Psychological and Psychiatric Issues in the Etiopathogenesis of Insomnia

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Excessive arousal (both psychological and physiologic) may be the main factor in the etiology of insomnia. Hyperarousal may be the final common pathway in moving the sleep/wake balance toward excessive wakefulness and away from sleep. Psychiatric and psychological factors play a major role in increasing this hyperarousal. Most clinical forms of depression and anxiety disorders are clearly associated with severe insomnia, but stress, learning, and conditioning are also crucial parameters related to insomnia. Depression, anxiety, and stress appear to cause insomnia, not vice versa. The vulnerability of an individual to a specific stress may be a mediating variable, explaining why some persons develop severe insomnia when stressed, while others do not.

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Considerable progress in understanding the causes of insomnia has been achieved throughout the past 20 years. Hyperarousal has been identified as the key concept in the etiology of insomnia, possibly acting as the "final common pathway" (i.e., the funnel through which all other factors could work to cause insomnia). Although many factors may affect hyperarousal, psychiatric and psychological factors are paramount. These concepts are supported by the following details.

HYPERAROUSAL IN INSOMNIA

Whether a person is awake or asleep ultimately depends on the neurologic balance between the arousal circuits (ascending reticular activating system [ARAS]) and the sleep-inducing circuits that interact with the ARAS from about the level of the solitary tract nuclei up to the midbrain.[1] Falling asleep appears to be a 2-step process. First, arousal in the ARAS has to decrease,[2] leading to relaxed wakefulness and possibly stage 1 (alpha and theta) sleep. When arousal is low for a certain amount of time, then the much weaker sleep-inducing circuitry can start to dominate,[3] as indexed by spindles, K complexes, and delta waves. Thus, people with insomnia might either have too much activity in the ARAS or not enough in the sleep-inducing circuits. Clinically, the former appears to be most often the case, but some types of primary insomnia seem to be based on weak sleep-inducing systems (e.g., patients who spend a long time in alpha-dominated drowsiness and in stage 1 sleep but cannot easily get to stage 2).[4]

Arousal in the ARAS is clearly a complex concept that cannot be directly measured, at least in humans. There appear to be many different types of arousal, including psychological, typically conceptualized as psychological agitation, anxiety, intrusive thoughts, ruminations[5]; cortical (e.g., excess beta activity in the electroencephalogram [EEG])[6]; muscular, typically measured by assessing the mean electromyogram (EMG) level on a site such as the forehead[7]; and sympathetic, as evidenced by excess secretion of adrenaline.[5,8] There are many other types of arousal. Unfortunately, these different types of arousal correlate poorly with each other.[9,10] For example, a person might be psychologically quite anxious and aroused but still show low muscle tension and little sympathetic arousal. However, no matter which form of arousal is evaluated, patients with insomnia typically are much more aroused during both wakefulness and sleep than good sleepers.

In a series of interlocking experiments, Bonnet and Arand[11,12] showed that excess arousal may be the key concept in insomnia. Up to now, the key concepts in insomnia were incorrectly assumed to be sleep loss and sleep deprivation. Normal sleepers who are sleep deprived show a physiology and psychology that in many ways directly oppose those of insomnia: body metabolism and body temperature are increased in insomnia but decreased in sleep deprivation; the EEG shows faster waves in insomnia but a predominance of slower waves in sleep deprivation[13]; persons with insomnia are often agitated and anxious, while sleep-deprived patients are usually lethargic. The proper model for an insomniac patient is not a normal person who has been sleep deprived, but a normal person who

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has been given excessive amounts of caffeine on a chronic basis, resulting in an inability to sleep despite severe exhaustion.[14] Indeed, Bonnet even suggests that sleep loss in insomnia may be beneficial—that it is the attempt of the body to slow excessive arousal, as the sleep-deprived individual with insomnia does show a decrease in arousal.[15]

Laboratory studies on the amount of sleep in insomnia have often been puzzling. Although most patients with insomnia do sleep somewhat worse than normal persons sleep (more arousals, longer sleep latencies, shorter total sleep, less stage 3/4 and rapid eye movement [REM] sleep), the differences between normal sleep and sleep of patients with insomnia are often quite small, with much overlap. In the diagnostic category of sleep state misperception (SSM),[16] such patients bitterly complain about their severe insomnia, yet they experience a normal amount of sleep. Surprisingly, patients with SSM are also hyperaroused,[12] and they respond to benzodiazepines or behavioral therapy to the same extent as patients with true insomnia. After treatment, they feel that they sleep much better, even though laboratory studies do not show any obvious improvement in sleep. This reinforces the perception that the amount of objective sleep the insomnia patient actually obtains is probably almost irrelevant compared with the amount of arousal from which that person suffers.[11]

Where does the excess arousal in insomnia originate? Obviously, many etiologic factors may contribute, such as innate hyperactivity in the ARAS, medical conditions causing hyperarousal (e.g., pain, restless legs, drugs), or a mismatch between a patient's circadian arousal and when that person actually wants to sleep, as in advanced or delayed sleep phase syndrome. However, the most frequent causes of hyperarousal are psychiatric and psychological.

PSYCHIATRIC FACTORS IN INSOMNIA

Insomnia surveys invariably show a high association between sleep disturbances and psychopathology. As early as 1962, Weiss et al.[17] found that 72% of psychiatric patients (defined as people who had applied for treatment at a mental health clinic) reported a sleep disturbance compared with only 18% of controls. No specific psychiatric diagnoses were mentioned. In another study by Sweetwood et al.,[18] 51.4% of a "mixed psychiatric sample" (including affective disorders, schizophrenia, alcohol and drug abuse, character problems) reported insomnia at least several times per week, compared with only 16.5% of a nonpatient control sample. Severe problems with insomnia in that population (as opposed to just any problem) were reported by 36% of the patients and 5% of the nonpatient controls. Of interest in that study, the more intense the psychiatric symptoms, the worse the insomnia, although specific diagnosis did not relate very well to the existence of insomnia. More recently, Soldatos[19] reviewed this area and found that very powerful risk factors predict insomnia, such as advancing age, female gender, low socioeconomic status, low levels of education, poor physical health, and psychological/psychiatric problems. All these risk factors (except for female gender and age) are either directly or indirectly related to psychological issues. In almost every study that Soldatos[19] reviewed, psychopathology emerged as the most robust risk factor. For example, Ford and Kamerow[20] found "insomnia related to a mental disorder" to be at least 10 times more frequent than "insomnia related to a physical illness." Soldatos[19] observed that this factor of 10 is likely to be a very low estimate, because the psychometric instrument used in that study could not assess generalized anxiety disorders and personality disorders.

In all studies,[17–20] regardless of the psychometric assessment device used, the estimates of psychopathology in insomnia are almost certainly low. Most people with insomnia are repressors,[21] preferring to have a "medical disease" and rejecting the notion that they may have a "psychiatric" problem. Evaluative questionnaires have difficulty identifying such "repressed" psychopathology, as a considerably skilled interviewer is needed to detect this. Nonetheless, when an assessment is performed, a psychiatric diagnosis can be established in most patients presenting with the complaint of insomnia.[22] The most frequently encountered psychiatric diagnoses associated with insomnia are depression and anxiety disorders.[19]

Numerous studies document the association between depression and insomnia.[23–26] Van Moffaert[27] found that about 90% of patients suffering from unipolar major depression showed diminished sleep efficiency, long sleep latencies, increased awakenings, and early morning awakenings. The polysomnogram typically showed low delta sleep, especially in the first cycle, and a shortening of the first REM latency associated with an increase in total REM sleep early at night. In addition, Ford and Kamerow[20] showed that unresolved insomnia is often a forerunner of relapse in patients with depression, and Hauri et al.[28] found that even those depressed patients who seem to show almost total clinical recovery from depression still sleep more poorly than age-matched normal controls.

The prevalence of anxiety in insomnia had been estimated to be 25% to 42%.[20,23,24] Various anxiety disorders appear to be associated with different types and severity of sleep disturbances.[29] Although patients with social and simple phobias may often show very little sleep disturbance, those with obsessive-compulsive disorder and generalized anxiety disorder usually have increased sleep latency and decreased total sleep time. Bourdet and Goldenberg[30] suggest that the polysomnogram of anxious patients consistently shows increased sleep latency, reduced sleep time, less slow-wave sleep, a greater arousal index, and increased duration of wakefulness during sleep. Effective treatment of the core symptoms of such anxiety disorders will almost always cause a corresponding improvement in the associated sleep disturbance.[29]

PSYCHOLOGICAL FACTORS IN INSOMNIA

Stress is a complex and often misused term. In 1967, Holmes and Rahe[31] felt that stress could be objectively determined and quantified. For example, marriage might have a stress value of 50, while the death of a spouse might have a stress value of 100. Unfortunately, subsequent research has not borne this out. Vulnerability to stress is much more individualized than Holmes and Rahe thought. Partinen[32] found that stress caused an arousal reaction. When the arousal is sufficiently intense, it may lead to insomnia. Soldatos[19] has summarized that insomnia may often be associated with a combination of stressful life events[33] and a certain psychological vulnerability.[34,35] Vgontzas et al.[36] found that activity in the "stress system" (e.g., urinary free cortisol, catecholamine metabolites) was associated with chronic insomnia.

Hard to quantify are the "daily hassles" (stressors of everyday life), such as a poor marriage or poor job satisfaction. These factors are even more often associated with insomnia than the diagnosable psychiatric disorders. Boredom, such as might be encountered after retirement from a very busy executive position, can also severely disturb sleep. Fifteen minutes into an insomnia interview, the patient may discuss how life might be made more meaningful (e.g., after retirement) rather than delving into the details of the symptoms of insomnia.

Learning and conditioning often play an important role long after the initial stressor has disappeared.[21] Assume that a patient is severely distressed by an untoward event such as divorce. After having slept very poorly for 2 weeks or longer, that person cannot help but become concerned about poor sleep because it interferes severely with daytime functioning. It is then quite natural to try very hard to sleep, and the harder one tries, the less one is able to sleep. Similarly, having lain in the same bed many nights with high tension and inability to sleep causes classical conditioning (associating the bedroom stimuli with tension). This person, on entering the bedroom or seeing that nighttime is approaching, will become anxious and tense just as surely as the Pavlovian dogs will salivate in response to the bell. In these learned or conditioned insomnias, there is clearly a diathesis/stress relationship. A person who innately has a strong sleep system will have less of a conditioning effect than one who inherently has some tendency toward poor sleep. The International Classification of Sleep Disorders labels this type of insomnia *psychophysiological*.[16]

THE CAUSE-EFFECT ISSUE

Thus far, only statistical associations between psychiatric/psychological factors and insomnia have been discussed. Which causes which? Clearly, there is a cyclical pattern: being anxious about an event causes poor sleep; but having slept poorly, one is also less able to deal with the daily stressors. Soldatos,[19] however, stated that the relationship between the specific psychopathology of depression or anxiety and insomnia most likely is causal. Similarly, in a recent lengthy review of all available evidence (surveying more than 400 different papers) commissioned by the American Academy of Sleep Medicine, Sateia et al.[37] stated, "it appears unlikely that sleep . . . , per se, is causative of psychological disturbance. Rather, the evidence more strongly suggests that psychological disturbance . . . play[s] a key role in the genesis and maintenance of chronic insomnia."(p250) These conclusions are based on the fact that patients with SSM show psychological findings similar to those with "objective" insomnia and on the findings of Bonnet and Arand[38] that the experimental induction of "objective insomnia" by sleep deprivation in normal sleepers does not cause the psychopathology typically seen in patients with insomnia.

CONCLUSION

In summary, hyperarousal seems to be a key concept in the development and maintenance of insomnia. Although there are many reasons for hyperarousal, psychiatric disorders and psychological distress are the most salient causes of insomnia. However, individual vulnerability to stress and psychological disorders may also be very important, in conjunction with the robustness and balance of that person's neurologic sleep/wake circuitry. No psychological questionnaire or test exists that can detect such vulnerability with enough sensitivity to be considered useful. Therefore, at least for the foreseeable future, a skilled psychological/psychiatric interview is still the best avenue to detect the roots of most insomnias.

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