Understanding Insomnia in the Primary Care Setting: A New Model

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IS INSOMNIA A DISORDER OF AROUSAL?

Prevalence of Insomnia

Insomnia is the most common sleep disorder. Its prevalence varies according to the definition. One-fourth to one-third of the general population reports complaints of difficulty with falling and/or staying asleep whereas ten percent seek medical help for insomnia. Insomnia occurs more frequently with increasing age, in women, in patients of lower socioeconomic status, and those reporting psychologic disturbances. Further, insomnia is associated with increased morbidity, including hypertension, as well as with the potential development of major psychiatric disorders. Chronic insomnia is difficult to treat and has major psychosocial consequences, including its frequent negative impact on the patient’s family and employment.

Historical Perspectives on Insomnia – The Internalization Hypothesis

Traditionally, the field of sleep medicine, driven by patient complaints, has focused its efforts to improve the quality and quantity of nighttime sleep of insomniacs with pharmacotherapeutic and/or psychotherapeutic techniques. The main outcome variables in sleep pharmacology studies are the subjective and objective estimates of the increase in sleep length. This approach, to some extent, is responsible for the common perception that insomnia is a disorder of sleep loss.

The association of insomnia to emotional and psychiatric factors was documented and reported, even from the early stages of the sleep disorders medicine field, in the 1970s. In clinical and psychometric studies, it was reported that insomnia is frequently associated with depression, anxiety, rumination, and inhibition of emotional expression. At about the same time, other studies pointed to the presence of increased physiologic activation, such as increased heart rate, peripheral vasoconstriction, elevated rectal temperature, and increased body movement before and during sleep. These findings led to the formulation of the “internalization hypothesis” (Figure 1). This hypothesis suggested that insomniacs are individuals that,
Preliminary data demonstrated a positive correlation between polysomnographic measures of sleep disturbance at night and 24-hour urinary excretion of cortisol and catecholamines. Also, in another study where adrenocorticotropic hormone (ACTH) and cortisol were measured every half-hour for 24-hours, insomniacs demonstrated higher levels of ACTH and cortisol compared to normal sleepers. Although the hypersecretion was more marked during the evening and the first part of the night, it was present throughout the 24-hour sleep-wake cycle. In contrast to these findings, studies that have assessed the effects of nonstressful total or partial sleep deprivation in young, normal sleepers, cortisol was either unaffected or slightly decreased. Furthermore, the pattern of secretion of stress hormones and fatigue-inducing cytokines is different in insomnia vs. sleep deprivation. For example, it has been shown that in sleep deprivation, decreased HPA axis activity and increased secretion of fatigue-inducing cytokines is associated with deep sleep and sleepiness whereas in insomnia, increased HPA axis activity and increased cytokine secretion is associated with poor sleep and fatigue. In summary, data from these polysomnographic, EEG, physiologic, and stress-axis studies suggest that insomnia and sleep deprivation are two distinct states.

Is Sleep Loss or Disturbance a Manifestation of Insomnia?

The first polysomnographic studies confirmed that objectively insomniacs slept less compared to normal sleepers although they tended to exaggerate the amount of sleep loss. It soon became clear that sleep loss was not the cause of insomnia disorder but one of its associated symptoms. Sleep loss in normal sleepers is associated with increased sleepiness the next day as measured by a multiple sleep latency test (MSLT – measures the mean time it takes to fall asleep when given several opportunities to take a nap during the day.) In contrast, insomnia is associated with increased sleep latency (time to sleep onset) during daytime testing and inability to fall asleep as quickly as normals. These findings are surprising given the frequent complaints of insomniacs of daytime fatigue to the point of exhaustion.

Underlying Physiological and Psychological Changes

Other studies indicated that insomniacs exhibit increased cortical activation as indicated by increased higher frequency (beta and gamma) wave activity and decreased delta wave activity. Also, the metabolic rate in insomniacs was increased compared to controls. These data that suggested that insomniacs experience a central and peripheral activation were further supported from studies on the activity of the stress system and, particularly, the hypothalamic-pituitary-adrenal (HPA) axis.

by “internalizing” their negative emotions, were in a chronic state of emotional hyperarousal, which led to physiologic activation and sleeplessness. Furthermore, the fear of sleeplessness resulted in a worsened emotional arousal, which led to a vicious cycle of further physiologic activation and more severe insomnia. In that model, stressful life events were important in the initiation of the vicious cycle of emotional and physiologic arousal.

Figure 1

Kales 1984

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Insomnia and Depression vs. Depression Alone

Insomnia, although frequently associated with depression, appears to be different from depression based on polysomnographic, HPA axis, and clinical studies. For example, in insomnia, sleep efficiency measures are the primary variables that are affected whereas in depression, it is both sleep efficiency
disturbance is not the cause but only one of the manifestations of insomnia (Table 1). It appears that central nervous system hyperarousal, either as a pre-existing condition and/or induced by psychiatric pathology and worsened by stressful events and inadequate coping mechanisms, and aging-related physiological decline of sleep mechanisms, is at the core of this common sleep disorder (Figure 2).

**Physiological and Psychological Changes as Vulnerability Factors**

The role of psychological characteristics, including certain predisposing personality traits and inadequate coping mechanisms as vulnerability factors increasing the susceptibility of a subject to insomnia, has long been recognized. It appears that vulnerability to insomnia is also increased by physiological sleep changes associated with aging, such as decreased sleep pressure and increased alertness. Middle-aged subjects are more vulnerable to the sleep-disturbing effects of arousal peptides, such as corticotropin-releasing hormone (CRH) compared to young individuals. This suggests that the marked increase in the frequency of insomnia among middle-aged individuals may be related more to weakened sleep mechanisms, e.g., significant drop of slow-wave-sleep than to increased life stressors. Also, in old, healthy individuals, impaired nighttime sleep is associated with elevated plasma cortisol and interleukin-6 (IL-6) levels, suggesting that increased basal function of the stress system combined with chronic low-grade inflammation process may lead to poor quantity and quality of sleep.

These studies combined suggest that sleep loss/

**Table 1**

<table>
<thead>
<tr>
<th>Insomnia, A Disorder of Arousal</th>
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<tbody>
<tr>
<td>Physiologic activation, i.e., heart rate, core temperature</td>
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<tr>
<td>Increased fatigue but not sleep propensity</td>
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<tr>
<td>Increased beta/gamma activity, decreased delta activity</td>
</tr>
<tr>
<td>Increased secretion of ACTH – cortisol</td>
</tr>
<tr>
<td>Aging related sleep weakness is associated with increased vulnerability to sleep disruption</td>
</tr>
<tr>
<td>Excessive worry, anxiety, depression</td>
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**Insomnia is both a Symptom and a Disorder**

*Defining Short-Term vs. Chronic Insomnia*

**Short-term** insomnia often results from stressful life events or the recent onset of medical disorders. **Chronic** insomnia, by contrast, often becomes a central complaint and a distinct disorder itself.

Acute or short-term insomnia can be associated with a variety of situational problems (work-related, interpersonal, or financial difficulties) or with medical problems, including pain, cardiopulmonary and gastrointestinal disorders, thyrotoxicosis, or the febrile prodromes to influenza. Various drugs, such as caffeine, nicotine, alcohol, steroids, amphetamines, stimulating antidepressants, central adrenergic blockers, and bronchodilators, can impair both falling asleep and staying asleep.

Besides the above possible causes, psychological distress is the most common cause of chronic insomnia. Some degree of psychological distress is present in almost all chronic insomniacs, even in those with “primary insomnia.” Most insomniacs are diagnosed with minor psychiatric disorders, such as dysthymia, anxiety disorders, or subsyndromal states,
e.g. compulsive personality traits. Patients with long-standing sleep difficulties show less than adequate coping mechanisms for stressful life events.

### GUIDELINES FOR TAKING A SLEEP HISTORY IN INSOMNIA

- Define the specific sleep problem
- Assess the onset and clinical course of the condition
- Evaluate 24-h sleep/wakefulness patterns
- Assess stressful events and personality patterns
- Determine the presence of anxiety and/or depression
- Determine the presence of other sleep disorders
- Obtain a family history of sleep and psychiatric disorders
- Evaluate the medical, psychiatric, and personal impact of insomnia

Table 2

### DIAGNOSIS AND MANAGEMENT OF INSOMNIA IN THE PRIMARY CARE SETTING

The family physician’s skills in establishing rapport and meaningful communication with patients greatly enhance his/her ability to evaluate chronic insomnia. To begin with, patients are encouraged to volunteer relevant information about their sleep difficulties (Table 2). Furthermore, the family physician is familiar with the patient’s past and can identify important aspects of his or her life that may pertain to insomnia. In cases of transient (acute) insomnia, this familiarity enables physicians to identify precipitating life-stress events, and in case of chronic insomnia, they will most likely be aware of any premorbid personality factors, any predisposition to chronic sleep difficulty, e.g., “light sleeper,” significant life-stress events, recent psychological conflicts, and current and past medical conditions.

### Identifying Therapeutic Priorities

Because of these advantages, physicians in the office setting are best able to establish and implement therapeutic priorities for the patient. For example, when insomnia is secondary to depression, they can identify suicidal ideation and help to prevent an actual attempt at self-harm. They also will be able to intervene when a life-threatening medical disorder is at the root of insomnia, such as severe coronary artery insufficiency. In such cases as these, **identification and treatment of the medical problem takes precedence over treating the patient’s sleep disturbance**, which will probably be alleviated when the medical problem is treated.

Although sleep apnea and nocturnal myoclonus/periodic limb movements only occasionally cause the primary complaint of insomnia, symptoms of these disorders necessitate a detailed history, including obtaining information from the bed partner. If a patient has a primary complaint of insomnia and is obese, hypertensive, and/or diabetic, and especially a male, the physician should take a detailed history regarding sleep apnea (pauses in breathing during sleep due to obstruction of the airway). The history would assess for excessive daytime sleepiness, snoring, and nocturnal breath cessation. Sleep apnea may cause difficulty maintaining sleep through frequent, brief awakenings, whereas a patient with restless legs/periodic limb movement disorder (unpleasant sensations of periodic jerking movements in the limbs accompanied by an urge to move) may cause difficulty both in initiating and maintaining sleep.

### Efficiently but Adequately Evaluating the Patient with Insomnia Symptoms

An inadequate evaluation often results in superficial treatment of insomnia; the physician may minimize the problem, provide counseling when it is inappropriate, or merely prescribe pharmacologic treatment. For example, a hypnotic drug might be prescribed as the sole pharmacologic treatment for endogenous depression in a suicidal patient. Similarly, counseling would be inappropriate if it were directed toward a particular psychologic symptom when sleeplessness was directly caused by medication.

On the other hand, an overly extensive evaluation can be unnecessarily time consuming and even harmful in some cases of transient insomnia. For example, if a patient has transient sleep disturbance because of a change in work shift, the physician should not focus on the marital relationship or other emotional difficulties that might be present.

### Multifactorial Etiology > Multidimensional Approach

Most sleep specialists advocate a multidimensional approach, including education about the multifactorial etiology of the disorder, with emphasis on the biologic-genetic predisposition, psychological-stress issues, and weakening of sleep mechanisms associated with aging; conveying a sense of hope but tempering unrealistic expectations for “perfect” sleep; general
measures for improving sleep hygiene and lifestyle; supportive, insight-oriented, or cognitive-behavioral psychotherapeutic techniques; and hypnotic or anti-depressant medication. The overall goal of these interventions is to decrease the physiologic and emotional hyperarousal present throughout the 24-hour sleep-wake cycle.

**Sleep Hygiene Integral to Therapeutic Approaches**

Measures for improving sleep hygiene and lifestyle include regularizing the patient’s daily activities schedule, emphasizing that the bedroom should be used for rest and sleep rather than conflict and worry, and improving the sleep environment by minimizing noise and disruptions. Insomniacs should be educated about the adverse effects of unhealthy daytime habits and practices, such as cigarette smoking, caffeine use, and irregular sleep schedules, including work shifts. Also, regular exercise, not close to bedtime, has been shown to increase early-night slow-wave sleep in normal sleepers. In the elderly, special instructions include education regarding age’s effects on sleep patterns; discouraging multiple naps (but, if taken, including nap sleep in the 24-h total for sleep time); and suggesting daytime activities, hobbies, and special interests.

**Behavioral-Cognitive Therapies**

In counseling the insomniac, it is helpful to explain how anxiety participates in the vicious circle that exacerbates and maintains the condition. Patients can be taught to reduce stress and anxiety by managing emotions more effectively through stress management techniques (Table 3). Behavioral-cognitive techniques have become increasingly popular and are reported to be effective, particularly in elderly insomniacs.

**Pharmacologic Therapies**

Benzodiazepine and the newer non-benzodiazepine hypnotics (collectively benzodiazepine receptor agonists, or BzRA) are widely used in the pharmacologic treatment of insomnia, primarily because of their greater margin of safety and degree of effectiveness. The choice of a specific BzRA is based on its side-effect profile and the therapeutic needs of the patient. BzRA hypnotics with a short half-life hasten sleep at bedtime and for a few hours afterward, but are more likely to cause withdrawal difficulties including rebound. In the elderly, smaller doses of hypnotics with an intermediate half-life are more appropriate.

The use of antidepressants as “hypnotics” in

| **GENERAL MEASURES IN TREATING INSOMNIA** |
| **RECOMMENDATION** | **IMPLEMENTATION** |
| **Education** | Discuss the multi-factorial nature of the disorder (biologic-genetic, psychologic, physiologic) |
| | Inform about the chronic nature of the disorder and its resistance to treatment |
| | Convey a sense of hope but temper unrealistic expectations for a “perfect” sleep |
| **Sleep-hygiene measures** | Minimize use of caffeine, cigarettes, stimulants, and other medications |
| | Recognize that alcohol may cause fragmentation of sleep |
| | Maintain a regular sleep schedule and go to bed only when sleepy |
| | Exercise regularly and not close to bedtime |
| | Avoid napping, particularly after 2:00 p.m. |
| **Stress-management measures** | Recognize association between stressful events and sleeplessness |
| | Ventilate conflicts and anger to avoid internalization |
| | Address daily worries a few hours before bedtime |
| | Be tolerant of occasional sleeplessness |
| | Avoid rumination over sleep difficulty |
| | Try relaxation techniques |

Table 3

Insomniacs without a diagnosis of major depression has expanded markedly because insomnia is often a chronic, nonremitting disorder, and benzodiazepines are not recommended for long-term use. The sedative qualities of the old tricyclics, as well as of the newer antidepressants (e.g. trazodone, mirtazapine), administered in relatively low doses at bedtime, appear to improve nighttime sleep by reducing the physiological and mental hyperarousal of the insomniac including a reduction of cortisol levels. In contrast, stimulant antidepressants, such as fluoxetine, may have sleep-disturbing effects when given at bedtime. However, a disadvantage of the sedative antidepressants, particularly the tricyclics, is their strong anticholinergic effects that may adversely affect the subjects by inducing dry mouth, blurred vision, orthostatic hypotension, constipation, and cognitive problems, particularly in the elderly. Neuroleptics with sedative effects are preferred for
psychotic patients who have insomnia. Low doses of atypical neuroleptics may be useful in agitated, confused elderly who present with insomnia. Finally, melatonin does not appear to be beneficial in the large majority of chronic insomniacs.

Based on our thesis that in insomniacs, hyperarousal is present throughout the 24-hour sleep-wake cycle, it is possible that pharmacological strategies that decrease the CNS hyperarousal during the day may be useful. For example, low doses of a benzodiazepine during the daytime may improve sleep in chronic insomniacs. Also, antidepressants that down regulate the HPA axis given during the day in low doses may enhance the therapeutic effect of benzodiazepines or sedative antidepressants administered at night. Finally, in elderly insomniacs, strategies to reduce low-grade inflammation by administration of sex steroids, decreasing fat through diet and exercise, and controlling adequately chronic pain and inflammation with nonsteroidal anti-inflammatory agents, may improve sleep, daytime alertness, and performance, which, in turn, may decrease the risk of common ailments of old age, e.g., metabolic and cardiovascular problems, cognitive disorders, and osteoporosis.

In summary, data from physiologic, EEG, hormonal, and behavioral studies suggest that insomnia and sleep deprivation are two distinct states and that sleep loss is not the cause of insomnia disorder but one of its associated symptoms. Although insomnia is often associated with depression, the pathophysiology of the two disorders is different. A gamut of psychological and physiological factors contribute to the onset and perpetuation of chronic insomnia, such as anxious-ruminative personality traits, stressful events, age-related weakening of sleep mechanisms, and biologic-genetic diathesis for CNS hyperarousal. Most insomniacs can be evaluated adequately and efficiently in the primary care setting where therapeutic priorities can be set. The overall goal of the multidimensional therapeutic approach is to decrease the physiologic and emotional hyperarousal present throughout the 24-hour sleep-wake cycle.
References


