Sleep-disordered breathing is common after stroke and most often presents as obstructive sleep apnea (OSA), with an estimated prevalence between 43% and 70% compared with 4% and 24% in the general population. OSA is the result of partial or complete closure of the upper airway while attempting to breathe during sleep. It is characterized by snoring, hypopnea, and apnea with intermittent awakening accompanied by gasping or choking.1 Daytime sleepiness and fatigue are common features as well. The severity of OSA is measured using the apnea–hypopnea index, oxygen desaturations, and arousals during each hour of sleep.4 OSA after stroke has been shown to increase poststroke mortality and morbidity.2,5 Untreated, OSA may worsen cognition, functional outcome, and mood after stroke.6

Pathophysiology
Several pathophysiologic changes occur in OSA that contribute to stroke, including repeated episodes of hypoxia, resulting in increased negative thoracic pressure with remodeling of the atria and the development of abnormal electric conduction, presenting as atrial fibrillation.3 The oxidative stress of intermittent hypoxia activates the sympathetic nervous system and renin angiotensin aldosterone system, resulting in hypertension and increased plasma fibrinogen levels and platelet activity, creating a state of hypercoagulability. OSA also induces inflammation, which contributes to the atherosclerosis and hypertension commonly present in stroke.3 The hypertension and chronic bouts of hypoxia and hypercapnia associated with OSA may also remodel systemic and cerebral vasculature and impair cerebral autoregulation.3,4 An example of impaired cerebrovascular response is illustrated by the steal phenomenon, referred to as the reversed Robin Hood syndrome seen in some acute ischemic stroke patients with OSA. In the presence of hypoxia and hypercapnia, the cerebral blood flow is diverted away from areas of poor perfusion to areas of good perfusion. This may be a cause of secondary neuronal injury in patients with stroke and OSA.5

In contrast, poststroke CSA occurs as a result of a lack of central input from the brain to the muscles of respiration. The medulla may be less responsive to rising PCO2 levels during sleep. Autonomic networks responsible for respiratory control may be disrupted with bilateral hemispheric infarcts.7

OSA is often unrecognized until stroke occurs. Once the stroke occurs, the cerebral infarct may contribute to a new or preexisting OSA.8 Although primary prevention is optimal, a mounting body of evidence indicates that the diagnosis and effective management of OSA after stroke may decrease the risk of recurrent stroke and enhance stroke outcome.1,6,10,11

Diagnosis
The diagnostic workup of the patient with suspected OSA requires identification of risk factors prior to formalized...
testing. Risk factors for OSA include male sex, obesity defined as a body mass index ≥35 kg/m², neck circumference ≥40 cm, a recessed lower jaw, positive family history for OSA, hypertension resistant to treatment, type 2 diabetes mellitus, atrial fibrillation, congestive heart failure, and previous stroke.4 This information can be readily obtained by the acute or critical care nurse on admission in addition to the nurse’s direct observation of snoring, hypopnea, and apnea and oxygen desaturations.

If OSA is suspected, screening tools such as the Epworth Sleepiness Scale, Berlin Questionnaire, or Snore-Tired-Observed-Pressure-Body Mass Index-Age-Neck-Gender Questionnaire (STOP-BANG) may be used by the bedside nurse to assess for signs and symptoms of OSA.12 Each of these screening tools is easily administered within 10 minutes and have moderate-to-good sensitivity and specificity in the patient with OSA but without stroke. The sensitivity and specificity of the Berlin Questionnaire are 86% and 77%, respectively, in the nonstroke adult outpatients.12,13 The sensitivity and specificity of the Epworth Sleepiness Scale are higher at 94% and 100%, respectively, in the nonstroke adult population.12 The STOP-BANG Questionnaire, which relies on physiological parameters, has a sensitivity and specificity of 90%.12 However, each of these sleep scales is of less value in OSA poststroke because subjective sleepiness, snoring, and obesity may not be present.13–15 The Berlin Questionnaire sensitivity and specificity, for example, decreases to 66.7% and 55.6%, respectively, in the poststroke population.13

The standard workup for OSA is polysomnography consisting of simultaneously recording numerous physiological variables, including electroencephalography, electrooculogram, chin and lower extremity electromyogram, electrocardiography, oximetry, airflow, snoring, and abdominal and chest movements. Polysomnography has historically been exclusively conducted in a sleep laboratory, but more recently, diagnostic testing has been piloted with portable monitors for patients with acute ischemic stroke on hospital units for earlier diagnosis.15 Cardiopulmonary recorders similar to Holter monitors record oximetry, airflow respiratory effort, and snoring sounds. Limitations of portable monitors include possible over- or underestimation of the apnea–hypopnea index.15,16

Management

Once the diagnosis of OSA is made, similar to management of nonstroke-related OSA, continuous positive airway pressure (CPAP) is the standard treatment. CPAP’s mechanism of action is to splint the airway, increase functional residual capacity, and dilate the pharynx.11,17 CPAP may be administered by a nasal mask, a full face mask, or a nasal pillow. The CPAP device must fit securely to maintain the prescribed pressure during inspiration without air leak. CPAP devices may be autotitrating, fixed setting, or variable titration by staff. The most common complications include skin irritation or breakdown or nasal congestion. Increased risk of aspiration should be considered for the patient with stroke because of loss of protective cough and gag reflexes, poststroke nausea, and particularly with a full face mask, the possible inability to remove the mask if vomiting should occur. Also if the patient requires a nasogastric tube, an adequate seal will be unlikely. If the patient requires long-term enteral feeding, a percutaneous tube will be needed to move the feeding tube away from the face. CPAP therapy has been initiated for patients with stroke in acute care, critical care, and inpatient rehabilitation, as well as in home care.11,17

Implementation of CPAP during hospitalization poststroke has been demonstrated to decrease blood pressure at night13 and decrease length of hospital stay by 12 to 13 days.19 Other demonstrated benefits to CPAP for OSA after stroke include improved functional outcome, attention and executive function, neurological status, decreased depression and delirium, and an improved sense of well-being.19,20

Although CPAP therapy has been demonstrated to successfully treat OSA in both the general population, as well as the stroke population, adherence to CPAP therapy is a significant issue. Compliance has ranged from 40% to 84% in the general population and even less in the stroke population, reportedly as low as 30% to 50% short term21,22 and as low as 15% long term.17 Withdrawal from research trials and poor compliance is common in stroke OSA CPAP trials.22 Nurses are key to increasing adherence through education, decreasing contributing factors, and positive reinforcement.21,22

Alternatively, bilevel positive airway pressure (BIPAP), in which the inspiratory positive airway pressure is greater than the expiratory positive airway pressure, has been used in OSA patients who continue to have obstructive sleep events or in those who do not tolerate CPAP. BIPAP lessens the difficulty of exhaling against the higher airway pressures of inspiration. BIPAP may be titrated upward to eliminate airway obstruction and lessen discomfort. A difference of 4 cm H₂O to 10 cm H₂O pressure gradient of inspiratory and expiratory positive airway pressure has been recommended.22 The inspiratory tidal volume goal may be 5 to 7 ml/kg.23 An early study failed to demonstrate greater effectiveness or adherence to BIPAP than to CPAP in nonstroke OSA.23 However, BIPAP has been shown to be well tolerated in ≤90% of acute ischemic stroke survivors with OSA.23

A simple strategy that may improve OSA related to stroke include side lying while sleeping. The backrest position may increase the frequency of apnea/hypopnea events. OSA with supine positioning has been seen in ≤23% to 65% of patients with stroke.25–27 A lateral recumbent position is easily achieved. The judicious use of opioids, sedatives, and hypnotics may also decrease the severity of OSA in the patient with stroke.28

Other interventions that may decrease OSA include weight loss, oral appliances, and bariatric or otolaryngologic surgical procedures. Weight loss is not easily accomplished and maintained, particularly in the patient with stroke and impaired mobility. Oral appliances may be poorly tolerated.22 Surgical procedures are generally reserved for those patients with OSA who are noncompliant or have failed more conservative therapies. The risks and benefits of surgical procedures must be thoroughly assessed prior to undertaking surgery for weight loss or airway management in a patient with a history of stroke and OSA.28

Surgical procedures that may be undertaken to relieve the airway obstruction in OSA include alteration of the uvula, palate, nares, tongue, and jaw. However, the effectiveness of these procedures is inconsistent. Tracheostomy bypass the
upper airway obstruction but is generally considered a surgery of last resort.\textsuperscript{28} Surgical procedures are ineffective in CSA. Although a tracheostomy in CSA provides a stable airway for mechanical ventilation, it does not alleviate the inherent central mediated apnea.\textsuperscript{7}

In addition to direct care, patient education, and support, bedside nurses along with advanced practice nurses may provide much of the input in the development of stroke-related OSA protocols, procedures, order sets, patient education tools, quality improvement projects, and clinical research. The nurse scientist may lead or serve as a member of an interprofessional team in the discovery of new knowledge, ending controversy or substantiating what is known with the conduct of scientific investigations and the dissemination of those findings through publication and presentation.

**Research to Date and Future Directions**

Research related to poststroke OSA has just begun. Unanswered or incompletely answered questions range from additional confirmation of the effect of OSA on stroke in regard to mortality and morbidity and the effectiveness of management strategies to improve stroke outcomes; the impact of interventions to improve adherence; and the reduction of OSA-associated pathophysiology with adherence to an effective management plan. Further investigation into nocturnal blood pressure changes in OSA may also provide insight regarding hemorrhagic transformations after ischemic stroke and intracerebral hemorrhage.\textsuperscript{29,30}

**References**


**Disclosures**

None.


**Key Words:** central sleep apnea | nursing | obstructive sleep apnea | sleep | sleep-disordered breathing | sleep disorders | stroke
Sleep-Disordered Breathing After Stroke: Nursing Implications
Patricia A. Blissitt

Stroke. 2017;48:e81-e84; originally published online February 13, 2017;
doi: 10.1161/STRKEAHA.116.013087

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/48/3/e81