

# Hypersomnolence Disorder

DSM-5-TR 780.54 | ICD-10-CM G47.10

*Excessive sleepiness despite at least 7 hours of sleep, with unrefreshing long sleep, severe sleep inertia, and irrepressible daytime naps.*

**PREVALENCE**  
~1% of US adults

**TYPICAL ONSET**  
Late teens to early 20s

**SEX RATIO**  
Roughly equal

**COURSE**  
Chronic and stable once set

## CLINICAL PICTURE

- Patients sleep 9 or more hours yet wake unrefreshed, describing sleep drunkenness with confusion and irritability that can last an hour or more.
- Naps are long, typically over an hour, and non-restorative, in sharp contrast to the brief refreshing naps that characterize narcolepsy.
- Severe sleep inertia is the signature: stacked alarms across the room, family members required to wake the patient, and automatic behavior.
- Sleepiness produces microsleeps, memory lapses, and drowsy driving, and academic or occupational failure usually precedes the referral.
- Autonomic complaints are common, including orthostatic lightheadedness, cold hands and feet, headaches, and Raynaud-type phenomena.
- Cataplexy, sleep paralysis, and hypnagogic hallucinations are absent, which is the clinical line separating this from narcolepsy type 1.

## DIAGNOSTIC CRITERIA AT A GLANCE

- Self-reported excessive sleepiness despite a main sleep period lasting at least 7 hours, accompanied by at least one of three specific features.
- Those features are recurrent lapses into sleep within the same day, a nonrestorative main sleep period exceeding 9 hours, or difficulty being fully awake after abrupt awakening.
- Symptoms occur at least three times per week for at least 3 months with significant distress or impairment in cognitive, social, or occupational domains.
- Sleepiness cannot be better explained by another sleep disorder, by a substance, or by a coexisting mental or medical condition.
- Specify acute under 1 month, subacute 1 to 3 months, or persistent beyond 3 months, and grade severity by the number of impaired days per week.

## NEUROBIOLOGY & PHYSIOLOGY

- Unlike narcolepsy type 1, cerebrospinal fluid hypocretin-1 is normal, so orexin cell loss does not account for the sleepiness in this population.
- A subset shows a cerebrospinal fluid factor that potentiates GABA-A signaling, the rationale for trials of **flumazenil** and **clarithromycin**.
- Long sleep time variants may reflect a lengthened intrinsic circadian period together with abnormally slow dissipation of homeostatic sleep pressure.
- Polysomnography shows high sleep efficiency with short latency, and the MSLT typically yields mean latency under 8 minutes with fewer than two SOREMPs.
- Familial clustering is reported in roughly a third of cases, although no single causal gene has been established for the phenotype.
- Untreated sleepiness carries a several-fold increase in motor vehicle crash risk and is associated with obesity and depressive disorders.

## PSYCHOLOGICAL MECHANISMS

- Chronic unexplained sleepiness is repeatedly attributed to laziness or depression, producing shame, identity damage, and diagnostic delay of years.
- Patients build elaborate compensatory routines with stacked alarms, caffeine loading, and rigid scheduling that partially mask true severity.
- Behavioral activation and activity pacing help separate anergia driven by depression from genuine sleepiness driven by the sleep disorder.
- Illness acceptance work and disability accommodation planning reduce the catastrophic thinking that follows repeated occupational failure.
- Self-report diaries are unreliable during severe sleep inertia, so collateral report from a bed partner or parent is essential to assessment.

## DIFFERENTIAL & COMORBIDITY

- Narcolepsy type 1 features cataplexy and low hypocretin-1, while type 2 requires two or more SOREMPs with mean MSLT latency under 8 minutes.
- Exclude obstructive sleep apnea, insufficient sleep syndrome, delayed sleep phase, and shift work before diagnosing a central hypersomnolence.
- Major depression with atypical features, bipolar depression, and seasonal patterns commonly present with hypersomnia and must be assessed directly.
- Medications are a frequent cause: sedating antidepressants, antipsychotics, antihistamines, gabapentinoids, opioids, and cannabis products.
- Screen for hypothyroidism, anemia, traumatic brain injury, Kleine-Levin syndrome, and myotonic dystrophy in atypical or abrupt presentations.

## Treatment EVIDENCE-BASED MANAGEMENT

### PHARMACOLOGIC

- Modafinil 100-400 mg/day** or armodafinil 150-250 mg/day is first-line; counsel on reduced hormonal contraceptive efficacy and rare severe rash.
- Methylphenidate** and amphetamine derivatives are second-line when wake-promoting agents fail; monitor blood pressure, weight, and misuse risk.
- Soliamfetol 75-150 mg/day** and **pitolisant** are approved for narcolepsy and sleep apnea sleepiness and are used off-label here.
- Lower-sodium oxybate** is FDA-approved for idiopathic hypersomnia in adults, dosed nightly under a REMS with monitoring for respiratory depression.
- Clarithromycin** and **flumazenil** target the GABA-A potentiating factor and are reserved for refractory cases at specialist sleep centers.

### PSYCHOTHERAPY

- CBT** adapted for hypersomnia targets morning routines, activity scheduling, and the guilt and self-blame accumulated over years of misattribution.
- Behavioral sleep medicine sets a fixed wake time and limits time in bed, since extended sleep opportunity worsens rather than relieves inertia.
- Psychoeducation with family and employers reframes the problem as neurological rather than motivational and helps secure accommodations.
- Treat comorbid depression concurrently, favoring activating agents such as **bupropion** over sedating antidepressants in this population.
- Peer support and illness acceptance work reduce the isolation patients report while living with a poorly recognized central hypersomnia.

### ADJUNCT & SUPPORTIVE

- Track response with the **Epworth Sleepiness Scale**, where scores above 10 indicate pathologic sleepiness and a 3-point fall is meaningful.
- Confirm the diagnosis with 1 to 2 weeks of actigraphy, then overnight polysomnography followed immediately by a five-nap **MSLT**.
- Counsel explicitly about driving, document the conversation, and follow state requirements for reporting impaired drivers where they apply.
- Scheduled naps help far less than in narcolepsy because naps here are long and unrefreshing, so prescribe them selectively if at all.
- Arrange academic and workplace accommodations under the ADA: late start times, flexible scheduling, and protected breaks for rest.

- ☆ **CLINICAL PEARLS**
- Long naps that do not refresh point away from narcolepsy toward hypersomnolence.
  - Sleep drunkenness is the tell: ask who wakes the patient and how long it takes.
  - Rule out apnea and insufficient sleep before calling sleepiness central.

# References

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## NOTE

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