuning circuit-level precision rather than by globally blunting dopamine. Psychosis is then a disorder of **precision-weighting**, not of perception per se. [3]

THE BRIDGE TO SENSORY DEPRIVATION

Conditioned hallucination paradigms (Powers, Mathys, Corlett) show that strong priors plus weak sensory data produce hallucinations even in non-psychotic subjects, with the effect size scaling by hallucination-proneness. Charles Bonnet syndrome and hearing-loss hallucinations sit on the bottom-up end of this spectrum (weak sensory evidence, normal priors). Primary psychosis sits on the top-down end (normal sensory evidence, overweighted priors). An elderly deaf person with schizophrenia sits at both ends at once. [4]

> THE TEACHING PEARL
>
> *In Charles Bonnet syndrome the cortex is doing what it should, filling absent input with stored templates. In schizophrenia the cortex is doing too much filling regardless of input. Same machinery, different gain setting.*

03 · THE COGNITIVE ACCOUNT

Source monitoring and reality discrimination

FAILURE TO TAG INTERNAL EVENTS AS INTERNAL

Bentall and colleagues' cognitive model frames hallucinations as failures in distinguishing self-generated mental events from externally generated perceptions. In auditory hallucinations, inner speech, memory fragments, intrusive thoughts, or verbal imagery may be misattributed to an external source. Schizophrenia models add impaired corollary-discharge or forward-model signalling: the brain's prediction that "I am about to speak" normally suppresses the sensory response to the resulting speech; if that suppression fails, the patient's own inner speech is heard as external.

THE SENSORY-DEPRIVATION BRIDGE

In sensory deprivation, internal imagery becomes more vivid because external input is weaker. Reality testing becomes harder in prolonged monotony, stress, fatigue, and sleep deprivation. The source-monitoring failure that drives schizophrenia hallucinations and the source-monitoring failure that drives sensory-deprivation hallucinations may share a final common pathway, with different upstream causes.

04 · THE CONTEXTUAL ACCOUNT

Arousal, stress, isolation, and sleep

SENSORY DEPRIVATION IS RARELY NEUTRAL

Sensory deprivation in clinical practice is almost never a clean experimental manipulation. It comes packaged with anxiety, loneliness, hypervigilance, sleep loss, reduced reality-checking through social feedback, reduced structured activity, and increased attention to internal sensations. Each of these independently increases the probability of perceptual misattribution. The cumulative effect is why solitary confinement, ICU admission, bereavement, lockdown, and institutional neglect produce hallucinations and psychosis-like states even in patients without chronic psychotic illness.

CLINICAL IMPLICATION

Treating sensory loss alone is rarely sufficient. The release phenomenon is real and the sensory restoration is necessary, but the lonely, sleep-deprived, frightened patient with restored hearing aids will continue to hallucinate. The clinical plan must address all four mechanisms simultaneously: input restoration, prior re-calibration through education, source-monitoring through reality engagement, and contextual stabilisation through sleep, structure, and social contact.

04 / SYNDROMES

The classical four

Four release syndromes are recognised by name in the literature. Each has a characteristic modality, demographic, and clinical signature. Each carries a low likelihood of converting to primary psychosis. Each is misdiagnosed in routine practice. The first three are common; the fourth is rare but mechanistically informative.

Charles Bonnet syndrome

Visual release hallucinations in a patient with visual impairment, with preserved insight and without delirium, dementia, or primary psychosis. First described by Charles Bonnet in 1760 as the experience of his grandfather, Charles Lullin, whose vision had been lost to cataracts. The 2025 meta-analysis by Christoph and colleagues across 49 studies and 20,303 patients pooled prevalence at 10.2 percent across ophthalmic populations, rising to 24.6 percent in vision-rehabilitation cohorts.[5] Indian tertiary-care samples report prevalence between 6.7 and 8.1 percent, with cataract dominating the denominator, making "treat the cataract first" the highest-yield clinical move.

SUPPORTIVE FEATURES

What argues for CBS

- Visual hallucinations are formed or complex (people, animals, patterns, landscapes, faces, figures).
- Hallucinations are purely visual.
- Insight is retained, or can be restored with explanation.
- No primary delusions, formal thought disorder, prominent negative symptoms, or global cognitive fluctuation.
- Episodes worsen in dim light, inactivity, fatigue, isolation, sensory monotony.
- Documented visual impairment in the same modality as the hallucination.

AGAINST FEATURES

What argues against simple CBS

- Auditory hallucinations accompanying the visual.
- Fixed delusional interpretation of the experience.
- Command hallucinations.
- Disorganised behaviour or thought disorder.
- Fluctuating consciousness.
- New neurological signs or rapid cognitive decline.
- Substance exposure or withdrawal.

Hearing-loss auditory hallucinations

The auditory analogue of Charles Bonnet syndrome. Linszen and colleagues' 2019 study of ENT-audiology referrals reported recent auditory hallucinations in 16.2 percent of hearing-impaired participants versus 5.8 percent of non-impaired controls, with rates rising to 24 percent in the most profoundly impaired group.[6] Content included voices, music, doorbells, telephones, and other environmental sounds. The clinical implication is that voices in a hearing-impaired patient are not automatically schizophrenia, and that hearing should be assessed in any patient presenting with auditory hallucinations, especially late-onset or atypical cases.

Musical hallucinosis

Formed songs, instrumental music, or tunes perceived without external stimulus, with intact reality testing. Demographics: elderly, female, hearing-impaired, often socially isolated. The Vitorovic and Biller 2013 review remains the canonical definition, with extensive case-series follow-up since.[7] The differential matters because four entities can sound similar at the bedside.

ENTITY	ORIGIN	PHENOMENOLOGY	CLINICAL SIGNATURE
Earworm	Internal, voluntary or involuntary	Brief, recognised as own thought	90% of healthy adults; not a hallucination
Palinacousis	Perseveration of a real recent stimulus	Stimulus-locked, decays over minutes	Temporal-lobe lesion or seizure focus
Musical hallucinosis	Spontaneous, complex	Externally located, no recent triggering stimulus	Hearing loss usually present; elderly female predominance
Schizophrenic auditory hallucinations	Disordered source monitoring	Voices more than music; commentary, command, second or third person	Embedded in delusions or thought disorder

Treatment is mostly case-series-based. Sanchez 2011 and the Coebergh 2015 Frontiers in Psychology review summarised the field: cholinesterase inhibitors (donepezil) have the most consistent positive case reports, supporting a cholinergic-deficit mechanism in elderly hearing-impaired patients. SSRIs help when anxiety drives distress. Antipsychotics are second-line; quetiapine and risperidone have case-level support but bring extrapyramidal, metabolic, and fall risks, poor choices in the typical demographic. Hearing-aid optimisation and sound enrichment are first-line and frequently sufficient.[8]

Peduncular hallucinosis

Vivid, complex, formed visual hallucinations (people, animals, scenes) following lesions of the brainstem (midbrain, pons) or thalamus, classically described by Lhermitte in 1922. The patient often retains insight. Lesions disrupt ascending cholinergic and serotonergic pathways from the brainstem, removing inhibition on cortical visual areas. Rare; mechanistically informative because it shows that release hallucinations can arise from lesions central to the visual pathway, not just peripheral degradation.

05 / FRONTIER

Minor hallucinations as prodromal Lewy body disease

Of all the framings that have shifted in this differential over the past decade, this one matters most for the OPD. Minor hallucinations, passage, presence, and pareidolic, were treated as benign curiosities throughout most of twentieth-century psychiatry. They are now the single most reliable early marker of synucleinopathy that residents miss. The patient with isolated minor hallucinations and no other psychotic features is not a Charles Bonnet patient and is not a primary psychosis patient. Increasingly, they are a prodromal-DLB patient.

> THE CONTEMPORARY PICTURE
>
> Pagonabarraga, Onofrj and colleagues established through the 2014 to 2024 literature that minor hallucinations precede well-formed visual hallucinations in Parkinson's disease by years and frequently appear in the prodromal phase of dementia with Lewy bodies, often before motor symptoms. The 2025 npj Parkinson's Disease paper by Cao and colleagues showed a quantitative dose-response: the number of distinct minor hallucination types predicts conversion to well-structured visual hallucinations.[9] The 2020 prodromal DLB research criteria (McKeith et al.) explicitly included minor hallucinations as a supportive prodromal marker. The 2025 Lancet Neurology DLB biomarker update now pairs minor hallucinations with CSF alpha-synuclein seed amplification assay and hyposmia for a multi-marker prodromal flag.

The mechanistic account

The Onofrj TDDMN model, Thalamocortical Dysrhythmia plus Default Mode Network decoupling, is the leading mechanistic frame. Slowed thalamocortical oscillations, in combination with default-mode network over-activation, allow internally generated imagery to be perceived as external. The same mechanism may explain why minor hallucinations and well-formed visual hallucinations co-exist on a spectrum, with the latter representing a more severe expression of the same circuit pathology.

Why this matters in Indian elderly OPDs

Pareidolia and "ghost-on-the-veranda" presence hallucinations are routinely missed in Indian elderly OPDs because they are dismissed as cultural or religious phenomena. "She saw her dead husband again" is a sentence that closes the case in many family histories. The clinical reasoning piece needs to call this out: a Kannada-speaking 72-year-old reporting "someone passed behind me but no one was there" is a prodromal-DLB screen until proven otherwise. The work-up is ophthalmology and audiology first, then cognitive assessment (MoCA), then sleep history (REM sleep behaviour disorder is the strongest prodromal-DLB marker), then formal neurological assessment for early extrapyramidal signs, then olfactory testing where available.

> *The patient who sees her dead husband and finds it comforting is in the cultural register. The patient who sees figures passing in the periphery and is frightened, with intact insight, with a partner reporting that she has begun acting out her dreams, is in the synucleinopathy register. The phenomenology is the same. The work-up is different.*
>
> BEDSIDE REASONING PEARL

06 / SLEEP

Sleep deprivation as the related model

Sleep deprivation is the cleanest experimental analogue of sensory deprivation, in that the manipulation is well-characterised and the resulting perceptual progression is reproducible. Waters and colleagues' 2018 Frontiers in Psychiatry review of historical sleep-deprivation studies across twenty-one studies and 760 participants documented a recognisable progression that residents should know. [10]

24-48h	**Perceptual distortions.** Anxiety, irritability, depersonalisation, temporal disorientation. Reality testing intact but effortful.
48-90h	**Complex hallucinations and disordered thinking.** Visual phenomena most common, followed by somatosensory, then auditory. Reality testing wavering.
After 72h	**Delusional thinking may appear.** The clinical picture begins to resemble acute psychosis or toxic delirium. Many symptoms resolve after sleep recovery.

The clinical implication for the OPD is direct. Acute hallucinations after sleep loss, shift work, intoxication, mania, withdrawal, or ICU stay should trigger a sleep and circadian assessment before any antipsychotic decision is made. Recovery sleep is diagnostic information: if hallucinations resolve after restoration of sleep, the primary cause has been identified. If they persist, the primary cause is elsewhere.

07 / ICU

Critical illness as sensory deprivation

ICU-acquired psychiatric morbidity is increasingly framed as a sensory-deprivation syndrome layered on critical illness. The patient in an ICU is in five deprivation categories at once: organ degradation through critical illness, environmental deprivation through windowless room and PPE-masked staff, social deprivation through family-

contact restriction, circadian deprivation through 24-hour artificial light and overnight monitoring, and pharmacological deprivation through sedation. The 2026 prevention frame focuses on minimising each of these and is anchored in two acronyms.

ABCDEF BUNDLE

Assess and treat pain. **B**oth spontaneous awakening trials and spontaneous breathing trials. **C**hoice of analgesia and sedation. **D**elirium assessment and management. **E**arly mobility and exercise. **F**amily engagement and empowerment. Kundakci and colleagues' 2025 systematic meta-review of thirty-two reviews and 335 trials reported the bundle nearly halves delirium risk (relative risk 0.57, 95% confidence interval 0.36 to 0.90; six studies, n = 2,000) and reduces delirium duration by approximately 1.4 days. Mortality unchanged; evidence certainty graded low. [11]

ECASH CONCEPT

Early Comfort using Analgesia with minimal Sedatives and maximum Humanity (Vincent et al. 2016, still the canonical name). Explicit components: multimodal analgesia first, sedation only if required, **glasses and hearing aids reinserted as soon as possible**, daylight exposure via windows, noise reduction at night, family visits, communication boards. The 2024 Chinese multidisciplinary bundle RCT reported delirium incidence 11.4 percent versus 31.4 percent (intervention versus control). [12]

Post-ICU PTSD-psychosis overlap

Patients with delusional ICU memories, often paranoid, persecutory, involving staff or family as perpetrators, carry the highest post-ICU PTSD risk. The mechanism is partly degraded sensory plus altered consciousness plus sedation generating delusional encoding that persists after discharge. Standard PTSD screening with PCL-5 at three months post-discharge is now recommended in the relevant critical-care psychiatry literature.

The practical resident checklist

For the resident on rotation: glasses on, hearing aid in, curtains open during day, family allowed at agreed times, RASS sedation target documented, CAM-ICU screen at every shift. The Hubli ward routine of side-room isolation for "difficult" psychotic or catatonic patients is iatrogenic sensory deprivation. The patient lying in a dark side room with curtains drawn is being given the worst possible environment for recovery from catatonia or psychosis. Lorazepam plus sensory and social re-engagement is the standard of care; understimulation worsens what it is meant to contain.

08 / CONFINEMENT

Solitary confinement and isolation psychosis

Sensory deprivation in its most concentrated form is solitary confinement, and its psychiatric consequences are the most heavily documented in the deprivation literature. The cluster described by Grassian, perceptual distortions, hallucinations (auditory more than visual), paranoid ideation, hypersensitivity to stimuli, intrusive ruminations, cognitive impairment, sleep disturbance, self-harm, appears within days, reaches full picture within weeks, and has chronic sequelae including persistent psychosis, PTSD, and depression. Systematic reviews place new psychiatric morbidity prevalence in solitary populations at fifteen to thirty percent.

> THE LEGAL FRAME
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> The United Nations Standard Minimum Rules for the Treatment of Prisoners (the Mandela Rules), Rules 43 to 45, define prolonged solitary confinement as more than fifteen consecutive days and classify it as **torture or cruel, inhuman, and degrading treatment**. Indefinite solitary confinement is prohibited outright. The twenty-two hours per day without meaningful human contact threshold defines solitary confinement. Indian statutory position under **Section 12 of the Bharatiya Nyaya Sanhita 2023** caps a single bout of solitary confinement at fourteen days, with cumulative caps depending on sentence length. The framework is Mandela-compliant on paper. Documentation of confinement duration and conditions is mandatory when a psychiatric evaluation is requested for an inmate.

Clinical implication for the Indian psychiatrist

When called for a prison medico-legal evaluation, the clinical task is to assess the patient's mental state in the context of the confinement environment, not in isolation from it. Hallucinations in a solitary inmate, particularly auditory and persecutory, are presumptively reversible, they may resolve with restoration of social contact and sensory environment, and may not represent a primary psychotic disorder. The forensic and clinical interpretation of the same symptom differs sharply between a solitary inmate and a community-dwelling patient. Documentation of confinement duration, lighting, noise, social contact frequency, and timing of symptom onset relative to confinement is the load-bearing clinical content.

09 / DIFFERENTIAL

The clinician's question, in order

The bedside reasoning algorithm for hallucinations runs through seven questions. The first question is never "Is this schizophrenia?" The first question is "Is this actually a hallucination?"

01 **Is this a hallucination, or something else?** Illusion, intrusive imagery, flashback, dissociation, sleep-transition experience, tinnitus, misperception. Characterise the experience phenomenologically before reaching for a label.

02 **Is consciousness clear and attention intact?** If attention or awareness fluctuates, the working diagnosis is delirium until proven otherwise. Do not diagnose schizophrenia, CBS, or any other primary psychiatric disorder in a fluctuating confused patient.

03 **Is there sensory impairment in the same modality?** Visual hallucination with low vision: think CBS. Auditory hallucination with hearing impairment: think hearing-loss auditory hallucinations or musical hallucinosis. Assess the sensory organ first.

04 **Is sleep severely impaired?** Total sleep time, duration of deprivation, mania or hypomania signs, stimulant or substance use, withdrawal patterns, ICU or night-shift context. Recovery sleep is diagnostic.

05 **Is there intoxication, withdrawal, medication effect, or medical cause?** Anticholinergics, steroids, dopaminergic drugs, stimulants, cannabis, hallucinogens, alcohol withdrawal, metabolic disturbance, seizure, delirium, dementia, neurological illness, autoimmune encephalitis.

06 **Is there a mood syndrome, trauma syndrome, primary psychosis, or culturally shaped non-pathological experience?** Mood-congruence, trauma-link, broader psychotic syndrome, normative cultural register all change the frame.

07 **What is the level of insight, distress, danger, dysfunction, and behavioural consequence?** Insight retained does not mean fake; insight lost does not automatically mean schizophrenia. Decide treatment intensity based on these five axes, not on the symptom alone.

> ALGORITHM A: HALLUCINATIONS IN A LOW-VISION PATIENT
>
> (1) Check attention, orientation, delirium, intoxication, withdrawal. (2) Confirm hallucinations are purely visual. (3) Assess insight and delusional interpretation. (4) Screen cognition and parkinsonism in older adults; check for minor hallucinations and REM sleep behaviour disorder. (5) Review medications. (6) Refer ophthalmology and optimise vision; treat cataract first in the Indian denominator. (7) Explain CBS if criteria fit; use lighting, activity, social contact, coping manoeuvres. (8) If severe distress or risk persists, consider cautious pharmacotherapy and re-check diagnosis.

> ALGORITHM B: VOICES IN A HEARING-IMPAIRED PATIENT
>
> (1) Characterise voices, music, environmental sounds, tinnitus. (2) Assess hearing impairment and hearing aid use. (3) Screen psychosis: delusions, disorganisation, negative symptoms, mood syndrome. (4) Check sleep, substances, medications, cognition. (5) Treat hearing loss and enrich sound environment. (6) Use CBT for psychosis or voice-coping where distressing. (7) Use antipsychotic if primary psychosis, severe risk or distress, fixed delusional interpretation, or persistent disabling symptoms.

> ALGORITHM C: ACUTE HALLUCINATIONS AFTER SLEEP LOSS
>
> (1) Check duration of sleep deprivation. (2) Screen mania, stimulant use, withdrawal, delirium, medical illness. (3) Restore sleep safely. (4) Monitor after sleep recovery. (5) If psychosis persists beyond sleep restoration or mood and substance correction, reassess for primary psychosis or mood disorder.

10 / TREATMENT

Match the intervention to the mechanism

The principle is mechanism-matching. Sensory restoration is the treatment for release hallucinations. Sleep restoration is the treatment for sleep-deprivation psychosis. Substance correction is the treatment for substance-induced states. Antipsychotic is the treatment for primary psychosis. Confusing these is the most common error in the differential and the most consequential, the elderly low-vision patient who is started on risperidone for CBS gets nothing for the visual loss and everything for an anticholinergic burden she did not need.

Universal management steps

1. **Establish safety**: command hallucinations, suicidal or violent commands, self-harm, aggression, neglect, inability to eat or sleep, wandering, vulnerability. Assess insight and behavioural response. Consider

admission if risk, severe agitation, delirium, catatonia, severe mania or depression, or uncertain medical cause.

2. **Normalise without dismissing**: explain that hallucinations can happen from brain, eye, ear, sleep, stress, substances, mood, trauma, or psychosis mechanisms. Reduce shame and fear of being "mad."
3. **Look for reversible causes**: vision and hearing assessment, medication review, substance and withdrawal history, sleep restoration, delirium screen, medical and neurological examination.
4. **Match intervention to mechanism**: sensory loss: restore and enrich input. Sleep deprivation: restore sleep and circadian rhythm. Delirium: treat underlying cause. Substance-induced: withdrawal or intoxication management plus relapse prevention. Primary psychosis: antipsychotic plus psychosocial. Trauma or dissociation: stabilisation and trauma-informed care.

Charles Bonnet syndrome, management

First line is non-pharmacological: education and reassurance, optimisation of vision and lighting, reduction of isolation and inactivity. The explanation that the experience is real to the brain but is not a sign of "madness" is therapeutic in itself. Coping manoeuvres include changing lighting, blinking, shifting gaze, looking away or toward the image, engaging another task. Medication is usually unnecessary. Consider it only if severe distress, dangerous behaviour, severe insomnia, or persistent disabling hallucinations after sensory and environmental measures. Evidence is weak and case-based for antipsychotics, SSRIs, anticonvulsants, and cholinesterase inhibitors. In elderly patients, be conservative, falls, metabolic effects, anticholinergic burden, QTc, parkinsonism, and cognitive risk all matter.

Hearing-loss auditory hallucinations, management

First line is audiology and ENT treatment, hearing aids or amplification when indicated, sound enrichment, psychoeducation, and reduction of stigma so patients report symptoms. Consider antipsychotic when there is fixed delusional interpretation, severe distress or risk, multimodal hallucinations with psychotic syndrome, persistent hallucinations after correction of hearing and sleep and substance and medical factors, or coexisting schizophrenia, mood psychosis, delirium, dementia psychosis, or substance-induced psychosis.

Musical hallucinosis, management

Hearing aid optimisation and sound enrichment first line and frequently sufficient. If pharmacotherapy is needed: donepezil has the most consistent positive case reports in elderly hearing-impaired patients (supporting the cholinergic-deficit mechanism). SSRIs help when anxiety drives distress. Antipsychotics are second-line; quetiapine and risperidone have case-level support but bring EPS, metabolic, and fall risks. Pearl: if an elderly patient says "the radio is playing in the next room but no one is there," check the hearing aid before the SMI register.

Sleep-deprivation and isolation-related psychosis-like states

Sleep restoration is first line. Reduce stimulation if manic or agitated but avoid total sensory isolation. Correct circadian rhythm. Treat mania, withdrawal, pain, ICU delirium, or stimulant use. Hydration, nutrition, medical stabilisation. Pharmacology may include short-term sedative or hypnotic where clinically appropriate; benzodiazepines for alcohol or benzodiazepine withdrawal, catatonia, or severe agitation; antipsychotics if severe agitation, frank psychosis, danger, mania, delirium-related distress after non-pharmacological measures, or persistence despite sleep restoration.

11 / INDIAN FRAME

Disability, certification, and the legal lens

Indian psychiatric practice intersects with sensory disability and mental illness disability under the Rights of Persons with Disabilities Act 2016 and the DEPwD assessment guidelines. The 14 March 2024 DEPwD guideline and the RPwD (Amendment) Rules 2024 (gazetted 22 October 2024) updated the operational framework in five practice-relevant ways.

01 **Mandatory UDID portal route.** All applications submitted through the UDID and Swavlamban portals. Walk-in paper certificates are being phased out.

02 **Three-month statutory turnaround.** Medical authorities must issue the certificate and UDID card within three months of application.

03 **Colour-coded UDID cards.** White for less than 40 percent, yellow for 40 to 79 percent, blue for 80 percent or more. The disability tier is visible on the card, eliminating earlier ambiguity around "benchmark disability."

04 **Residual disability despite appropriate treatment** is now an explicit gating phrase for permanent mental-illness certification. The two-year duration requirement is retained.

05 **Private specialists may join boards** under supervision of a government medical officer, widening access in tier-2 and tier-3 cities.

IDEAS scoring

The Indian Disability Evaluation and Assessment Scale remains the canonical instrument for mental illness disability certification.[13] Four domains: self-care, interpersonal activities and social relationships, communication and understanding, work (employment, housework, education, role functioning). Each scored 0 to 4 (0 none, 1 mild, 2 moderate, 3 severe, 4 profound). Duration of illness is added to the total. Global disability categories: 1 to 6 mild (less than 40 percent), 7 to 13 moderate (40 to 70 percent), 14 to 19 severe (71 to 99 percent), 20 profound (100 percent). Minimum threshold for most benefits and concessions is 40 percent benchmark disability.

Sensory deprivation cases and disability

PRESENTATION	DISABILITY FRAMING
Low vision with CBS, intact insight, preserved function	May qualify for **visual disability** if visual impairment meets RPwD criteria. CBS itself is *not* automatically mental illness disability.
Hearing impairment with auditory hallucinations, no other psychotic syndrome	May qualify for **hearing disability** if audiological thresholds meet criteria. Hallucinations alone do not create mental illness disability.
Schizophrenia with visual or hearing impairment, both with documented impairment	May have both **mental illness disability and sensory disability**. Multiple disability rules apply.
Hallucinations due to delirium, acute substance state, sleep deprivation, brief stress reaction	Usually **does not qualify** for permanent mental illness disability on that basis alone.

> MULTIPLE DISABILITY COMPOSITE FORMULA
>
> Multiple disabilities are **not additively summed**. The composite percentage uses the formula **a + b(90-a)/90**, where *a* is the higher disability percentage and *b* is the lower, capped at 100 percent. Disabilities below the individual threshold (typically 40 percent) usually cannot be combined to reach benchmark status. A patient with 35 percent visual disability and 30 percent mental illness disability does *not* compose to 65 percent benchmark disability. This is a high-yield exam trap and a frequent clinical disappointment.

Karnataka UDID workflow

Applications via swavlambancard.gov.in proceed to the District Medical Board (DHO office, district hospital), where assessment is conducted by the notified psychiatrist plus ophthalmologist or audiologist for sensory components, with upload at the portal and electronic issuance of the UDID. Karnataka helpline 080-22052750. KMCRI Hubli is a notified centre; Dharwad and Belgaum district hospitals also issue. For combined sensory plus mental illness disability, the board sits jointly. Expect two to four visits in practice despite the three-month statutory limit.

MHCA 2017 implications

The Mental Healthcare Act 2017 applies when admission, treatment, or capacity questions arise. The Act's emphasis on dignity, least restrictive care, advance directives, nominated representatives, and capacity assessment does not change with the underlying cause of hallucinations. The practical difference at the bedside is that a CBS or hearing-loss release-hallucination patient with preserved general functioning often has preserved capacity for treatment decisions; the threshold for supported admission processes should be calibrated to functioning, not symptom presence.

12 / THE PATIENT

The case in the lede, resolved

The 78-year-old widower hearing children in the courtyard. Four months of symptoms. Bilateral cataracts awaiting surgery. Hearing aids dead for eighteen months. Poor sleep. Bereavement plus social withdrawal. Conviction that what he hears is real.

The working diagnosis is **release hallucinations in the context of combined sensory deprivation**, with auditory release hallucinations (hearing loss) and likely Charles Bonnet contribution if visual hallucinations are also present (cataracts). The contributing factors are sleep loss, bereavement-related social isolation, and possible nutritional and medical deconditioning. The conviction the patient holds is incomplete insight, which is recoverable with explanation.

The clinical plan is mechanism-matched: **audiology referral and hearing-aid restoration first**, with new batteries and an assessment for an updated device; **ophthalmology referral for cataract surgery**; **sleep restoration** with sleep hygiene, a structured routine, and brief pharmacotherapy if needed; **social re-engagement** with daughter-in-law family meetings and structured activity; **cognitive screen** with MoCA and a focused inquiry for minor hallucinations and REM sleep behaviour disorder to screen for prodromal Lewy body disease; **medical work-up** with vitals, basic blood work, B12 and folate, urinalysis. Review at four weeks for hallucination response to sensory restoration and sleep correction. Antipsychotic is held in reserve.

The two diagnoses that need to be excluded by the four-week review are **prodromal Lewy body disease** (if minor hallucinations, REM sleep behaviour disorder, hyposmia, or early extrapyramidal signs emerge) and **delirium superimposed on chronic deprivation** (if attention or consciousness has fluctuated). The diagnosis that is least likely, and that should never have been the intern's first answer, is **schizophrenia, late onset**. The patient's symptoms are explained by the available mechanisms; no syndrome of schizophrenia has been demonstrated; the burden of proof for a psychiatric label is on the clinician, not on the patient.

The teaching point

The shortest version of this issue's argument is: hallucinations are not the same as psychosis; psychosis is hallucinations plus impaired reality testing, interpretation, function, behaviour, syndrome context, or diagnostic exclusions. The shortest viva answer is: define hallucination, list the modalities, explain the sensory deprivation and release mechanism, give the CBS and hearing-loss examples, differentiate from schizophrenia, delirium, substance-induced states, mood psychosis, dementia, trauma and dissociation; mention workup red flags; manage cause first and antipsychotics only when syndrome, risk, or distress warrant. In India, assess disability by function and law: RPwD 2016, IDEAS, UDID, 2024 guidelines, benchmark disability, multiple disability composite formula.

13 / PEARLS

Short clinical pearls

- Visual hallucinations in an elderly low-vision patient are **ophthalmology until proven otherwise**.
- Auditory hallucinations in a deaf or hard-of-hearing patient are **audiology plus psychiatry**, not psychiatry alone.
- Sleep deprivation can mimic psychosis; **recovery sleep is diagnostic information**.
- Multimodal hallucinations, delusions, disorganisation, negative symptoms, and functional decline raise **primary psychosis probability**.
- Fluctuating attention means **delirium until proven otherwise**. Do not diagnose schizophrenia or CBS in a fluctuating confused patient.
- Insight retained does not mean fake; insight lost does not automatically mean schizophrenia.
- Social isolation is treatment-relevant, not just background history.
- Disability certification is about durable functional impairment after treatment, not symptom drama on the day of interview.

- Minor hallucinations in an elderly patient are **prodromal Lewy body disease until proven otherwise**. Ask about presence and passage hallucinations, ask about REM sleep behaviour disorder, screen olfaction, screen cognition.
- In the ICU, the patient lying in a dark side room with curtains drawn is being given the worst possible environment for recovery. **Sensory and social re-engagement is treatment**.
- The matchbox sign in delusional infestation belongs to F22 (delusional disorder), not to this issue's differential, but it is the bedside reminder that somatic hallucinations and somatic delusions can coexist.
- Religious or culturally-framed perceptual experiences in Indian patients are **not automatically pathological**; assess against the patient's reference group, not against textbook content.

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

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Colophon. Aporia is a serialised clinical reasoning publication by Dr. Wilfred D'souza, third-year MD Psychiatry resident at KMCRI, Hubli. Issue 04 was written in May 2026. The structural backbone came from a 998-line research handoff prepared by Luna inside Claudex (the Codex-side bounded sandbox), drawing on local KIMS Hubli papers on hallucinations, the IPS CPG 2026, the ICD-11 CDDR, the DEPwD 14 March 2024 assessment guideline, IDEAS, and the Indian disability literature. The 2024 to 2026 deepening pass surfaced the Christoph 2025 CBS meta-analysis, the Keller and Sterzer 2024 predictive-processing synthesis, the Cao 2025 dose-response between minor hallucinations and well-structured visual hallucinations, the Kundakci 2025 ICU delirium meta-review, the eCASH framework, the 2024 RPwD amendment and colour-coded UDID system, and the Section 12 BNS 2023 framework on solitary confinement. A verifier pass confirmed the handoff's numerical claims (Waters 21/760 progression, Linszen 16.2/5.8 prevalence) and the ICD-11 codes. The piece is set in Switzer, JetBrains Mono and Newsreader. Comments to wilfred@weave.clinic.