Insomnia (Sleeplessness) — Causes and Treatment

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Insomnia is a sleep disorder that is marked by difficulty in the initiation, maintenance, and consolidation of sleep, giving it poor quality and altered quantity. Symptoms of insomnia are seen in up to 30 % of the population, but only 10 % have severe symptoms that progress to chronic disease. The disease is largely idiopathic but genetic, environmental, social and psychiatric influences have been incriminated. Sometimes, it may be associated with a medical or psychiatric condition (secondary insomnia).

Definition of Insomnia

Insomnia is a sleep disorder that is characterized by difficulty in falling or staying asleep. The DSM V defines insomnia as the dissatisfaction with quality and quantity of sleep that is **characterized by a difficulty in the initiation and maintenance of sleep,** as well as an early morning awakening that is accompanied by the inability to go back to sleep.

Epidemiology of Insomnia

The worldwide incidence of insomnia, that is, people living with difficulty in sleeping, is about 30–35 %. **Approximately 10 % of the population suffers from a chronic or**

severe sleeping disorder that requires medical attention. In England, a steady increase in insomnia prevalence has been reported over the years, with the rate standing at 38.6 % in 2016. The trend is believed to be reciprocated worldwide.

Insomnia often is associated with the female gender (they have 1.4 times the likelihood of developing insomnia), an increased age (more common beyond 60 years), the existence of depression or unemployment, as well as economic inactivity and divorced/separated couples.

Etiology of Insomnia

According to the international classification of sleep disorders (ICSD–2) common etiologies of **insomnia include the following nine main issues:**

1. Idiopathic insomnia
2. Medical conditions such as cancer, chronic pain as in neurological conditions, arthritis, hyperthyroidism, gastroesophageal reflux disease or obstructive respiratory diseases that cause discomfort
3. Mental diseases such as depression, bereavement
4. Drug and substance abuse. Mainly stimulants such as caffeine and other drugs such as alcohol
5. Inadequate sleep hygiene (daytime napping and watching TV in bed)
6. Behavioral insomnia where there is an alteration of the sleep cycle
7. Environmental influences of sleep such as an increase in night temperatures, light, and noise
8. Use of medications such as blood pressure medications, common cold, asthma and allergy drugs
9. May be caused by an underlying sleep disorder such as restless leg syndrome

Classification of Insomnia

Acute insomnia

This form of insomnia is mainly characterized by a **transient/short term state of lack of sleep for less than one month**. It takes two forms:

- **Adjustment insomnia** occurs due to situational stress such as working late into the night to beat a deadline, but resolves upon removal of the stressor.
- **Transient insomnia** occurs in association with a certain trigger agent.

Chronic insomnia

This is an alteration in sleep quality or quantity for more than three nights in a week over a month or longer. It is commonly associated with a medical or psychiatric condition and can be classified into:

Primary insomnia

This is insomnia that is not associated with other health conditions.

Secondary insomnia

This is a sleep disturbance in association with other medical or psychiatric conditions such as asthma, depression, cancer, and heartburn.

Sleep onset insomnia

This state is seen when you have difficulties falling asleep despite feeling tired and sleepy. **Normally, one takes 15–20 minutes to fall asleep after going to bed**.

Sleep maintenance insomnia

This is a type of insomnia where you have difficulty staying asleep. **It is normal to wake up during sleep, but normal people hardly remember the interruption**. In insomniacs, they remain awake for hours in the night and wake up in the morning while still feeling tired.

Pathophysiology of Insomnia

Neurophysiology of the sleep-wake cycle

The sleep- and wakefulness cycle is tightly controlled by the brain via response to environmental light and specific time in the 24-hour cycle. **The brain's sleep centers are in the pons, medulla, and hypothalamus.** Communication exists between these regions and messages are carried via norepinephrine, serotonin, dopamine and acetylcholine neurotransmitters.

Sleep is thus regulated by homeostasis that induces sleep after long periods of wakefulness. This is achieved by activation of the inhibitory neurotransmitter GABA that inhibits wakefulness signals in the brainstem. The circadian rhythm, on the other hand, inhibits the homeostatic signal with the presence of environmental triggers such as light. This is achieved by activation of the suprachiasmatic nucleus that triggers inhibition of melatonin production from the pineal gland, causing wakefulness.

Normal sleep stages

Normal sleep stages as characterized by polysomnography include the state of Wakefulness, Stages 1–4 NREM.

Wakefulness

Low-voltage, fast-frequency EEG pattern (Alpha pattern), voluntary eye movements, tonic muscle tone.

Stage 1 NREM sleep

This stage **describes a small increase in EEG amplitude** with slowing of EEG frequency and represents the transition from wakefulness to sleep; it makes up 5 % of sleeping time.

Stage 2 NREM sleep

Stage 2 NREM sleep is **characterized by an increase in amplitude and further slowing of EEG frequencies**, the presence of 'K complexes' (isolated large-amplitude slow waves) and 'sleep spindles' (episodic bursts of fast EEG activity) with a reduction in muscle tone.

Stage 3 NREM sleep

In this stage is observed a large-amplitude, slow EEG activity also known as "slow-wave

sleep" that comprises 20–50 % of a 30-second epoch, low muscle tone, and an EOG pattern that mirrors the EEG activity.

Stage 4 NREM sleep

Stage 4 mainly contains stage 3's sleep issues, except the delta activity constitutes > 50 % of an epoch. Both increase duration with sleep deprivation.
REM sleep represents 25 % of total sleep and is characterized by the occurrence of dreams and phasic rapid eye movement. The muscle tone is essentially absent. There is an increase in respiration and pulse rate with increased brain oxygenation.

Pathogenesis of insomnia

Two theories have been put forward to explain the occurrence of insomnia, and both agree that the sleep disorder arises from the interaction of biological, physical, psychological and environmental factors.

Hyperarousal theory

This theory postulates that **patients suffering from insomnia have increased brain arousal during NREM sleep,** thus they have reduced activation of key brain areas, which, once activated, cause inhibition of brain pathways and induce sleep.
This is supported by the fact that they have higher levels of temperature, cortisol, ACTH, and adrenaline which increase arousal. Normal people that are deprived of sleep do not demonstrate hyperarousal. This indicates that insomnia has an underlying hyperarousal state.

Cognitive-behavioral model

This theory states that insomnia has a predisposing factor, such as genetic predisposition among family members, that interacts with precipitating factors such as bereavement, depression and medical conditions. In the presence of perpetuating factors such as cognition and behavioral response that alters homeostasis (daytime nap), chronic insomnia is induced that is irreversible even with the removal of the precipitant.

Clinical features of Insomnia

The diagnosis of these patients heavily relies on thorough history taking.

Sleep history

A sleep history provides the assessment of sleeping patterns, such as a two-week sleep log to define patterns of sleep and day activities. These include sleep hygiene trends such as day time naps, caffeine intake, watching television in bed and daytime tiredness, as well as any history of awakening at night and difficulty falling back asleep. That includes any kind of disturbances during sleep, such as noisy and lighted rooms.

Medical history

A medical history provides any history of known medical conditions that may induce insomnia. This is **usually done with a general medical questionnaire that contains relevant indicators**, such as any bothering pain, known malignancy or chronic disease

that may cause discomfort, as well as the history of medication use such as theophylline and antidepressants that would alter the sleep pattern.

Psychiatric history

A general psychiatric questionnaire exists to rule out any mental illnesses that may predispose one to insomnia. It includes:

Loss of interest or low mood

- Depression
- Anxiety and irritability
- Difficulty in paying attention

Social history

- How the person relates to other people
- Divorced or separated

Family history

- History of similar disease in the family.

Note: the patients should be examined for findings that may indicate the cause of insomnia, such as inadequate airway in obstructive sleep apnea.

Investigations of Insomnia

Polysomnography	Entails • Electroencephalography (EEG) that demonstrates brain activity. • Electrooculography (EOG) that demonstrates eye movement during an assessment. • Electromyography (EMG) measures muscle tone.
Actigraphy	• Records the gross motor activity providing an indirect measure of sleep and wake pattern • Mainly used for diagnosing circadian rhythm disorders
Genetic testing	• Sequencing of the PRNP gene
Record sleep diary	Patient records the time when: • He/She goes to bed. • Lies awake on the bed. • Fell asleep. • Woke up from sleep. • Other events to be monitored include daytime naps, exercises, and stimulant use.
Brain imaging	• PET studies have been carried out to study hypermetabolic thalamus and cortex.

Differential Diagnosis

Obstructive sleep apnea	• A sleep disorder accompanied by partial or complete airway obstruction leading to pauses in breathing and desaturation, making it difficult to sleep • Accompanied by physical findings consistent with the disease such as a large neck, nighttime snoring and Malampati class III or IV of the airway
Major depressive disorder	• Associated with a low mood that may result from a condition that may not be a feature of insomnia at onset.
Restless leg syndrome	• A neurological condition in which a person has an uncomfortable sensation of needing to move his/her legs. Differentiated on multiple sleep latency testing (MSLT) is abnormal.
Circadian rhythm disorders	• Differentiated on multiple sleep latency testing (MSLT) is abnormal.
Substance abuse disorder	• Accompanied by the history of substance use and other symptoms, such as psychosis, that are related to substance use.

Management of Insomnia

The treatment of the disease entails non-pharmacological approaches such as sleep hygiene and behavioral therapy or pharmacological therapy with benzodiazepines. The disease is commonly associated with anxiety and depression which should be managed, too.

Non-pharmacological therapy

The doctor-patient relationship is important in treating and preventing the emergence of complications such as psychological dependence and misuse of hypnotics.
Counseling and psychotherapy are useful, especially when the cause is identified as anxiety, depression or emotional instability. This treatment can be instituted together with other therapies.

Sleep hygiene

Includes, e.g., stimulant control treatment like avoiding alcohol, caffeine and watching TV in bed.

Stimulus control therapy

The patient is trained to associate the bed with sleep and sexual activities only. This is achieved by going to bed when sleepy, refraining from day time naps and engaging in light soothing activities such as knitting and listening to soft music while waiting to get sleepy instead of lying in bed awake.

Sleep restriction therapy

With this therapy, the hours in bed are controlled, and thus sleep latency is shortened, efficiency increased and bedtime gradually increases.

Note: Engage your body in relaxing activities such as meditation and yoga that relieve tension on your body and relax your mind. They also help in initiating sleep.

Pharmacological therapy

The pills should be taken shortly before bedtime and in combination with other methods of sleep induction.

Over the counter sleep medications (scopolamine and diphenhydramine

They rely on the anti-histaminergic activity of the ingredients to induce drowsiness. Some are used in combination with analgesics such as acetaminophen or alcohol. Their effects are known to spill over into the next day causing a hangover effect.

Sedative hypnotic drugs (benzodiazepines such as estazolam, flurazepam, and quazepam)

Short-acting zolpidem and zaleplon are the first-line agents for the management of insomnia. They are administered for four weeks and are tapered off slowly to avoid adverse effects of rebound insomnia, hangover effect, dependence and alteration of memory.

Sedating antidepressant agents (amitriptyline, trazodone, zaleplon, and lunesta)

Their mechanism of action is like that of benzodiazepines but they possess a safer drug profile.

Melatonin receptor agonist sleeping pills (Ramelton)

It is the newest drug in use and mimics the body’s regulation of the hormone melatonin**.** It is best used for people with problems in initiating sleep and not good for those with

trouble staying asleep. The drug has been associated with some level of dependence.

Acupressure for insomnia

Holding acupressure points while taking deep breaths for a few minutes before going to bed is believed to relieve and prevent insomnia. Acupressure points examples include the joyful sleep (a point inside the heel just below the inner ankle bone) or calm sleep (outside the heel just below the outer ankle bone).

Complications of Insomnia

The medications used in the management of insomnia are associated with certain adverse effects:

- Rebound insomnia that is seen after sudden discontinuation of short-acting agents
- Anxiety
- Seizures
- Psychosis
- Delirium tremens

To avoid these adverse effects, it's recommended to taper the doses over several weeks.
If untreated, the condition leads to poor quality of life, inability to concentrate, loss of memory and increased mortality.

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