The Correlation Between Sleep-Disordered Breathing and Psychiatry

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Sleep-disordered breathing is common in patients with mood and anxiety disorders. This article explores the implication for practicing psychiatrists whose patients have sleep disorders.

CASE VIGNETTE
Alex is a 58-year-old married, certified public accountant who snores loudly and has frequent nocturnal awakenings, sometimes with gasping or choking. He generally goes to bed at midnight and wakes up at 6:30 am. On weekends, he usually sleeps 1 or 2 hours longer and sometimes takes a 1-hour nap.
He gained 12 lb in the past year but is not obese (his BMI is 28.5). He reports increasing back pain, fatigue, irritability, and cognitive problems. He occasionally feels anxious and moody, especially when his aches and pains are exacerbated. Six months ago, nocturia developed, but prostate-specific antigen (PSA) test results were normal. At that time, he also began having morning dry mouth, morning headaches, sadness, tiredness, and generally depressed mood. Treatment with an SSRI was initiated; he also had a brief trial with methylphenidate.
He was referred for polysomnography, which revealed severe obstructive sleep apnea with significant oxyhemoglobin desaturations occurring during REM sleep. There were also some central-type SDB events. The patient was treated with bilevel positive airway pressure, with good effect.
This case illustrates a fairly common story. When a middle-aged man becomes less active, works at a sedentary job, gains weight, and begins to snore, chances are that he has or is developing SDB. Job demands and family responsibilities promote sleep schedule restriction, often prompting increased caffeine intake. Nonetheless, sleepiness, tiredness, and fatigue almost invariably follow. Obvious signs include fatigue, attention problems, less effective coping, and depressed mood. A patient may recognize cognitive dulling, as in this case.
Less obvious symptoms include nocturia, morning headache, and difficult to control pain. Our
training dictates that we immediately suspect prostate disease when nocturia develops. However, the negative intrathoracic pressure created by attempting to inhale against an occluded airway provokes release of atrial natriuretic peptide, especially during REM sleep. The resulting nocturia is often periodic, occurring at 1- to 2-hour intervals, with minimal voiding; PSA test results are normal. The negative intrathoracic pressure also creates afterload on the heart and may be associated with increased levels of inflammatory cytokines. Morning headaches can be provoked by hypoxemia during REM-related SDB. REM sleep episodes become progressively longer during the sleep period, and obstructive SDB is usually more severe during REM sleep. In patients who smoke cigarettes or who have a history of smoking, lung function declines more quickly as a function of age. In patients with SDB, severe hypoxemia can result. Sleep loss and pain coexist in a vicious circle: sleep loss lowers pain threshold and pain disrupts sleep. Sedative hypnotics to promote sleep and opioid analgesics reduce respiratory drive, raise arousal threshold, and generally worsen SDB. The net result is greater sleep disruptions and continued pain. **TABLE 1**

**Clinical spectrum of sleep-disordered breathing**

**Pathophysiology**

SDB includes a wide spectrum of disorders that manifest as compromised breathing during sleep (**Table 1**). At one end of the spectrum is primary snoring, ie, snoring without any daytime symptoms or clinically significant reduction in the inspiratory flow. At the other extreme is obstructive sleep apnea, a serious, chronic, and sometimes debilitating condition. SDB also includes central sleep apnea and sleep-related hypoventilation.

**What is already known about sleep-disordered breathing (SDB) and the correlation with psychiatry?**

Psychiatric medical conditions, particularly depression and anxiety, are common in patients with SDB. Multiple mechanisms are proposed to explain this relationship.

**What new information does this article provide?**

Sleepiness and impaired coping mechanisms link SDB, depression, and anxiety disorders. More proactive case findings and SDB treatment in patients with depression and anxiety may help improve management of the psychiatric condition. However, some psychiatric medications can adversely affect sleep and breathing.

**What are the implications for psychiatric practice?**

SDB signs and symptoms overlap with mood, anxiety, and other psychiatric illnesses. In some cases, they may masquerade as these disorders, but SDB can also provoke and/or exacerbate other psychiatric conditions. In addition to obvious symptoms in at-risk populations, SDB should be considered in menopausal women in whom insomnia and fatigue have recently developed and in children who snore and have attention problems. Some screening tools and evaluation techniques in psychiatric
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practices may help identify the SDB at-risk population.

Obstructive types of the sleep-related breathing disorders involve narrowing and flow limitation in the upper airway. During inhalation, this can lead to collapse (apnea), partial collapse (hypopnea), or increased resistance. As airway resistance increases, the effort to breathe and the work of breathing increase. If the effort to breathe increases past some threshold or if the airway collapses, a CNS arousal ultimately occurs, returning ventilation to voluntary control so that the airway can be dilated and breathing can resume. This process is often accompanied by cascading snores, possibly followed by a period of silence (that may persist for several seconds to more than 1 minute), and finally an explosive breath (with possible snorting, gasping, and/or coughing). The patient may quickly fall back to sleep and resume snoring, possibly repeating this sequence hundreds of times during the night. TABLE 2

| Table 2 | lists risk factors, common symptoms, and clinical features of SDB. Many consequences of SDB are also common psychosomatic symptoms. In this case, however, the etiology is well documented and the problem diminishes or disappears when the sleep-disordered breathing is treated.
| Various questionnaires are available to help identify persons at high risk for SDB. One such questionnaire is the STOP BANG questionnaire that can be used in clinical practice. |

Treatment for SDB
Positive airway pressure (PAP) therapy is the first-line treatment for most patients with obstructive sleep apnea. The most common is continuous PAP, which provides a constant airflow that is used to “pneumatically splint” the vulnerable portions of the nasopharyngeal airway by having air blown into the patient’s nose and/or mouth using a nasal or full face mask, nasal pillows, or nasal prongs.

Bilevel PAP provides 2 pressure levels—one during inhalation and a lower one during exhalation. The pressure drop during exhalation increases comfort for patients who have difficulty in exhaling against the incoming airflow. The automatic self-adjusting PAP uses computer-controlled flow variations to determine and supply optimal pressure. Finally, noninvasive positive pressure ventilation devices provide ventilatory assist. These devices are similar to bilevel PAP; however, the rate of oscillation between the two pressures is specified.

Overall, continuous PAP is extremely safe and very effective in patients with SDB, and its benefits are well sustained. Patients who received continuous PAP and conservative therapy (sleep hygiene and weight loss) had decreased sleepiness and improved quality of life compared with patients who received only conservative therapy. Similarly, in a randomized, double-blind, placebo-controlled study, patients who received therapeutic continuous PAP had less sleepiness and better quality of life than patients who received subtherapeutic continuous PAP. However, negative study results have also been reported. TABLE 3

Risk factors, symptoms, and clinical features of sleep-disordered breathing
Comorbid psychiatric illnesses

Studies show a higher prevalence of psychiatric conditions in patients with SDB. Using Department of Veterans Affairs administrative databases, we compared patients with and without SDB. A high prevalence of psychiatric comorbid conditions was seen in more than 100,000 patients with this diagnosis (Table 3). Similar results have been found in non-veteran populations. Mood disorders. Depressive symptoms are frequently reported in patients with obstructive sleep apnea. However, the association between depression and obstructive sleep apnea remains controversial. Findings from our study as well as those from others indicate a higher prevalence of depressive symptoms in patients who have obstructive sleep apnea. In contrast, other studies cast doubt on such a relationship.

Although causality cannot be discerned from these studies, intervention studies provide important clues. Means and colleagues showed that treatment of obstructive sleep apnea with continuous PAP reduced the depressive symptoms. Continuous PAP reduced depression scores in patients with major depression and obstructive sleep apnea who were already receiving optimal antidepressive pharmacotherapy. Furthermore, a systematic review of randomized trials of continuous PAP and obstructive sleep apnea showed improved mood following therapy.

The mechanism(s) underlying the connection between SDB and psychiatric symptoms is a matter of debate. From a psychiatric perspective, one can conceptualize the relationship as stemming from a direct physiological consequence of the general medical condition (in this case, sleep deprivation and nocturnal hypoxia/hypercapnia). Therefore, the increased prevalence of depression with SDB would not be surprising given that such a relationship is seen in other diseases that produce hypoxia and impair quality of life (eg, chronic obstructive pulmonary disease). On the other hand, excessive sleepiness and fatigue as a result of sleep apnea produce significant social and personal problems and result in depression. Ishman and colleagues showed that the daytime sleepiness is a strong predictor of depressive symptoms in patients with SDB.

Sleep apnea and depression can be bridged conceptually by vital exhaustion. Vital exhaustion refers to a state characterized by elevated somatic and cognitive symptoms of depression without affective symptoms. Our group as well as others demonstrated that vital exhaustion profiles were affected in patients with SDB. Therefore, depressive manifestations in patients with sleep apnea may reflect the patients’ vital exhaustion, which differs dramatically from melancholic affective mood changes observed in more typical forms of depression and dysthymia. As such, depressive phenomena in patients who have sleep apnea should be more akin to depression secondary to chronic medical illness.

Anxiety disorders. An association between obstructive sleep apnea and anxiety has also been seen. Our data reveal a higher prevalence of clinically diagnosed anxiety associated with SDB. Yue and colleagues found higher anxiety scores in patients with SDB than in controls (measured by the General Severity Index).

Many years ago, researchers began discussing a link between PTSD and REM sleep behavior disorder (RBD). Under normal circumstances, the hypopolarization of alpha and gamma motor neurons during REM sleep maintains a functional paralysis that prevents the sleeper from enacting his or her dream content. In RBD, the mechanisms that produce REM-related atonia fail and fictive movements become real actions, sometimes resulting in serious injury. Combat veterans with PTSD seem especially vulnerable to REM atonia failure, and dream-enactment reports are more common than in the general population.
More recent findings reveal possible parasomnias involving sleep-related behaviors triggered by SDB—in particular, sleepwalking, night terrors, RBD, and sleep-related eating disorder. Further complicating the matter is that sedative/hypnotics can provoke both SDB events and these behavioral parasomnias. Ongoing studies are attempting to untangle these interwoven factors. Perhaps most intriguing is that SDB events may provoke nightmares, RBD, and sleepwalking episodes. It stands to reason that any choking episode can initially produced intense fear, anxiety, and/or panic. Pagel and Kwiatkowski studied nightmares in 400 patients who were also evaluated polysomnographically for SDB. Patients with severe SDB had fewer recalled nightmares than those with milder disease (30% vs 70%, respectively). This may be because patients with severe SDB have reduced REM sleep, are less likely to fully awaken, or have habituated to the awakenings with choking and gasping. It would be interesting to see whether this relationship reverses in patients with PTSD, given that severe SDB is associated with increased norepinephrine turnover due to sympathetic nervous system up-regulation in response sleep disturbance.

Sleep problems (and especially nightmares) are cardinal symptoms of PTSD. Our data strongly support an association between SDB and PTSD. This association has also been reported by others. Krakow and colleagues posit an arousal-based mechanism initiated by posttraumatic stress–promoting sleep apnea improved insomnia, nightmares, and PTSD symptoms, which may indicate that causality is bi-directional. Sleep loss or disturbance, whether associated with SDB or other conditions, can impair an individual’s coping mechanisms. When sleepy, we are easily frustrated and must make more effort to perform otherwise mindless and rote tasks. In patients who already have difficulty in functioning, especially those prone to impatience, anger, panic, and/or denial, the additional stressor of impaired sleep can exceed the capacity of their defense mechanisms. Thus, SDB can create a special vulnerability to psychological problems—especially anxiety.

**Attention deficit.** Recent studies suggest a relationship between SDB and ADHD in pediatric patients. Findings suggest that many children and adolescents (25% to 50%) with ADHD have sleep problems. In attention and hyperactivity among general pediatric patients have been shown to be associated with increased daytime sleepiness, snoring, and other symptoms of SDB. Yousef and colleagues found a high incidence (20% to 30%) of obstructive sleep apnea in patients with full ADHD syndrome; once obstructive sleep apnea was treated, improvements in behavior, inattention, and overall ADHD were seen. Naseem and colleagues reported on 3 patients with adult hyperactivity; all 3 patients suffered from symptoms of obstructive sleep apnea. With continuous PAP, 2 of the 3 patients showed improvement in their sleep and hyperactivity symptoms. The vast majority of patients with SDB report sleepiness. If the sleepiness level exceeds the individual’s compensatory alertness mechanisms, he may resort to additional strategies to maintain focus (eg, caffeine ingestion). Arousal and attention are fundamental to most cognitive tasks, particularly those that require quick or well-timed responses. However, beyond simple attentiveness, diminished executive task abilities (sometimes considered frontal lobe functions) were correlated with sleepiness and, thus by association, with SDB.

**Psychiatric medications**

SDB is adversely affected by most sedating medications. Exacerbation of SDB is not restricted to barbiturates, benzodiazepines, and other sleeping pills. Any sedating drug—antidepressant, antipsychotic, analgesic, or antihistamine—can adversely affect breathing during sleep. While these compounds reduce respiratory drive (eg, opioid analgesics), more importantly, they can raise arousal threshold (eg, sedative/hypnotics). If an airway obstruction occurs, ventilation is resumed in response to CNS arousal that allows voluntary dilation of the airway. The more sedated the individual, the more difficult it is to awaken him and the longer it takes to open the airway. Some substances also exacerbate preexisting SDB by compromising muscles responsible for maintaining upper airway patency (eg, benzodiazepines) or allowing the base of the tongue to occlude the airway (eg, alcohol). Insomnia is a common symptom in many psychiatric disorders and may act synergistically in that sleep loss worsens the overall condition. As such, treatment of insomnia can be critical to managing the psychiatric condition. If the insomnia worsens or if the patient continues having nonrefreshing sleep after a sedative/hypnotic is administered and the duration of sleep is adequate, consider possible SDB. This is especially important if the patient snores, is obese, reports fatigue, or has hypertension. If suspicion is high, a polysomnographic sleep evaluation is indicated.

**Summary**
Psychiatric comorbid conditions, especially mood disorders and anxiety disorders, are common in patients with SDB. A thorough sleep evaluation is warranted in patients with psychiatric conditions that have SDB risk factors. Screening questionnaires, such as STOP BANG, may help identify persons at risk for SDB. These include middle-aged (or older) postmenopausal women in whom insomnia or snoring has developed, patients who are obese, and patients whose sleep problems worsen when they are taking sedative/hypnotics. An SDB diagnosis, appropriate therapy, and follow-up in these patients may improve management of their comorbid psychiatric conditions.

References:
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