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Review article

An overview of insomnia and its treatment

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Abstract

Background: Insomnia is a condition of unsatisfactory quality and/or quantity of sleep, with difficulty falling asleep, remaining asleep, or waking early and being unable to return to sleep. Transient insomnia occurs at times of stress but frequently accompanies various medical and psychiatric conditions and is often associated with substance misuse. Insomnia affects one-third of adults occasionally, and 9 to 12 per cent on a chronic basis.

Objectives: To develop a better understanding of insomnia as a whole with its different sub types and various treatment modalities for effective management of cases with insomnia.

Methods: A search was carried out on the web including Google Scholar, Medline, HINARI and several other web portals for English-language articles containing the following keywords- sleep, insomnia, epidemiology of insomnia, etiology of insomnia, pathology of insomnia, presentation of insomnia and treatment of insomnia. Relevant chapters in some authoritative textbooks were also consulted.

Results: Insomnia is more common in women, the elderly, shift workers and in patients with medical and psychiatric conditions. For diagnosis of insomnia disorder, DSM-5 requires insomnia to be present for at least three nights per week for 3 months and to cause clinically significant distress or functional impairment. The insomnia should not be adequately explained by another sleep or mental disorder, and not be attributable to the effects of a substance or medical condition. Both pharmacological and non-pharmacological approaches can be used to treat insomnia. Among pharmacological agents, short and intermediate acting benzodiazepines and benzodiazepine receptor agonists are the mainstay. Non-pharmacological interventions include a range of cognitive and behavioral techniques, including sleep hygiene. Hypnotic drugs were widely prescribed in the past, but non-pharmacological approaches are now recommended as the first-line treatment.

Conclusions: Sleep disturbance is ubiquitous among general people and insomnia is the most common type of sleep disorder. An individual at any age can develop insomnia as a primary sleep disorder, as part of another mental disorder or as a complication of a medical illness. The presentation differs among patients and so does the choice of appropriate treatment modality. Nevertheless, most patients significantly benefit from therapeutic interventions.

Declaration of interest: None

Keywords: Insomnia; presentation of insomnia; treatment of insomnia; review

Introduction

Insomnia is a condition characterized by dissatisfaction with sleep quantity or quality associated with persistent difficulties with initiating or maintaining sleep, or inability to return to sleep after early morning awakening, that occurs despite adequate opportunity for sleep and results in some forms of functional impairment. Although non-restorative sleep is often included as a symptom of insomnia, it has different epidemiological and functional correlates than other insomnia symptoms, including higher prevalence in young adults and a greater degree of daytime impairments such as sleepiness and fatigue.[1] Because different people need different amounts of sleep, insomnia is defined by the quality of sleep and how we feel after sleeping. Even if we are spending eight hours a night in bed, if we feel drowsy and fatigued during the day, we may be experiencing insomnia.[2] Transient insomnia occurs at times of stress or as 'jet

lag'. Short-term insomnia is often associated with personal problems for example, illness, bereavement, relationship difficulties or stress at work. Sleep-onset insomnia can be a relatively common complaint among adolescents. Insomnia in clinical practice is usually secondary to other disorders, notably painful physical conditions, depressive disorders and anxiety disorders; it also occurs with excessive use of alcohol or caffeine, and in dementia. It can also be provoked by prescribed drugs. To understand sleep and its disorders, it helps to begin with the sleep's three basic characteristics: (1) Sleep is a process required for proper brain function. Failure to sleep impairs thought processes, mood regulation and a host of normal physiological functions. (2) Sleep is not a single process; there are several distinct types of sleep. These different types of sleep differ both qualitatively and quantitatively. Each type of sleep has unique characteristics, functional importance and regulatory mechanisms. Selectively depriving one particular type of sleep produces compensatory rebound when an individual is allowed to sleep ad lib. (3) Sleep is not a passive process; sleep can be associated with a high degree of brain activation and metabolism. Several physiological mechanisms regulate sleep and when these systems go awry, sleep disorders occur.[3]

The functions of sleep and impact of sleep deprivation

Debate continues about the various theories concerning the functions of sleep, each of which has emphasized physical and psychological restoration and recovery, energy conservation, memory consolidation, discharge of emotions, brain growth and various other biological functions including somatic growth and repair and maintenance of immune systems.[4] The adverse effects of chronic sleep loss (considered to be common in modern society) on mood, behavior and cognitive function can be substantial, with various consequences for personal, social, occupational, educational and family functioning. Large sample meta-analyses have shown that patients with insomnia have mild or moderate dysfunction in attention, episodic memory, working memory and executive function compared with healthy controls.[5]

Epidemiology

Population-based estimates indicate that about one-third of adults report insomnia symptoms, 10-15% experience associated daytime impairments and 6-10% have symptoms that meet criteria for insomnia disorder. Insomnia disorder is the most prevalent of all sleep disorders in primary care settings, approximately 10-20% of individuals complain of significant insomnia symptoms.[6] Although insomnia may be categorized as either a symptom or an independent disorder, it is most frequently considered a co-morbid condition in relation with another medical condition or mental disorder.[7] For instance, 40-50% of individuals with insomnia also present with a co-morbid mental disorder. Risk factors include depression, female sex, older age, lower socioeconomic status, concurrent medical and mental disorders, marital status (greater risk in divorced/ separated vs. married or never married individuals) and race (greater risk in African American vs. white race).[8] Prevalence among older adults has been estimated at up to 25%.[9] Insomnia follows a chronic course in 40-70% of individuals over 1-20 years.[10,11] Sleep maintenance symptoms are most prevalent among individuals with insomnia (approximately 50-70%), followed by difficulty initiating sleep (35-60%) and non-restorative sleep (20-25%).[12]

Etiology of insomnia

Many patients report having been marginal light sleepers before developing insomnia.[13] Sleep disturbance often arises during life change or stress and that may represent a normal transient disruption of sleep. However, secondary factors, such as anxiety over sleep and faulty sleep-wake conditioning, may exacerbate and maintain the insomnia as a chronic problem when sleep itself becomes a focus for concern. People with insomnia may be hyper aroused relative to normal sleepers, for example, having higher levels of cortisol and ACTH and also find it difficult to down-regulate their arousal at bedtime.[14,15,16] Insomnia in clinical practice is usually secondary to other disorders, notably painful physical conditions, depressive disorders, anxiety disorders and dementia. It also occurs with misuse of alcohol, caffeine and other drugs or prescribed medications. In about 15% of cases of insomnia, no cause is found.[17]

Pathophysiology of insomnia

Although insomnia is considered a sleep disorder, its pathophysiology suggests hyper arousal during sleep and wakefulness.[18] Insomnia results due to an imbalance between sleep inducing neurotransmitters gamma-aminobutyric acid (GABA) and adenosine, and the arousal neurotransmitters (noradrenaline, serotonin, acetylcholine, orexin and dopamine).[19] In local sleep theory proposed by Krueger et al.[20] sleep is defined as a fundamental emergent property of highly interconnected neurons or cortical columns. Local sleep propensity and slow wave amplitude are posited to be dependent on accumulation of sleep-regulatory substances (tumor necrosis factor-α and IL-1 β)[21,22] resulting from prior neuronal use. Synchronous firing within cortical columns is postulated to propagate slow wave activity in adjacent regions through humoral and electric interactions, leading eventually to a global sleep state in the entire organism.

Clinical features

The clinical presentation is commonly of a frustrated patient, trapped in a vicious circle of anxiety and poor sleep, who reports having 'tried everything'.[23,14] Typical reports relate to light sleep and sleep felt to be unrestorative. There can be cognitive effects, such as fatigue, sleepiness, inattention and some impairments in performance or emotional effects, such as irritability and anxiety.[14]

Common symptoms are: Difficulty falling asleep, waking up during the night, waking up too early, general tiredness, daytime tiredness or sleepiness, not feeling well-rested, problems with

concentration or memory, difficulty focusing on tasks, irritability, depression or anxiety, increased errors or accidents, ongoing worries about sleep.

Insomnia subtypes

Neither DSM-5 nor ICSD-3 formalizes diagnostic criteria for clinical and pathophysiological insomnia sub types. Nonetheless, ICSD-3 describes the following sub types briefly. Understanding the sub types is essential for creating an effective treatment plan.

Psychophysiological insomnia: Psychophysiological insomnia (PPI) involves conditioned arousal associated with the thought of sleeping. Objects related to sleep (e.g., the bed, the bedroom) likewise have become conditioned stimuli that evoke insomnia. Daytime adaptation is usually good, however, there can be extreme tiredness. Other features characteristic of PPI include: (1) excessive worry about not being able to sleep (2) trying too hard to sleep (3) rumination—inability to clear one's mind while trying to sleep (4) increased muscle tension when getting into bed (5) other somatic manifestations of anxiety (6) ability to fall asleep when not trying to (e.g., when watching television) (7) sleeping better away from own bedroom (including the sleep laboratory).

Idiopathic insomnia: Idiopathic insomnia characterizes patients with a lifelong inability to obtain adequate sleep. The insomnia predates any psychiatric condition and other etiologies must be ruled out or treated, including psychophysiological insomnia, environmental sleep disturbances, and practices constituting poor sleep hygiene. It is assumed that there is a defect in the sleep-wake system.

Paradoxical insomnia: Paradoxical insomnia, at its core, involves a dissociation between sleep and its usual attendant unconsciousness. In Paradoxical insomnia, an individual thinks he or she is awake and having insomnia, even though brain electrophysiological activity pattern is consistent with the correlates of normal sleep.

Inadequate sleep hygiene: Inadequate sleep hygiene refers to insomnia produced by behaviors that are not conducive to good sleep, that is when performance of daily living activities that are inconsistent with the maintenance of good quality sleep. For example, consuming caffeine or nicotine at night or engaging in excessive emotional or physical stimulation within a few hours of bedtime, taking daytime naps and maintaining a large variation of the daily sleep-wake schedule.

Behavioral insomnia of childhood: Behavioral insomnia of childhood, or childhood insomnia, results from the child's dependency on specific stimulation, objects or setting for initiating sleep or returning to sleep. Without presence of the teddy-bear or parent, sleep onset is significantly delayed. The child refuses or delays going to bed by strategies like getting up to use the bathroom, requests for another drink, wanting another bedtime story, or complaints of hunger, fear of the dark, monsters under the bed, or some other factitious creation.

Insomnia co-morbid with mental disorder: Insomnia co-morbid with mental disorder is very common. Thirty-five percent of patients seen in sleep disorder centers with insomnia as their chief complaint, had a mental disorder and half of these patients had a mood disorder. Ninety percent of patients with major depressive disorder (MDD) have insomnia. Insomnia is a risk factor for depression on 3-year follow-up (odds ratio 3.95), for suicide in patients with MDD and 1 month after a trauma, is predictive of PTSD within 1 year.

Insomnia co-morbid with medical condition: Insomnia accompanies many medical and neurological conditions. All medical conditions producing pain can (and usually do) disturb sleep while poor sleep lowers pain threshold. In other medical conditions, the sleep disturbance appears to be secondary. For example, sleep-related gastro-esophageal reflux disease (GERD) and chronic obstructive lung disease can interfere with sleep onset and sleep maintenance. Neurodegenerative diseases are frequent associates of various sleep disorders, including insomnia.

Insomnia due to drug or substance: Many prescription drugs, even when taken properly, can disturb sleep. These include antidepressants (i.e., fluoxetine), antihypertensives, corticosteroids, hypolipidemic medications, antiparkinsonian drugs, decongestants (i.e., pseudoephedrine), anorectics, stimulants and antiepileptic medications. Some sedatives and alcohol promote sleep onset initially but later cause fragmented sleep and rebound insomnia. Caffeine and theobromine present in coffee, chocolate and illicit substances, particularly stimulants (such as cocaine and amphetamines) interfere with sleep onset, sleep maintenance and decrease total sleep time.

Short sleepers: People who fall under this category require less than 5 hours of sleep per 24-hour period in order to maintain normal daytime functioning and mood. The patient may be distressed about not sleeping. However, if the short sleep duration does not provoke adverse daytime consequences, functional impairment or is associated with co-morbid conditions, criteria for insomnia disorder are not met.

Assessment of insomnia

Key elements of the assessment include the patient's sleep characteristics, daytime behaviors, medical-ps chiatric history, symptoms of other sleep disorders, and medications.[24] The "3-P" model, comprising the predisposing, precipitating and perpetuating factors, is a useful heuristic framework for assessment.[25] Clinicians can also use several tools to help assess insomnia. Most important are prospective sleep-wake diaries, which evaluate the timing and variability of sleep and may identify targets for behavioral interventions.[26]

Table 1: Assessment of insomnia

Evaluation of insomnia sleep history
The evaluation of insomnia rests on a careful clinical history.
Sleep and wakefulness affect each other in complex ways.
Key elements of the sleep history include:
• Temporal aspects of sleep: Times at which patient goes to bed, attempts to sleep, wakes up, gets out of bed
• Quantitative aspects of sleep: Sleep latency (time it takes to fall asleep), number and duration of awakenings, wakefulness after sleep onset, total sleep time

- Qualitative aspects of sleep: Subjective sleep quality, satisfaction
- Behavioral and environmental factors: Non-sleep activities in bed (phone, computer, TV), environment (temperature, light, sound), bed partners and pets, perceived causes of awakening
- Symptoms of other sleep disorders
- Daytime causes and consequences of disturbed sleep

Medical and psychiatric history

- Medical disorders
- Psychiatric disorders
- Medications

Other tools and tests

- Sleep-wake diary
- Video recording
- Wrist actigraphy
- Polysomnography (sleep study)
- Cerebrospinal fluid orexin (hypocretin) levels

Sleep-related scales

- Insomnia Severity Index
- Pittsburgh Sleep Quality Index

Diagnosis

According to DSM-5, for the diagnosis of insomnia disorder one or more of the following symptoms must be present: (a) difficulty initiating sleep, (b) difficulty maintaining sleep, or (c) early-morning awakening with inability to resume sleep. In case of children, insomnia can manifest as resistance to the caregiver designation of bed time and/or difficulty sleeping without caregiver intervention. The symptoms should cause clinically significant distress or impairment of social, occupational, educational, academic, behavioral or other important areas of functioning. Sleep difficulty should occur three times per week (or more) and present for 3 months or longer. The insomnia must not be better explained by another sleep, mental or medical disorder, must not be attributable to a medication or substance use and must persist when adequate opportunity for sleep occurs.

Differential diagnosis

The differential diagnosis of insomnia includes other sleep and medical disorders. Up to 50% of adults with obstructive sleep apnea (OSA) also complain of insomnia. Circadian rhythm sleep disorders, such as delayed sleep phase disorder and shift work disorder, abnormal sleep timing, restless leg syndrome often result in difficulty falling asleep. A separate insomnia diagnosis is not needed for all patients with medical, psychiatric or other sleep disorders, who have insomnia symptoms and should be made only if the symptoms are severe or constitute an independent focus of clinical attention.

Treatment of insomnia

The goals of insomnia treatment are to improve quantitative and qualitative aspects of sleep, to reduce the distress and anxiety associated with poor sleep and to improve daytime function.[24] Treatment of insomnia include two broad approaches: pharmacological and non-pharmacological. Hypnotic drugs were widely prescribed in the past, but non-pharmacological approaches are now recommended as first-line treatment. Treatment options are briefly described below.

Pharmacological treatment

Traditionally, insomnia has been treated pharmacologically. Benzodiazepines replaced barbiturates during the 1960s and were the most commonly prescribed sedative hypnotic medications for treating insomnia as they were safer in overdose and less addictive. However, from the mid-1970s, problems of tolerance and dependence became apparent. Longer-acting hypnotics were prone to carry-over effects of morning lethargy and shorter-acting drugs to 'rebound insomnia'.[27] Although BDZs when used for short periods/intermittently can maintain effectiveness, these are not the treatment of choice in chronic insomnia.[28] Besides they are not suitable for older adults and patients with respiratory difficulties. Eventually BZRAs, often called Z drugs (such as zolpidem, zaleplon, zopiclone and eszopiclone), have replaced BDZs as the standard for hypnotics because of their minimal residual effects and low potential for tolerance and dependence. Subsequent development of doxepin (a low-dose tricyclic), ramelteon (a melatonin receptor agonist) and suvorexant (an orexin antagonist) have expanded the range of approved agents for treating insomnia. However, unapproved use of sedating antidepressants (e.g., trazodone) and antipsychotics (e.g., quetiapine) are common. Non-prescribed sleep aids include sedating antihistamines, L-tryptophan and melatonin. Most hypnotic medications are approved for short-term (2-4 weeks) use, but zolpidem, eszopiclone and ramelteon are exceptions. When properly used, hypnotics can provide immediate and adequate relief from sleeplessness. Insomnia however, usually returns upon discontinuation of dosing.

Table 2: Benzodiazepine receptor agonist (BzRA) drugs

Class/drug	T_{Max} (hour)	Elimination half-life (hour)	Usual hypnotic dose (mg)	Approved for insomnia
Benzodiazepines				
Triazolam	1-2	1-2	0.125- 0.25	Yes
Temazepam	1-2	1-2	15-30	Yes
Estazolam	1.5-2	1.5-2	1-2	Yes
Quazepam	2-3	2-3	7.5-15	Yes
Flurazepam	1.5-4.5	1.5-4.5	15-30	Yes
Alprazolam	0.6-1.4	0.6-1.4	-	No
Lorazepam	0.7-1	0.7-1	1-4	No
Clonazepam	1-2.5	1-2.5	0.5-3	No
Benzodiazepine receptor agonists				
Zaleplon	0.5-2	0.8-1.3	5-20	Yes
Eszopiclone	0.5-2	5-8	1-3	Yes
Zolpidem	0.5-1.5	1.4-4.5	5-10	Yes

Sources: FDA-approved prescribing information and sources [29,30]

Table 3: Other drugs commonly used as hypnotics

Class/drug	T_{Max} (hour)	Half-life (hour)	Usual hypnotic dose (mg)
Antidepressants			
Doxepin	1.5-4	10-30	3-6
Amitriptyline	2-5	5-45	10-100
Trazodone	1-2	7-15	25-150
Mirtazapine	1-3	20-40	7.5-30
Antipsychotics			
Olanzapine	4-6	20-54	2.5-20
Quetiapine	1-2	6	25-50
Melatonin agonists			
Melatonin	0.3-1	0.6-1	0.5-3
Ramelteon	0.5-1.5	1-2.6	8
Antihistamines			
Diphenhydramine	1-4	4-8	25-50
Doxylamine	2-3	10	25
Anticonvulsants			
Gabapentin	1.6-3	5-9	100-900
Pregabalin	1.5	6.3	50-300

Sources: FDA-approved prescribing information and sources [31,32]

Non-pharmacological treatment

Cognitive-behavioral therapy for insomnia (CBTi): This treatment modality combines behavioral and cognitive techniques to overcome dysfunctional sleep behaviors and misperceptions, distorted, disruptive thoughts about sleep. A treatment plan is designed using cognitive and behavioral techniques deemed relevant and appropriate for the patient. Five meta-analyses and numerous systematic reviews have demonstrated that CBT is associated with large effect size changes (measured in standardized z-scores) in the primary symptom measures of sleep latency (difficulty getting to sleep) and wake time after sleep-onset (difficulty remaining asleep).[33,34] Recent controlled studies have shown that CBT may be effective in general practice settings with nurses delivering the intervention according to a standard protocol.[35,36] Studies have repeatedly shown that short-term benefits of CBTi are similar to that of medications while CBTi provides sustained benefits, even 36 months after treatment, and does not cause rebound insomnia on stopping.

Sleep education and universal sleep hygiene: The simple provision of information ameliorates the sense of being out of control. Inaccurate attributions are challenged and misunderstandings corrected by understanding what sleep is, how common insomnia can be, how sleep changes with age, good practices, and some facts about insomnia. The focus of universal sleep hygiene is on modifiable environmental and lifestyle components that may interfere with sleep as well as behaviors that may improve sleep.

Table 4: Sleep hygiene

Do	Don't
Maintain regular hours of bedtime and arising.	Take naps.
If you are hungry, have a little snack before bedtime.	Watch the clock so you know how bad your insomnia actually is.
Maintain a regular exercise schedule.	Exercise right before going to bed to.
Give yourself approximately an hour to wind down before going to bed.	Watch television in bed.
If you are preoccupied or worried about something at bedtime, write it down and deal with it in the morning.	Eat a heavy meal before bedtime.
Keep the bedroom cool.	Drink tea or coffee in the afternoon and evening.
Keep the bedroom dark.	Smoke a cigarette if you cannot sleep.
Keep the bedroom quiet.	Use alcohol to help in going to sleep.
	Read in bed when you cannot sleep.
	Eat in bed.
	Exercise in bed.
	Use computer or cellphone in bed.

Stimulus control therapy: Stimulus control therapy is a deconditioning paradigm developed by Richard Bootzin and colleagues at the University of Arizona. By attempting to undo conditioning that undermines sleep, stimulus control therapy helps reduce both primary and reactive factors involved in insomnia. The rules attempt to enhance stimulus cues for sleeping and diminish associations with sleeplessness.

Sleep restriction therapy: Sleep restriction therapy is a strategy designed to increase sleep efficiency by decreasing the amount of time spent awake while lying in bed. Developed by Arthur Spielman, this therapy specifically targets those patients who lay awake in bed unable to sleep. Restricting time in bed can help consolidate sleep. When sleep efficiency reaches 85% (averaged over five nights), time in bed is increased by 15 minutes.

Relaxation therapy and biofeedback: Self-hypnosis, progressive relaxation, guided imagery, deep breathing exercises, biofeedback, meditation, autogenic training are all effective if they produce relaxation. The goal is to find the optimal technique for each patient. Biofeedback provides stimulus cues for physiological markers of relaxation and can increase self-awareness. Relaxation techniques readily lend themselves to being combined with sleep hygiene and stimulus control therapies.

Cognitive control: This technique aims to deal with thought material in advance of bedtime and to reduce intrusive bedtime thinking. The person with insomnia is asked to set aside 15 to 20 min in the early evening to rehearse the day and to plan ahead for tomorrow, thus putting the day to rest. It is a technique for dealing with unfinished business and may be most effective for rehearsal, planning and self-evaluative thoughts which are important to the individual and which, if not dealt with, may intrude during the sleep-onset period. Cognitive restructuring: Cognitive restructuring challenges faulty beliefs which maintain wakefulness and the helplessness which many people with insomnia report. It appears to work through appraisal by testing the validity of assumptions against evidence and real-life experience. If maladaptive cognitions, for example 'I am going to be incapable at work tomorrow' are not challenged, they will create high levels of preoccupation and anxiety and sleep is unlikely to occur.

Thought suppression: Thought-stopping and articulatory suppression attempt to interrupt the flow of thoughts. No

attempt is made to deal with thought material per say, but rather to attenuate thinking. With articulatory suppression, the patient is instructed to repeat, sub vocally, the word 'the' every 3s. The type of material most likely to respond is repetitive but non-affect-laden thoughts, not powerful enough to demand attention.

Paradoxical intention: This is a cognitive technique with conflicting evidence regarding its efficacy. The theory is that performance anxiety interferes with sleep onset. So, when the patient tries to stay awake for as long as possible, rather tries to fall asleep, performance anxiety will be reduced and sleep latency will improve.

Advice about management

Non-pharmacological treatment using CBT procedures should be preferred over pharmacological treatment, in cases of severe persistent insomnia. Hypnotic agents should be recommended mainly for short-term or occasional use. The practitioner should be aware of residual effects and potential problems of withdrawal and dependency.

Course and prognosis

There has been little research on the natural course of insomnia. However, untreated psychophysiological insomnia can last for decades, and may gradually worsen over time. Indeed, there is a developmental trend for sleep pattern to deteriorate, with increasing age. On the other hand, delayed sleep-phase syndrome and insufficient sleep hygiene can be associated with lifestyle problems and may ameliorate as these are resolved.

Conclusions

Insomnia is an almost invariable feature and complication of psychiatric disorders from childhood to old age, with the risk of further reducing the individual's capacity to cope with their difficulties. Persistent and severe insomnia represents a considerable public health concern. There is insufficient knowledge of the natural course of transient sleep disorders. Although the association of life events and stressors with the onset of insomnia is well-established, systematic research is required to establish the 'setting conditions' for the secondary maintenance of insomnia beyond an initial normative reaction to events. Nevertheless, the patients should be advised against their tendency to maximize opportunity to sleep when insomnia symptoms develop in order to facilitate the return of normal sleep pattern.

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