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DESCRIPTION

Sleep apnea is usually a chronic (ongoing) condition that disrupts sleep three or more nights each week. One often moves out of deep sleep and into light sleep when one's breathing pauses or becomes shallow resulting in poor sleep quality and consequently fatigue during the day. In fact, sleep apnea is one of the leading causes of excessive daytime sleepiness. The Greek word “apnea” literally means “without breath.” There are three types of apnea: obstructive, central, and mixed; of the three, obstructive is the most common. Despite the difference in the root cause of each type, in all three, people with untreated sleep apnea pause in breathing or take shallow breaths, sometimes hundreds of times during the night, which can last for a few seconds to minutes often occurring 5–30 times or more an hour. Typically normal breathing resumes with a loud snort or choking sound as air tries to squeeze past the blockage (American Sleep Apnea Association, 2008).

NEUROPATHOLOGY/PATHOPHYSIOLOGY

The underlying physiology of sleep apnea varies based on the type of sleep apnea presented. Obstructive sleep apnea occurs when the muscles in the back of the throat relax. These muscles support the soft palate, the triangular piece of tissue hanging from the soft palate (uvula), the tonsils, and the tongue. When the muscles relax, the airway narrows or closes when taking a breath, and breathing momentarily stops. This may lower the level of oxygen in the blood. The brain senses this inability to breathe and briefly rouses the patient from sleep so that the patient can reopen the airway. This awakening is usually so brief that most patients don't remember it. Obstructive sleep apnea is caused by repetitive upper airway obstruction during sleep as a result of narrowing of the respiratory passages. Patients with the disorder are most often overweight, with associated peripharyngeal infiltration of fat and/or increased size of the soft palate and tongue. Some patients have airway obstruction because of a diminutive or receding jaw that results in insufficient room for the tongue. These anatomic abnormalities decrease the cross-sectional area of the upper airway. Decreased airway muscle tone during sleep in combination with the pull of gravity in the supine position further decreases airway size, impeding airflow during respiration. Initially, partial obstruction may occur and lead to snoring. As tissues collapse further or the patient rolls over on his or her back, the airway may become completely obstructed. Whether the obstruction is incomplete (hypopnea) or total (apnea), the patient struggles to breathe and is aroused from sleep. Often, arousals are only partial and are unrecognized by the patient, even if they occur hundreds of times a night. The obstructive episodes are often associated with a reduction in oxyhemoglobin saturation.

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A sleep disorder that is characterized by a person experiencing difficulties in sleeping due to interruptions in breathing. Following...

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With each arousal event, the muscle tone of the tongue and airway tissues increases. This increase in tone alleviates the obstruction and terminates the apneic episode. Soon after the patient falls back to sleep, the tongue and soft tissues again relax, with consequent complete or partial obstruction and loud snoring (Patil, Schneider, Schwartz, & Smith, 2007).

The combinations of the physiological characteristics causing obstructive sleep apnea may vary considerably among patients. Most obstructive apnea patients have an anatomically small upper airway with augmented pharyngeal dilator muscle activation maintaining airway patency awake, but not asleep. However, individual variability in several phenotypic characteristics may ultimately determine who develops apnea and how severe the apnea will be. These include: (1) upper airway anatomy, (2) the ability of upper airway dilator muscles to respond to rising intrapharyngeal negative pressure and increasing CO_2 during sleep, (3) arousal threshold in response to respiratory stimulation, and (4) loop gain (ventilatory control instability). As a result, patients may respond to different therapeutic approaches based on the predominant abnormality leading to the sleep-disordered breathing.

Occasionally, obstructive sleep apnea can be caused by less common medical problems, including hypothyroidism, acromegaly, and renal failure. Neuromuscular disorders such as postpolio syndrome can result in inadequate neuromuscular control of the upper airway and lead to obstructive sleep apnea. Restrictive lung disease from scoliosis has also been associated with the disorder.

Central sleep apnea, which is far less common, occurs when the brain fails to transmit signals to the breathing muscles. The patient may awaken with shortness of breath or have a difficult time getting or staying asleep. Like obstructive sleep apnea, snoring and daytime sleepiness can occur. Central sleep apnea, in its various forms, is generally the product of an unstable ventilatory control system (high loop gain) with increased controller gain (high hypercapnic responsiveness) generally being the cause. High plant gain can contribute under certain circumstances (hypercapnic patients). The most common cause of central sleep apnea is heart disease, and less commonly, stroke. People with central sleep apnea may be more likely to remember awakening than people with obstructive sleep apnea (White, 2005).

Mixed (complex) apnea, as the name implies, is a combination of the two. With each apnea event, the brain briefly arouses people with sleep apnea in order for them to resume breathing, but consequently sleep is extremely fragmented and of poor quality. People with complex sleep apnea have upper airway obstruction just like those with obstructive sleep apnea, but they also have a problem with the rhythm of breathing and occasional lapses of breathing effort (Mayo Clinic, 1998).

In general, sleep apnea may occur whether the patient is young or old, male or female. Even children can have sleep apnea. But certain factors put individuals at increased risk. In the case of obstructive sleep apnea, risk factors include: excess weight; neck circumference; high blood pressure (hypertension); a narrowed airway; being male; being older (sleep apnea occurs two to three times more often in adults older than 65); family history; use of alcohol, sedatives or tranquilizers; and smoking. In the case of central sleep apnea, risk factors include: being male, heart disorders, and stroke or brain tumor (these conditions can impair the brain's ability to regulate breathing).The same risk factors for obstructive sleep apnea are also risk factors for complex sleep apnea. In addition, complex sleep apnea may be more common in people who have heart disorders (Mayo Clinic, 1998).

NEUROPSYCHOLOGICAL/CLINICAL PRESENTATION

The most common complaints associated with sleep apnea involve loud snoring, disrupted sleep, and excessive daytime sleepiness. Patients with apnea suffer from fragmented sleep and may develop cardiovascular abnormalities because of the repetitive cycles of snoring, airway collapse, and arousal. The snoring and apneic episodes may be worse after the patient drinks alcohol or takes sleeping pills because these sedatives decrease pharyngeal muscle tone and can exacerbate obstructive sleep apnea. Although most patients are overweight and have a short, thick neck, some are of normal weight but have a small, receding jaw. Because many patients are not aware of their heavy snoring and nocturnal arousals, obstructive sleep apnea may remain undiagnosed; therefore, it is helpful to question the bedroom partner of a patient displaying symptoms such as chronic sleepiness and fatigue.

People with sleep apnea may also complain of memory problems, morning headaches, mood swings or feelings of depression, a need to urinate frequently at night (nocturia), and impotence. Gastroesophageal reflux disease (GERD) may be more prevalent in people with sleep apnea. Children with untreated sleep apnea may be hyperactive and may be diagnosed with attention deficit hyperactivity disorder (ADHD).

Daytime fatigue and sleepiness are the most significant complaints of the patient with obstructive sleep apnea with symptoms ranging along a continuum, for example, the patient falls asleep during sedentary activities, such as watching television or sitting in a movie theater. This can progress to falling asleep in embarrassing situations, such as during meals or when sitting in a car stopped at a traffic light. The patient often has to nap during the day, but typically wakes up unrefreshed.

Patients with obstructive sleep apnea show neuropsychological impairments ranging from vigilance decrements, attentional lapses, and memory gaps to decreased motor coordination. In terms of neurobehavioral performance, studies have revealed objective daytime somnolence but little impairment in memory and motor domains (Kelly, Claypoole, & Coppel, 1990). Cerebral data have shown gray matter loss in the frontal and temporo-parieto-occipital cortices, the thalamus, hippocampal region, some basal ganglia and cerebellar regions, mainly in the right hemisphere. A decrease in brain metabolism is generally right-lateralized, but more restricted than gray matter density changes, and involves the precuneus, the middle and posterior cingulate gyrus, and the parieto–occipital cortex, as well as the prefrontal cortex. Even in cases of patients displaying only minor memory and motor impairments, there are still significant cerebral changes in terms of both gray matter density and

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metabolic levels, indicating that these patients may benefit from cognitive reserve and compensatory mechanisms. Therefore, it is possible that cerebral changes in obstructive sleep apnea patients may precede the onset of notable neuropsychological consequences (Khalid, 2009).

Neuropsychological evaluations can address the general deficits associated with sleep apnea such as the Beck Depression Inventory to assess depression, the Hopkins Verbal Learning Task to assess memory, and the Stroop Test to assess attention, mental speed, and mental control. Alternatively, a battery of tests such as the Halstead–Reitan or Luria–Nebraska Neuropsychological Battery, assessing a broad range of functional domains may be administered. These evaluations could be repeated after a treatment protocol has been instituted in order to assess pre- and postfunctioning. The neuropsychological evaluation may also be tailored according to the specific needs of the patient. For example, if the effects of a treatment were being assessed in terms of a patient's driving ability, specific tests would be used to measure cognitive abilities accessed while driving a car. (1) Vigilance: subjects have to watch a beam on a screen moving up and down. They have to react on rarely occurring higher swings by pressing a button. (2) Alertness: simple reaction on a lamp flashing upwards. (3) Divided attention: patients have to press colored lamps when the corresponding color flashes on a board. In addition, they have to react to acoustic signs by pressing buttons and foot pedals when indicated by signals (Orth et al., 2005).

DIAGNOSIS

A physician may make an evaluation based on the symptoms or may refer the patient to a sleep disorder center to undergo further evaluation that often involves overnight monitoring of breathing and other body functions during sleep. Tests to detect sleep apnea may include nocturnal polysomnograph, where the patient is hooked up to equipment that monitors heart, lung, and brain activity, breathing patterns, arm and leg movements, and blood oxygen levels asleep.

Oximetry is another screening method that involves using a small machine that monitors and records the oxygen level in the blood while the patient is asleep. If the results are abnormal, the doctor may prescribe polysomnography to confirm the diagnosis. Oximetry doesn't detect all cases of sleep apnea, so the doctor may still recommend a polysomnogram even if the oximetry results are normal.

Portable cardiorespiratory testing has also demonstrated utility. Under certain circumstances, the doctor may provide the patient with simplified tests to be used at home to diagnose sleep apnea. These tests usually involve oximetry, measurement of airflow, and measurement of breathing patterns.

A patient with obstructive sleep apnea may be referred by his or her doctor to an ear, nose, and throat doctor (otolaryngologist) to rule out any blockage in the nose or throat. An evaluation by a heart doctor (cardiologist) or a doctor who specializes in the nervous system (neurologist) may be necessary to look for causes of central sleep apnea.

TREATMENT

For milder cases of sleep apnea, the doctor may recommend lifestyle changes such as losing weight, quitting smoking, avoiding alcohol and medications such as tranquilizers and sleeping pills (Veasey et al., 2006). Other recommendations include sleeping on one's side or abdomen rather than on one's back. Nasal passages can be kept open at night by using a saline nasal spray, nasal decongestants, or antihistamines. These medications, however, are generally recommended only for short-term use.

If these measures don't improve signs and symptoms or if apnea is moderate to severe, a number of other treatments are available. Certain devices can help open up a blocked airway. In other cases, surgery may be necessary (Mayo Clinic, 1998). Most alternative medicines for sleep apnea have not been well studied. Acupuncture has shown some benefit but also needs more study and should therefore be used in conjunction with standard treatments rather than as a replacement.

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