Management of sleep apnea in the neurology patient

Five new things

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Summary

Recognizing and treating sleep-disordered breathing (SDB) is essential in delivering neurologic care due to its association with a growing list of neurologic conditions (e.g., stroke, neurodegenerative disorders). Thus, increased proficiency in the recognition and management of SDB is likely to result in better outcomes, care, and utilization of health care resources. To date, continuous positive airway pressure remains the gold standard for patients with moderate to severe obstructive sleep apnea and has been shown to be effective in treating variations of SDB. Appropriate application of new methods and technology such as home sleep testing can help bridge the gap between the need and supply for sleep health care and delivery. Increased focus on efficacious strategies to further awareness and education for patients, caretakers, and providers is paramount to long-term adherence to treatment.

Sleep-disordered breathing (SDB) is one spectrum of sleep disorders that all physicians, particularly neurologists, should have ongoing educational exposure to in order to maintain a baseline level of knowledge proficiency. Mounting evidence suggests that untreated SDB has the potential to negatively affect both patient mortality and morbidity. Untreated sleep apnea, a type of SDB, is a primary risk factor for the development of several medical conditions particularly relevant to neurologists, including hypertension, coronary heart disease, and stroke.¹ SDB is not only common in neurologic patients but also may directly affect the manifestation and management of neurologic disorders.² Thus, it is imperative that neurologists are familiar with SDB and the effect it can have on clinical practice.

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Table 1 Definitions of sleep-disordered breathing

Obstructive apnea	Clinical definition: Apnea is defined as a cessation of airflow for at least 10 seconds. The event is obstructive if during apnea there is effort to breathe. Research definition: A clear decrease (>50%) from baseline in the amplitude of a valid measure of breathing during sleep lasting at least 10 seconds (note: little difference made between obstructive apnea or hypopnea). See figure 1.
Central apnea	Clinical definition: Apnea is defined as a cessation of airflow for at least 10 seconds. The event is central if during apnea there is no effort to breathe. Research definition: Same as above, but an esophageal balloon must verify lack of effort.
Mixed apnea	Clinical definition: Apnea is defined as a cessation of airflow for at least 10 seconds. The event is mixed if the apnea begins as central apnea but toward the end there is effort to breathe without airflow.
Hypopnea	Clinical definition: Several clinical definitions of hypopnea are in clinical use and there is no clear consensus. The Centers for Medicare & Medicaid Services-approved definition of hypopnea is an abnormal respiratory event with at least a 30% reduction in thoracoabdominal movement or airflow as compared to baseline lasting at least 10 seconds and with $\geq 4\%$ oxygen desaturation. Obstruction is often inferred from thoracoabdominal paradox, the shape of the flow signal, or when snoring intensity increases during the event. Research definition: A clear amplitude reduction of a validated measure of breathing during sleep (but less than 50% reduction from baseline) that is associated with an oxygen desaturation of $\geq 3\%$ or an arousal. Only an esophageal balloon can definitively demonstrate the hypopnea to be obstructive vs central.
Cheyne-Stokes	Clinical definition: Cheyne-Stokes respiration is a disorder characterized by recurrent central apneas during sleep with crescendo-decrescendo patterns of tidal volumes. It is often observed in patients with congestive heart failure. ^a
RERA	Research definition: Sequence of breaths with increasing respiratory effort leading to an arousal from sleep, as shown by progressively more negative esophageal pressure for at least 10 seconds preceding an arousal with resumption of more normal pressures.
Flow limitation	Clinical definition: Airflow limitation can be inferred from subtle qualitative changes in the inspiratory portion of the respiratory signals and occurs as a result of increased esophageal pressure that is not accompanied by a flow increase.
Primary snoring	Clinical definition: Noisy vibration of upper airway during sleep without associated alterations in sleep architecture. ^b
Upper airway resistance syndrome	Research definition: Airflow limitation with frequent arousals from sleep due to flow impairment.
Positional sleep apnea	Clinical definition: Apneas that occur mainly while sleeping in certain positions, particularly the supine position. ^c

Abbreviation: RERA = respiratory effort-related arousal.

Sources: Kushida 2005,^{e32} Naughton 1998,³ Dzierwas 2008.⁴

What neurologists should know about sleep-disordered breathing

What is SDB? SDB consists of anomalous respiratory events during sleep that are categorized as apnea(s), hypopnea(s), or both (see table 1 and figures 1 and 2). In most cases, SDB can accurately be determined by an overnight polysomnogram (PSG), the gold standard diagnostic tool to evaluate sleep. A standard PSG can vary in the data collected but at a minimum will include EEG to stage sleep, airflow to identify reductions or cessations of airflow, EMG for muscle tone and leg movements, electrooculogram for eye movements, single-lead ECG for heart rate and rhythm, oxygen saturation, and respiratory belts to measure chest and upper abdominal wall movement. Home sleep testing (HST) allows for the sleep study to be completed in the comfort of the patient's own home (discussed later). Based on specific features related to relative lack of respiratory flow, associated desaturations, respiratory effort, and cortical arousals, these events can collectively categorize an individual at a point along the SDB spectrum, which includes snoring, upper airway resistance, obstructive sleep apnea (OSA), central sleep

Figure 1 Sleep-disordered breathing: Comparative diagram

Source: Anthony Kwan. RERA = respiratory effort-related arousal.

apnea (CSA), complex sleep apnea, and mixed apnea. The clinical and research definitions of specific breathing events are given in table 1 and figures 1 and 2.

OSA, the most common type of SBD, is characterized by the repetitive collapse of the upper airway during sleep, often leading to blood oxygen desaturation and sleep fragmentation.⁵ According to the American Academy of Sleep Medicine (AASM), 2%–4% of Americans have OSA.⁶ OSA is defined as more than 5 disordered respiratory events (described as apneas, hypopneas, or respiratory effort–related arousals; see table 1 and figure 1) per hour of sleep. In some cases, patients will have their obstructive events only when sleeping in a specific position (i.e., positional apnea). These patients may undergo less costly therapies that are designed to shift the patient into positions less likely to be associated with apnea.

CSA is similar to OSA except that during a central event there is no effort to breathe based on minimal movement/stretch of the PSG respiratory belts. CSA is most commonly associated with heart failure, severe neurologic conditions such as large cortical or brainstem stroke, and progressive neurodegenerative disorders such as amyotrophic lateral sclerosis. Patients with mixed sleep apnea experience apneic events that represent a combination of obstructive and central features. Typically the individual event initially demonstrates a central characteristic (e.g., lack of respiratory flow and effort). However, by the end of the event the individual demonstrates effort with maintenance of no airflow and thus ends the event with an obstructive nature (see figure 2).⁴ Lastly, treatment-emergent sleep apnea (formerly known as complex sleep apnea) is a form of SDB in which central apneas emerge or persist during attempts to treat obstructive events with positive airway pressure.

There are other types of SDB events along the spectrum that are also worthy of mention for neurologists. Snoring, which is the audible result of air attempting to pass through a floppy and restricted upper airway, is present in 40%–60% of adults. Although snoring itself does not directly cause major alterations in sleep architecture, it is believed to represent a precursor to the more severe forms of SDB.^{7,8} Further along the spectrum is upper airway resistance syndrome, which involves a similar airflow limitation as snoring but is also associated with frequent arousals. Additionally, respiratory effort-related arousals (RERAs) are events that are associated with an even greater drop in airflow (>30%) and with a cortical arousal. RERAs are considered optional event distinctions that can be quantified on a PSG.⁹ Lastly, although Cheyne-Stokes SDB has never been directly linked to a neurologic disorder, its pattern is worth mentioning since there is robust evidence linking it to reduced heart function.

Who are the “typical” OSA sufferers? Studies have shown that OSA is more prevalent in older adults,¹⁰ men,^{11,12} and individuals of specific racial-ethnic origins.^{13–15} Although women are believed to be less likely to have sleep apnea than men, there is growing evidence that

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women continue to be underdiagnosed.¹⁶ Evidence suggests that women with OSA are far more likely to complain of insomnia, anxiety, depression, and other physiologic comorbidities than men.¹⁷ Men generally tend to experience longer and more severe apneic events.¹¹ Because 70% of patients with OSA are obese, providers often do not consider sleep apnea as a diagnosis in nonobese individuals.¹⁸ Obese individuals have extrinsic narrowing of the pharynx and increased soft tissue enlargement. These anatomical configurations in addition to neck circumference and size of retroglottal space are main determinants of OSA.¹⁹

Despite these commonly cited risk factors and symptoms presented in table 2, OSA remains underdiagnosed. Thus, physicians cannot rely on these common symptoms and risk factors alone and must be attuned to other important features related to patient presentation.

What all neurologists should know about sleep apnea

Who has SDB in your neurology clinic? Stroke and SDB. While OSA affects 2%–4% of the general population, it is observed in 73.7% of ischemic stroke patients.^{e3} Sleep apnea may cause considerable nocturnal perturbations and has been identified as an independent risk factor for both cardiovascular and cerebrovascular disorders.^{5,e4} Aside from the direct effect on stroke risk, SDB can also predispose patients to develop other comorbid conditions commonly associated with increased risk of stroke, such as hypertension, diabetes, obesity, and cardiovascular diseases. Several studies have investigated the potential mechanisms underlying this relationship. Heart rate variability may be altered during sleep as well as wakefulness in patients with sleep apnea.^{e4} Atrial fibrillation and other autonomic and electrophysiologic cardiac dysfunction may also occur during sleep in patients with OSA, increasing the risk for stroke.^{5,e5} Further, sleep apnea patients who do not experience typical blood pressure “dipping” during sleep are at an increased risk for stroke.^{5,13–15,e6} Extreme dipper, nondipper, or reverse dipper patterns in patients with or without apnea have all been associated with intracranial hemorrhages, ischemic strokes, silent brain infarcts, and stroke deaths.⁵ Patients with OSA are also more likely to suffer their stroke event while sleeping, suggesting that

Supplemental Data

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Figure 2 Obstructive airway vs normal airway

Source: Anthony Kwan.

Table 2 Common symptoms and signs of obstructive sleep apnea (OSA)

Common symptoms and signs of OSA	Common risk factors
Daytime sleepiness	Obesity
Loud snoring	Congestive heart failure
Witnessed breathing interruptions	Atrial fibrillation
Awakenings due to gasping or choking	Nocturnal dysrhythmias
Erectile dysfunction	Treatment-refractory hypertension
Morning headaches	Genetic disorders with craniofacial anomalies
Weight gain	Smoking
Changes in mood with increased irritability	Pulmonary hypertension
GERD	Type 2 diabetes mellitus
Multiple awakenings during the sleep period	Stroke
Insomnia	High-risk driving populations
Abbreviation: GERD = gastroesophageal reflux disease. Sources: Watson 2013, ^{e1} Epstein 2009. ^{e2}	

factors specifically related to the dynamics of the SDB event itself may also play a role. For instance, during an obstructive apnea event there is a reduction in cerebral blood flow and impaired cerebral autoregulation.^{5,e7}

While OSA is more commonly associated with stroke, it is important to remember that CSA has also been linked to stroke. CSA may be a consequence rather than a cause of stroke and can be a major symptom of vascular disease.^{e8} Development of CSA is heightened immediately following an acute stroke. It improves during the subacute phase and typically resolves within months after stroke.^{e8} CSA is also associated with congestive heart disease, a risk factor of cardioembolic strokes.^{e9}

Headaches and SDB. Rates of headaches are higher among patients with OSA than in the general population. Chronic daily headaches or frequent headaches upon awakening are more common in patients with OSA than the general population.^{e10} The relative risk of headache, especially daily headache, increases 2–3 times for those with SDB.^{e11} Patients with OSA are 8 times more likely to develop cluster headaches.^{e11} Patients with OSA who are overweight are 24 times more likely to develop cluster headaches.^{e11} Identification and treatment of OSA has been shown to improve cluster headaches.^{e12} Evidence also suggests that headaches may be a symptom of upper airway resistance,^{e13} and inadequate SDB treatment may result in morning headaches.^{e14} The frequency of SDB in migraine is reportedly comparable to the general population.^{e15} However, a small study suggests that treatment of OSA in those with migraine may improve migraine itself.^{e16}

Epilepsy and SDB. A bidirectional relationship has been demonstrated between seizure frequency/severity and presence of SDB.^{e17,e18} Between 20% and 80% of patients with epilepsy have SDB (variation secondary to the degree of severity of epilepsy and criteria for SDB), with some evidence suggesting increased risk of seizures during sleep in those with apnea.^{e19} Intermittent hypoxia and altered sympathetic activity experienced by patients with OSA is believed to activate epileptogenic regions of the brain, potentially lowering the seizure threshold.^{e18} Interestingly, treatment of OSA has been shown to reduce the frequency of seizures.^{e20,e21} Lastly, rates of central apnea in patients with epilepsy are comparable to the general population.^{e22}

Neurodegenerative disorders and SDB. The association between SDB and neurodegenerative disease has recently garnered increasing attention in the neuroscience research arena. The SDB prevalence varies depending on the specific neurodegenerative disorder. There is a particularly

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strong association between Alzheimer disease (AD) and OSA.^{e23,e24} Possible mechanisms underlying the SDB and neurodegenerative disorder relationship include cell loss in brainstem regions responsible for central respiratory regulation and diaphragmatic muscle control.^{e25}

The evidence surrounding Parkinson disease (PD) as a direct risk factor for SDB is unclear. For one, patients with PD have other confounding risk factors, such as age, which increases risk of OSA. On the other hand, patients with PD may be at lower risk of sleep apnea since they have lower weights than controls.^{e26} Mechanisms potentially predisposing patients with PD to OSA may be related to the manifestation of hypokinesia and rigidity that may be associated with airway obstruction, restrictive lung disease, and autonomic dysfunction that subsequently results in SDB pathology.^{e27}

Recognizing and treating SDB in patients with neurodegenerative diseases could improve quality of life.²⁰ When considering all the relevant potential factors involved in this association, neurologists must also be aware of the potential effects on SDB of medications used in this patient population. For example, anxiolytics and pain medications may worsen SDB. Conversely, donepezil has been shown to improve OSA in patients with AD.^{e27}

Neuromuscular disease and SDB. Sleep-related ventilatory difficulties such as diaphragmatic weakness or restrictive lung disease also occur in patients with neuromuscular disease.^{e28} For example, 40%–60% of clinically stable patients with myasthenia gravis are diagnosed with SDB. Given the extraordinary prevalence of clinically significant but routinely underdiagnosed SDB, this figure may be an underestimate of the true prevalence of SDB in patients with neuromuscular disease. Oropharyngeal muscle weakness, tonsillar hypertrophy, obesity, and craniofacial dysmorphias may represent some of the underlying factors increasing risk for SDB.^{e28,e29}

Similar to patients under management for neurodegenerative disease, patients with neuromuscular disease are often placed on medications that can indirectly affect their SDB. Thus, it is important to evaluate these patients for underlying SDB because not only can the SDB affect them clinically, but the medications used for their neurologic disorder may also further negatively affect manifestation and management of SDB.

Sleep diagnosis without a sleep lab? Roles and indications for home sleep testing. PSG is the most commonly used and recognized diagnostic sleep apnea tool. However, this method can be expensive and does require an in-lab overnight study.^{e30} Access to sleep centers and lengthy waiting periods may deter patients from getting tested.^{e31} Furthermore, PSG can be associated with intraindividual variability since the absolute apnea-hypopnea index (AHI) can fluctuate on a night-to-night basis as a result of a number of physiologic and environmental factors.^{e32}

Over the last few years, HST has gained an increasing role in the sleep diagnostic armamentarium. HST refers to the use of portable monitoring devices in the patient's home. HST equipment may be as simple as an overnight oximeter or as complicated as a full unattended PSG (i.e., no technician present). In appropriately identified patients, HST can be an alternative diagnostic tool. Studies have shown equivalence between HST and in-lab PSG with regards to patient satisfaction, quality of life, Epworth Sleepiness Scale and other sleep measurement tools, and continuous positive airway pressure (CPAP) adherence.^{e33} HST allows for less technical expertise, is less labor intensive and time consuming, and is easier for patients to access. HST is most appropriately utilized to diagnose individuals with high pretest probability of moderate to severe OSA.^{e34} The use of HST is not recommended for general screening or clinical use without available knowledge of the patient's sleep-related history

and symptoms or for patients with comorbid conditions or secondary sleep complaints. For instance, patients with comorbid illnesses including lung disease, heart failure, morbid obesity, and neuromuscular disease or patients with comorbid sleep disorders including hypoventilation syndromes, insomnia, parasomnia, or hypersomnia are not considered good HST candidates because these conditions may reduce the accuracy of the test.^{e33} Although HST devices come with varying degrees of automated scoring and interpretation capabilities, the AASM recommends full data access along with override scoring and interpretation capabilities. This allows the reading physician to optimize clinical care and correlation. Current guidelines also recommend that HST be performed only in conjunction with a comprehensive sleep evaluation and under the supervision of a board-certified sleep specialist.^{e35}

Is CPAP the only option? Indications and alternative therapies for clinically significant SDB

Established alternative devices and approaches for the management of apnea. CPAP has been shown to be beneficial in the incidence and recurrence of stroke in patients with OSA as well as in the clinical outcome.^{e36,e37} Despite this efficacy,^{e38} implementation of standard practices for OSA screening and CPAP therapy has been limited due to the unique adherence challenges encountered in these particular patients, often requiring a high degree of social support to ensure adherence.

In cases where administering CPAP poststroke is difficult, conservative approaches (e.g., weight loss and positional therapy) have also been successful. An alternative treatment, bilevel positive airway pressure, was found in a pilot study to result in similar changes in AHI levels to those of CPAP.^{e39} Adapt SV is an adaptive servo-ventilator (ASV) that is used to treat certain types of SDB, particularly those involving central apnea (i.e., CSA, emergent and mixed sleep apnea). ASV is similar to CPAP but works by adapting to the pressure needs of the patient in real time. ASV also lowers AHI^{e40,e41} in emergent sleep apnea. Another device is the average volume assured pressure support (AVAPS), which automatically adapts to disease progression and pressure needs of the patient, particularly in patients with persistent hypoxia or hypercapnia on CPAP. AVAPS and ASV modulate pressure toward set ventilation goals. Lastly, tracheostomy and nocturnal ventilation may be considered for more severe cases of SDB. Lastly, mandibular advancement oral devices are reasonable and approved by the AASM for patients with mild to moderate OSA.^{e36,e42}

Established surgical apnea interventions. Surgical treatment can also be effective for OSA (table e-1). Maxillomandibular advancement surgery (MMA) is an alternative treatment option.^{e43,e44} MMA enlarges and stabilizes the posterior airway with tightening of the upper airway muscles from the forward advancement of both the mandible and the maxilla. The procedure can have a multifaceted effect by correcting facial and occlusal deformities and simultaneously pulling the base of the tongue and soft palate forward and enlarging the size of the oral cavity. MMA is considered a safe treatment for OSA.^{e45} MMA should be considered for patients with severe OSA who have low adherence to CPAP therapy or for patients in whom oral apnea appliances have been found to be unsuccessful.^{e46} Alternatively, maxillomandibular osteotomy may be effective in patients who refuse or cannot tolerate conservative treatment, although further research is needed.^{e42} Other surgical options, including uvulopalatopharyngoplasty (UPPP), pillar implants, and hyoid suspension, are potential considerations, albeit with less evidence of efficacy.^{e47} In the treatment of moderate to severe OSA, UPPP (with or without tonsillectomy) does not reliably normalize the AHI.^{e46} Although UPPP has been shown to have a relatively low success rate, patients with mild to moderate OSA with palate position allowing visualization of uvula or tonsils and tonsil size have shown more than 80% efficacy.^{e48} Palatal implants, used to stiffen the soft palate thereby reducing relaxation and vibration of the tissue, may be a potential treatment option for patients with primary snoring and some patients with mild OSA in whom CPAP therapy or oral apnea appliances have been unsuccessful.^{e46}

The rapid increase in the prevalence of obesity has been linked to comorbidities such as OSA.^{e49,e50,e51} As a result, bariatric surgery is an increasingly common intervention aimed at weight reduction^{e52,e53} and has been reported to be associated with improvement in OSA,

likely through a reduction in intra-abdominal pressure.^{e52} Interestingly, a surgical success rate of above 80% was found in resolving OSA in patients with body mass index ≥ 35 .^{e52}

Experimental/exploratory apnea alternative options. Several devices and surgical procedures are being explored as potential alternatives to CPAP. Nasal expiratory positive airway pressure devices have been shown to be successful for patients with OSA.^{e54} Moreover, the nasal valve device (i.e., Provent) has been reported to improve SDB.^{e55} Noninvasive oral pressure therapy (Winx, ApniCure), which contains a pump applying negative pressure to the oral cavity, was found to be well tolerated while improving sleep, AHI, oxygen desaturations, and sleepiness.^{e56} Lastly, a surgically implantable hypoglossal nerve stimulating system consisting of a neurostimulator that delivers an electrical current to the hypoglossal nerve is the latest novel therapy for OSA. Stimulations are synchronized with respiration-sensing leads that measure changes in breathing.

In closing, determination of the severity and presence of OSA is recommended before initiation of surgical intervention geared toward OSA therapy. Surgical success rates and complications should be addressed with patients when discussing treatment options outside the AASM recommendations. Alternative treatment options such as nasal positive airway pressure and oral apnea appliances should also be discussed.^{e46} At this point in time, the gold standard for patients with moderate or severe OSA is positive airway pressure therapy (i.e., CPAP).

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- Continuous positive airway pressure remains the gold standard for patients with moderate to severe obstructive sleep apnea and has been shown to be effective in treating other sleep-disordered breathing variations.
- Appropriate application of new methods and technology such as home sleep apnea testing can help bridge the gap between the need and supply for sleep health care and delivery.
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