

Obstructive Sleep Apnea in Neurological Patients

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Abstract: Obstructive sleep apnea (OSA) is a serious condition that is common among neurological patients. If undiagnosed and untreated, it may lead to hypertension, coronary heart disease, pulmonary hypertension, myocardial infarction, stroke, psychiatric disorders, cognitive impairment, and, ultimately, death. One of the first steps in identifying OSA is to recognize its signs and symptoms. Nursing knowledge of OSA facilitates referral, diagnosis, and treatment of this potentially life-threatening condition.

Obstructive sleep apnea (OSA) is a common, potentially life-threatening condition. An estimated 80%–90% of Americans with OSA have not been diagnosed (Kapur, Strohl, & Redline, 2002). If untreated, OSA may lead to hypertension, coronary heart disease, pulmonary hypertension, myocardial infarction, stroke, psychiatric disorders, cognitive impairment, and, ultimately, death. OSA may present at any time across the lifespan, from early childhood to older age. The incidence of OSA in patients with certain neurological conditions such as Parkinsonism, myotonic dystrophy, and myasthenia gravis is higher than in the general population, primarily due to impairment of the nerves controlling the muscles of the upper airway. Neurological disorders and OSA can coexist and, potentially, exacerbate each other (Vaughn & D’Cruz, 2003).

One of the first steps in identifying individuals with OSA is educating nurses about the symptoms of and risk factors for this disorder so they can identify and refer appropriate patients for evaluation. Nurses working with patients who have neurological disorders must receive OSA-related education. Certain clinical features of OSA and neurological disorders are the same, making it more challenging to identify sleep problems. This article presents an overview of OSA and related health conditions, with an emphasis on neurological conditions, risk factors and symptoms, and assessment to guide nurses in identifying patients whose sleep patterns require further evaluation.

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Overview

OSA is characterized by repeated episodes of apnea (the cessation of breathing for at least 10 seconds) and/or hypopnea (an airflow reduction of at least 30%), accompanied by a 4% drop in blood oxygen saturation level during sleep due to obstruction of the pharyngeal airway despite persistent respiratory efforts (Kryger, Roth, & Dement, 2000). To describe the severity of this disorder, sleep literature uses the terms *apnea index* (AI), which is the number of apneas per hour, and *hypopnea index* (HI), which is the number of hypopnea episodes per hour. In addition, the terms *apnea-hypopnea index* (AHI) and *respiratory disturbance index* (RDI) are used interchangeably to reflect the sum of apneas and hypopneas per hour.

Pathology and Implications

The primary pathophysiologic event that occurs in instances of OSA is occlusion or near occlusion of the pharynx during sleep. When patients are awake, pharyngeal muscles are sufficiently activated to maintain patency of the upper airway. While they are asleep, however, there is decreased activation of this musculature. In OSA patients, the normal narrowing of the upper airway progresses to complete collapse during inspiration, or possibly at end-expiration. When patients exert increased effort to inspire against the occluded airway, it makes the situation worse by creating more negative airway pressure. Occlusion continues until arousal occurs and the resulting increased tone of the pharyngeal muscles reopens the airway. These “micro-arousals” may occur at rates of more than 90 per hour—whenever an apneic or hypopneic event disrupts sleep—with patients usually unaware they have occurred. When these apneic events are lengthy (some last for 2 minutes or longer), they can result in significant oxygen desaturation and decrease sleep levels necessary for physical and psychological restoration. Current criteria identify mild OSA as an AHI of 5–15, moderate OSA as an AHI of 15–30, and severe OSA as an AHI higher than 30 events per hour (American Academy of Sleep Medicine Task Force, 1999).

Incidence in Neurological Conditions

Any neurological condition that results in impaired tone of the upper airway muscles can add to airway obstruction and result in OSA, especially when the individual has additional risk factors, such as obesity or hypothyroidism. Obstruction can occur at the pharyngeal level because of primary bulbar weakness or the inability

Sources for Information About Sleep Apnea

National Sleep Foundation
729 15th Street NW, 4th floor
Washington, DC 20005
www.sleepfoundation.org

National Heart, Lung, and Blood Institute
P.O. Box 30105
Bethesda, MD 20824-0105
<http://www.nhlbi.nih.gov/health/public/sleep/index.htm>

American Sleep Apnea Association
www.sleepapnea.org/geninfo.html

The American Sleep Disorders Association
Web address: <http://www.asda.org>

Stanford University—Sleep Apnea Information and Resources
www.Stanford.edu/~dement/apnea.html

The Yale Center for Sleep Disorders
www.info.med.yale.edu/intmed/sleep

of the diaphragm and intercostal muscles to overcome changes in airway resistance. This problem is magnified during rapid eye movement (REM) sleep due to the natural loss of intercostal muscle tone during that period.

Common neurological and neuromuscular disorders associated with a higher incidence of OSA in adults include congenital myopathies, neuropathies, myotonic dystrophy, Duchenne's dystrophy, mitochondrial encephalomyopathy, myasthenia gravis, stroke, epilepsy, Parkinsonism, and Alzheimer's disease (Kryger et al., 2000). Neuromuscular disorders that may be associated with OSA in children include, but are not limited to Duchenne's dystrophy, myotonic dystrophy, nemaline myopathy, congenital muscular dystrophy, cerebral palsy, spinal muscular atrophy, transverse myelitis, and poliomyelitis (Seddon & Khan, 2003).

Sleep disturbance in patients with epilepsy frequently is overlooked, but may contribute to decreased daytime functioning and increased seizure activity (Bazil, 2000). Malow et al. (2003) studied adults and children with epilepsy, and found that 50% of those with both epilepsy and some risk factors for OSA who had a polysomnogram (sleep study) did have OSA. In addition, when treated with continuous positive airway pressure (CPAP) therapy, their seizure frequency was reduced by 45%. It is especially important to identify OSA in patients with epilepsy because most antiepileptic drugs' sedating effects can worsen OSA. (Manni & Tartara, 2000). Manni et al. (2003) found the coexistence of OSA with epilepsy to be 10.2% among the epilepsy patients they studied (15.4% of the men and 5.4% of the women). They reported that older age at onset of seizures was related significantly to comorbidity with OSA. Malow, Levy, Maturen,

and Bowes (2000) found that one-third of the studied epilepsy surgery candidates with histories of medically refractory epilepsy had OSA diagnosed following polysomnography. Previously undiagnosed OSA was common in these patients, especially among the men, older subjects, and those with seizures during sleep.

OSA, which frequently is identified in stroke patients, may be a factor in stroke development secondary to hypertension that results from nightly episodes of hypoxia and chronically elevated sympathetic tone, or a stroke that occurs due to impaired upper airway musculature control (Lattimore, Celermajer, & Wilcox, 2003; Yaggi & Mohsenin, 2004). Bassetti and Aldrich (1999) found that 62% of the stroke and transient ischemic attack patients they studied had an AHI index higher than 10. Wessendorf, Teschler, Wang, and Schreiber (2000) reported the prevalence of sleep-disordered breathing (primarily OSA) was 61% in first-time stroke patients. Good, Henkle, Gelber, Welsh, and Verhulst (1996) studied sleep-disordered breathing and functional outcomes following stroke. They found that 68% of studied stroke patients had an AHI higher than 20, and 53% had an AHI higher than 30, with desaturation events primarily due to obstructive apneas. They also found that sleep-disordered breathing, especially OSA, was an independent predictor of worse functional outcome during the rehabilitation period, and was associated with a higher mortality rate at 1 year.

Up to 60% of adult patients with myasthenia gravis have an above-average number of apneas and hypopnea periods during sleep. Duration of illness has been correlated with the severity of apneas in these patients (Amino et al., 1998).

Maria et al. (2003) found that 60% of patients with idiopathic Parkinson's disease (PD) who participated in their study met the criteria for OSA. In addition, they found that study participants with more severe PD had more severe OSA.

Risk Factors

Most patients diagnosed with OSA are obese, with more than 60% weighing at least 20% above the recommended body weight for their height. This relationship may be caused by decreased airway size resulting from fat deposits in the neck area.

Gender also appears to influence the incidence of OSA. The incidence in men is higher than in women in all age groups; after menopause, however, the incidence in women approaches that of men. It is thought that estrogen exerts a respiratory stimulant effect that may provide some protection to premenopausal women (Keef, Watson, & Naftolin, 1999). Men also tend to have shorter, thicker necks, which may result in decreased upper airway size.

Race also may be an OSA risk factor. In African Americans and Asians, the syndrome occurs twice as often as

in Caucasians, and tends to be more severe in the former groups (Ong & Clerk, 1998). This may be explained by differences in skull and upper airway shapes in certain ethnic populations.

Increasing age also is a risk factor for OSA (Shochat & Pillar, 2003). Most patients with OSA are 40 years of age or older, with the incidence increasing slightly with age. The weight gain that frequently occurs with aging may have a bearing on this finding. The increased incidence of OSA also may be due to an age-related decrease in muscle tone in the upper airways, allowing tissue in the pharyngeal area to collapse more readily.

OSA also is associated with acromegaly and hypothyroidism (Kryger et al., 2000). Narrowing of the upper airway due to abnormalities of ventilatory effort or mucoprotein deposition on the tongue and in the nasopharynx are two possible mechanisms for OSA in these patients.

Any myopathy, neuropathy, or neurological condition that causes weakness of the upper airway musculature predisposes an individual to OSA development.

In children, enlarged tonsils and adenoids are a significant OSA risk factor, primarily during early childhood (Nieminen, Tolonen, & Lopponen, 2000). Adenoid enlargement (hyperplasia) is normal in childhood, and it is not clear why some children develop hyperplasia severe enough to cause obstruction, while others do not. Neither adenoid thickness nor airway size, as determined by X ray, is a strong predictor of obstruction. Hyperplasia most commonly occurs at about the age of age 5, but symptoms have been reported in infants as young as 2 months.

Any myopathy, neuropathy, or neurological condition that causes weakness of the upper airway musculature predisposes an individual to OSA development. Neurological impairment, primarily impairment of the genioglossus muscle, is considered the main factor that causes this predisposition.

Other medical conditions, such as nasal obstruction secondary to rhinitis, sinusitis, allergies, tumors, or cysts, place individuals at higher risk of obstruction. Rheumatoid arthritis that involves the temporomandibular joint also may predispose individuals to develop OSA.

Tobacco use is also known to have a detrimental effect on sleep. Smokers have a fourfold to fivefold greater risk than those who never smoked of having at least moderate sleep-disordered breathing (Wetter, Young, Bidwell, Badr, & Palta, 1994). It is thought that cigarette smoking elicits mucosal edema, thereby contributing to a narrowed airway.

Symptoms

Individuals with OSA may present with a variety of symptoms with which nurses should be familiar (Fig 1); some of these symptoms appear during both waking hours and the usual sleep period. Excessive daytime sleepiness is one of the most frequently reported OSA symptoms. Naps taken during the day are not refreshing, and the person awakens feeling even more groggy. Those with OSA frequently are involved in motor vehicle collisions, as it is often difficult for them to stay awake while driving. Individuals with OSA often fall asleep while watching television, reading, or participating in other sedentary activities. Daytime sleepiness can have life-threatening consequences for teens and adults who drive during the day.

Children past the age of 8 or 9 years typically present with many symptoms similar to those of adults. It is important to note that younger children may present with hyperactivity and/or aggressive behavior rather than excessive sleepiness, because younger children often fight sleepiness with agitation. These symptoms may result in an incorrect diagnosis of attention deficit hyperactivity disorder (ADHD), when, in fact, the correct diagnosis is OSA, most often caused by hypertrophy of the adenoids and/or tonsils.

Characteristic loud pharyngeal snoring is reported in the majority of patients diagnosed with OSA. Typical loud snoring occurs first, followed by periods of decreased sound and apnea. These periods of apnea often last from 20 seconds to 2 or more minutes, usually ending with a loud snort. This cycle typically repeats itself many times throughout the night, on an intermittent basis, with patients usually unaware of their abnormal respiratory patterns and snoring. Consequently, a spouse or other family member often first identifies the problem by observing these abnormal breathing patterns. It is not unusual for patients who are evaluated at sleep disorder centers to experience 200–600 apnea events throughout the night.

Periodic leg jerks, other abnormal sleep movements, and general restlessness often are observed as individuals reposition themselves to facilitate respiratory efforts. The exact mechanism by which these leg jerks develop and are related to OSA is not clearly understood.

Cognitive changes, such as periods of confusion, the inability to concentrate, or short-term memory impairment, also occur frequently with OSA. Morning headaches in the frontal area that resolve about 2 hours after awakening also may occur. These symptoms are thought to occur as the result of high levels of carbon dioxide retention after apneas and/or hypopnea episodes that result in cerebral vasodilation. It is not uncommon for oxygen saturation levels to drop 15% or more immediately following apneic events, and these desaturations may contribute to decreased mental alertness and shortened attention span throughout the waking hours.

Symptoms During Waking Hours

Excessive daytime sleepiness
Generalized fatigue
Declining overall performance
Deterioration of memory and judgment
Personality changes—irritability, aggressiveness, depression, and inattentiveness
Hyperactivity (children)
Decreased sexual drive/impotence
Morning headaches
Hypertension/sinus arrhythmias

Symptoms During Sleeping

Loud snoring, with explosive gasps followed by periods of silence and apnea
Abnormal activity during sleep—restlessness/sudden leg movements
Nocturnal enuresis (children)/urinary frequency (adults)
Gastroesophageal reflux
Drooling

Source: Bassiri, A., & Guilleminault, C. (2000). Clinical features and evaluation of obstructive sleep apnea-hypopnea syndrome. In M. H. Kryger, T. Roth, & W. Dement (Eds.), Principles and practice of sleep medicine (3rd ed., pp. 869–893). Philadelphia: Saunders.

Fig 1. List of common awake and sleeping symptoms

Personality changes, including anxiety, depression, irritability, and decreased sexual drive or impotence, also may occur as a result of severely disrupted sleep patterns. Work and school performance, as well as personal relationships often suffer because of the personality changes induced by sleep fragmentation and lack of REM sleep.

OSA causes cardiac stress, which increases the secretion of atrial natriuretic peptide, which in turn stimulates the kidneys, and increases urine production and abdominal pressure. These actions form the basis for nocturia in adult OSA patients and nocturnal enuresis in children with OSA. It is not unusual for adults with OSA to awaken and urinate several times during the night (Umlauf et al., 2004)

Gastroesophageal reflux is another frequently observed symptom among OSA patients. Reflux occurs secondary to decreased airway pressures during periods of upper airway obstruction and a subsequent increase in breathing effort and abdominal pressure.

Continual sympathetic stimulation during the night often leads to cardiovascular symptoms that persist during waking hours. These symptoms include elevations of blood pressure, sinus arrhythmias, and nocturnal angina (Foresman, Gwartz, & McMahon, 2000). It is believed that these symptoms, which occur in response to decreased oxygen saturation levels, result in cardiac hypoxia. On average, systolic and diastolic blood pressure increase approximately 25% with apnea events, with the greatest elevations occurring during REM sleep when desaturation is most severe. The exact mechanism by which

these increased values persist into the waking hours is not understood. Marcus, Greene, and Carroll (1998) found that children with OSA had a significantly higher diastolic, but not systolic, pressure during both sleeping and waking hours, with pressure slightly lower during sleep. Adults with OSA often have elevated systolic and diastolic measurements.

Nursing Interventions

Nurses can help OSA patients prevent life-threatening physiologic changes and improve the quality of their lives and the lives of those around them. Nursing intervention begins with assessment, and continues with referral and educational and psychological support.

Assess symptoms and risk factors. The first important nursing intervention occurs during the nurse's initial contact with the patient, when the nurse identifies risk factors and assesses for symptoms. The patient should be asked about excessive daytime sleepiness, headaches, and difficulty with work, school, or social relationships. A history of alcohol intake, smoking, and nighttime symptoms, such as urinary frequency, should be obtained. Also ask family members or caregivers if they have witnessed the patient exhibiting apneas during sleep, snoring, or changes in mood or personality. A list of appropriate questions to elicit this information is presented in Fig 2. Also ask the patient to maintain a sleep diary for about a week in which he or she documents total hours of sleep, exact bedtime, bedtime rituals, frequency of awakenings, number of naps, and feelings of overall sleepiness during the day. A physical exam may reveal additional information suggestive of OSA, and should include measurements of body weight, height, pulse, and blood pressure. Inspect the oral cavity, looking for enlarged tonsils, adenoids, or tongue, and for the presence of a large, droopy, soft palate. Look at the nasal passages carefully for patency. Careful cardiac auscultation should be performed with attention to the presence of arrhythmias or signs of pulmonary hypertension, such as an increased pulmonic component to the second heart sound. To get a better idea of the patient's breathing behavior, it is helpful to ask a family member or caregiver to mimic the way the patient breathes.

Refer for medical evaluation. Prompt referral to a physician certified in sleep medicine is important. The definitive test for OSA is an overnight polysomnogram performed in a certified sleep laboratory. Following the study, results are interpreted, a diagnosis is made, and treatment prescribed, depending upon the findings. Weight loss is encouraged for obese or overweight patients. In children, removing tonsils and adenoids often results in obstruction relief. In adults, treatment most often includes a prescription for CPAP. In mild cases or for patients who are not able to tolerate CPAP,

Ask the patient the following questions:

1. Do people say that you snore? If so, how often and how loudly?
2. Has anyone ever told you that you are restless or that your legs jerk or twitch when sleeping?
3. Do you often wake up feeling tired or with a headache? If so, how often?
4. Do you find yourself sleepy during the day? Have you ever fallen asleep at work or school, or while driving a car?
5. Do you feel drowsy or confused upon awakening?
6. Are you currently having difficulty at work, with school, or with social relationships?
7. Do you have any allergies or airway obstructions?
8. Do you drink alcohol? In so, when and how much?
9. Do you smoke? If so, how much?
10. Does anyone in your family have sleep apnea?
11. Do you have hypothyroidism or any neurological disorders?
12. Do you have a history of chronic conditions, such as high blood pressure, cardiovascular or respiratory disease, or endocrine or neurologic disorders?
13. Do you get up at night to urinate? If so, how often?
14. Do you experience symptoms of gastric reflux that awakens you from sleep?
15. Do you have decreased sexual interest or impotence?
16. Are you menopausal?
17. Do you regularly take any prescriptions or over-the-counter medications, such as sleeping aids, sedatives, or decongestants?

Ask family members or caregivers the following questions:

1. Have you seen the patient have any apneas (pauses in breathing) during sleep?
2. Does the patient snore? If so, please describe how the snoring sounds.
3. Is the patient excessively restless during sleep, or sleep in any unusual positions?
4. Have you noticed any recent mood or personality changes in the patient?
5. Does the patient ever wet the bed?

Fig 2. *Sleep apnea assessment questions*

an oral mandibular advancement device might be prescribed (Yantis, 2003). This device, usually fitted by a dentist or oral surgeon, advances the mandible slightly, bringing the tongue forward and slightly increasing the diameter of the airway space. The long-term effects of the various upper airway tissue reduction procedures such as somnoplasty and laser-assisted uvulopalatopharyngoplasty have yet to be proven beneficial.

Following diagnosis, it is important to provide psychological support, because OSA can be a frightening diagnosis. Patients may have difficulty adjusting to treatments, such as CPAP devices. Promote airway patency by advising patients to avoid the ingestion of alcohol (especially before bedtime) and sedating medications. Weight loss is an important factor in reducing the amount of excess tissue around the upper airway. Encourage patients to avoid using nasal sprays, which may cause rebound nasal congestion. Also advise patients to wear a medic-alert bracelet or necklace that can alert medical personnel to their OSA.

Application to Neuroscience Nursing Practice

It is incumbent upon neuroscience nurses to assess for OSA and refer when appropriate (Bader & Littlejohns, 2004). Patients with untreated OSA have decreased oxygen saturation levels, which may worsen neurological or neuromuscular symptoms. In turn, these symptoms can exacerbate OSA because of decreases in muscle strength, respiratory effort, or cognitive function. Neuroscience

patients may develop a circular pattern of disease/disorder and OSA advancement if their disorder goes undiagnosed and untreated.

Assessing neuroscience patients for OSA may be problematic because of their neuroscience disorder(s). Nevertheless, assessment is necessary if symptoms are noted. Stroke patients or patients with neuromuscular disorders that affect speech may not be able to articulate the answers to questions clearly or appropriately. In these instances, ask family members to describe behaviors such as snoring, any worsening of the underlying condition, or increases in seizure activity. In some patients, swallowing difficulties may be more pronounced; assess for choking potential. Also assess for decreases in neurological status after rest or sleep periods accompanied by snoring. For patients in rehabilitation programs, watch for sleep occurrences that may indicate OSA during therapies or while they are eating. Again, assess patients for abnormal movements during sleep. It is easiest for neuroscience nurses to monitor inpatients while they are sleeping. If the patient is at home, instruct family members to report OSA signs or symptoms to the healthcare provider for evaluation. Finally, if the patient takes blood pressure medications and the dosages or types of medications change, evaluate sleeping patterns for possible OSA.

Summary

Most of us equate sleep with rest, but sleep is a potential menace for millions of people. In patients with

neurological disorders, the incidence of OSA is increased, making careful nursing assessment imperative. By identifying and promptly referring these patients for evaluation, nurses become an important part of their improved health and quality of life.

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