CHEST

Sleep Apnea and Stroke
A Narrative Review

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TOPIC IMPORTANCE: Stroke is the second-leading cause of death worldwide. OSA is an independent risk factor for stroke and is associated with multiple vascular risk factors. Poststroke OSA is prevalent and closely linked with various stroke subtypes, including cardioembolic stroke and cerebral small vessel disease. Observational studies have shown that untreated poststroke OSA is associated with an increased risk of recurrent stroke, mortality, poorer functional recovery, and longer hospitalizations.

REVIEW FINDINGS: Poststroke OSA tends to be underdiagnosed and undertreated, possibly because patients with stroke and OSA present atypically compared with the general population with OSA. Objective testing, such as the use of ambulatory sleep testing or in-laboratory polysomnography, is recommended for diagnosing OSA. The gold standard for treating OSA is CPAP therapy. Randomized controlled trials have shown that treatment of poststroke OSA using CPAP improves nonvascular outcomes such as cognition and neurologic recovery. However, findings from randomized controlled trials that have evaluated the effect of CPAP on recurrent stroke risk and mortality have been largely negative.

SUMMARY: There is a need for high-quality randomized controlled trials in poststroke OSA that may provide evidence to support the utility of CPAP (and/or other treatment modalities) in reducing recurrent vascular events and mortality. This goal may be achieved by examining treatment strategies that have yet to be trialed in poststroke OSA, tailoring interventions according to poststroke OSA endotypes and phenotypes, selecting high-risk populations, and using metrics that reflect the physiologic abnormalities that underlie the harmful effects of OSA on cardiovascular outcomes.

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OSA is characterized by episodes of upper airway obstruction that ultimately lead to disruption of ventilation, intrathoracic pressure changes, and arousals during sleep; they can also cause oxygen desaturation.[1] The degree of OSA severity has traditionally been measured according to the apnea-hypopnea index (AHI), which is the average number of apneas and hypopneas per hour of sleep. An AHI ≥ 5 confers a diagnosis of

ABBREVIATIONS: AHI = apnea-hypopnea index; HSAT = home sleep apnea testing; RCT = randomized controlled trial; TIA = transient ischemic attack

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OSA. However, published normative values for polysomnography metrics in healthy adults may provide more accurate cutoffs for any given age and sex.[2]

The aim of this narrative review was to provide an overview of the association between sleep apnea and stroke. Most, but not all, authors of the papers included and cited in this narrative review explicitly included only patients with stroke with OSA, which is a subtype of sleep-disordered breathing. Sleep-disordered breathing encompasses a spectrum of sleep-related breathing disorders, including OSA, central sleep apnea, and sleep-related hypoventilation. OSA is the most common form of sleep-disordered breathing and can be both a risk factor for stroke and a sequela of stroke.[1]

Literature Search

Links Between OSA and Stroke

Sex Differences in OSA: Risk factors for OSA include increased age, increased BMI, and male sex.[1] In patients with stroke who have been found to have OSA, female participants had greater functional impairment and increased stroke severity compared with male participants, despite having lower OSA severity than male participants.[3] Overall, the study of sex differences in poststroke OSA is limited, and further inclusion of female participants in trials in poststroke OSA may help ensure that results from trials are generalizable across both sexes and facilitate more personalized care for patients with stroke.

OSA and Vascular Risk Factors: OSA is known to be closely associated with multiple vascular risk factors for stroke, particularly hypertension. In a large prospective, population-based study, a dose-response association was noted between sleep-disordered breathing at baseline and the presence of hypertension 4 years later, independent of confounding factors.[4] OSA is also independently associated with a diagnosis of hyperlipidemia and has a negative influence on cholesterol levels in patients with severe OSA.[5] Furthermore, severe, untreated OSA is closely linked with atrial fibrillation and myocardial infarction.[6,7]

OSA as an Independent Risk Factor for Stroke: OSA is an independent risk factor for vascular risk factors that are associated with an increased risk of stroke. However, prior work has also found that OSA itself is an independent risk factor for stroke. A diagnosis of OSA has been found to be associated with stroke or death from any cause; following adjustment for both modifiable and nonmodifiable stroke risk factors (ie, age, sex, race, smoking status, alcohol-consumption status, BMI, diabetes mellitus, hyperlipidemia, atrial fibrillation, hypertension), a diagnosis of OSA was found to be significantly associated with stroke or death.[8] These findings support that OSA, particularly more severe forms, should be considered an independent risk factor for stroke. OSA should be treated as any other vascular risk factor.

Prevalence of OSA After Stroke: OSA is also prevalent following stroke.[9] In a meta-analysis that assessed the prevalence of poststroke OSA, it was found that 72% of patients with stroke had an AHI $>$ 5, 63% had an AHI $>$ 10, 38% had an AHI $>$ 20, and 29% had an AHI $>$ 30.[10] OSA was found to be highly prevalent even 6 months following stroke.[11]

Association of OSA With Stroke Etiology: OSA has been linked most closely with a cardioembolic stroke etiology, and evolving evidence also suggests an association with small vessel disease of the brain.[12,13] The pathophysiology of OSA can help explain these associations (Fig 1).

Small Vessel Disease: A recent meta-analysis suggested a relationship between OSA and cerebral small vessel disease.[13] This meta-analysis found that OSA was associated with up to a fourfold likelihood of having white matter hyperintensities on neuroimaging. Furthermore, OSA severity was associated with the presence of white matter hyperintensities in a dose-response manner. Moreover, moderate to severe OSA (AHI $\geq$ 15) was associated with elevated risk of lacunar infarcts. Collectively, these findings suggest that increasing OSA severity may be linked with different forms of small vessel disease of the brain. The association between OSA and cerebral small vessel disease can be explained by the overcompensatory responses of the autonomic nervous system that occur in the context of OSA, which include surges in sympathetic activation and decreased parasympathetic activity to the blood vessels and end organs. Activation of the sympathetic nervous system can result in hypertension, inflammation, and increased platelet activity, which contribute to the endothelial dysfunction that may result in small vessel disease of the brain.[14] Moreover, the intermittent hypoxemia seen in OSA may also give rise to oxidative stress and inflammation, which can further exacerbate the endothelial dysfunction seen in small vessel disease of the brain.[15]

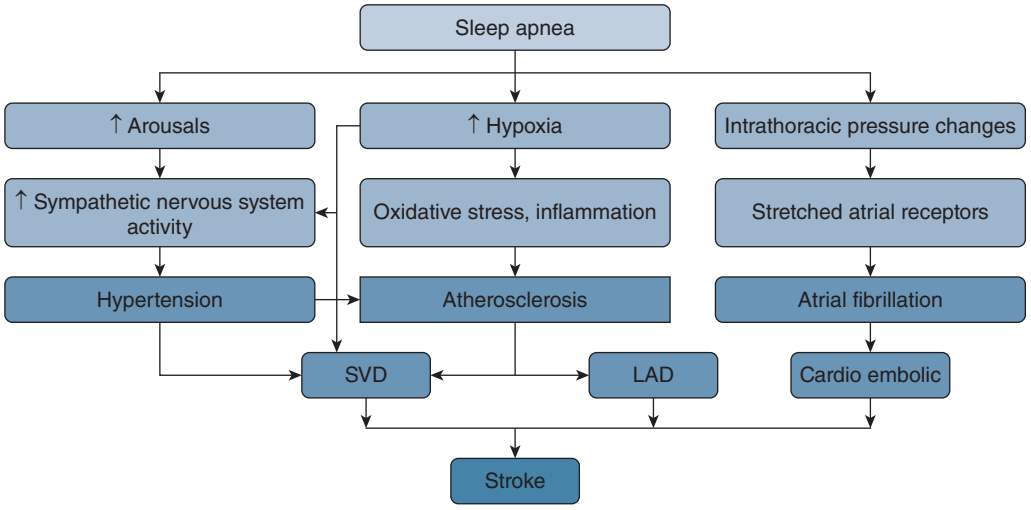

Figure 1 – *Relationship between OSA and stroke. LAD = large artery disease; SVD = small vessel disease.*

Atrial Fibrillation: Several studies have found that OSA is closely related to atrial fibrillation.[6,16,17] This is believed to be related to atrial stretch, which may be caused by OSA through hypoxemia, negative intrathoracic pressure surges, and sympathetic activation.[14] One study found that nocturnal hypoxia, measured by using mean oxygen desaturation from polysomnography, was an independent predictor of atrial fibrillation in patients with subacute ischemic stroke.[18] Furthermore, there is evidence that both nocturnal hypoxemia and pulse rate variability are independent predictors of atrial fibrillation incidence. The highest risk of incident atrial fibrillation was found in patients with a longer duration of oxygen saturation $< 90\%$ and those with greater pulse rate variability.[19]

An initial meta-analysis of CPAP therapy for OSA and risk of atrial fibrillation found that patients treated with CPAP had decreased risk of atrial fibrillation.[20] Subsequent small randomized controlled trials (RCTs) found that there was no reduction in atrial fibrillation in patients with sleep apnea who used CPAP; further adequately powered RCTs are needed, however.[17]

Impact of OSA Following Stroke: OSA is associated with numerous negative outcomes in stroke patients, including poorer cognitive and functional impairment, as well as prolonged hospitalizations compared with stroke patients without OSA.[21] Stroke patients with OSA are also found to have greater mortality and a greater risk of recurrent stroke compared with those without OSA.[22] RCTs have shown that treatment of poststroke OSA using CPAP improves neurologic recovery and quality of life, as well as reduces daytime sleepiness and depressive symptoms.[3,23] Overall, OSA detection and management may be an important strategy to improve poststroke outcomes.

Nocturnal Hypoxemia and Stroke: Older men with severe nocturnal hypoxemia were found to have significantly increased risk of incident stroke.[24] Another study found that severe nocturnal hypoxemia and greater central apneas were associated with mortality in patients with stroke; specifically, patients with stroke who died were found to have lower oxygen saturations and greater percentages of time spent below an oxygen saturation $< 90\%$ compared with survivors.[25]

One possible mechanism between stroke and nocturnal hypoxemia is the close relationship between hypoxemia and white matter hyperintensities. One study found that patients with nocturnal hypoxemia had significantly greater volumes of white matter hyperintensities on brain MRIs, even following adjustment for other vascular risk factors such as age, hypertension, and diabetes.[26] White matter hyperintensities, which are a form of small vessel disease of the brain, are associated with increased risk of stroke, dementia, and death.[27]

Changes in Brain Circulation Due to Nocturnal Hypoxemia: The "reverse Robin Hood" ("rob the poor to feed the rich") or "cerebral blood steal phenomenon" is used to describe the phenomenon that hypercapnia can paradoxically decrease blood flow to an already occluded artery during an ischemic stroke to shift blood to nonischemic areas.[28] This means that intermittent

hypercapnia, which is seen in patients with stroke with nocturnal hypoxemia and sleep apnea, may further compromise perfusion in potentially salvageable brain tissue. One RCT aimed to assess the use of initiating auto-titrating bilevel positive airway pressure within 24 h from stroke onset and compared this approach vs standard stroke care. Clinical outcomes such as National Institutes of Health Stroke Scale scores, functional status, stroke recurrence, and mortality were examined. Unfortunately, the study was stopped prematurely due to low recruitment. Further RCTs are needed to assess the feasibility and effectiveness of using noninvasive ventilation in the context of acute stroke management.

Detecting OSA Following Stroke

Based on well-established evidence that OSA is highly prevalent in patients with stroke and that untreated OSA is associated with poor outcomes, current stroke guidelines suggest that clinicians should consider a sleep study for patients with stroke.[29] However, screening for OSA in patients with stroke has been found to be uncommon.[30]

Questionnaires: Patients with stroke and OSA tend to present atypically compared with nonstroke patients with OSA. Compared with stroke-free individuals, patients with poststroke OSA have significantly lower Epworth Sleepiness Scale scores and lower BMIs.[31] One meta-analysis found that $>$ 25% of patients with stroke and OSA did not snore, whereas $>$ 50% of patients with stroke without OSA snored.[10]

Multiple clinical screening questionnaires are available for OSA; however, a systematic review found that most of these questionnaires had poor predictive value in detecting OSA following stroke.[32] Of the studies conducted to date, the STOP-BANG (or a variation thereof) and the NoSAS scores have shown the most promising results regarding detection of OSA after stroke.[33,34]

The STOP-BANG questionnaire consists of yes/no questions related to snoring, tiredness during daytime, observed apneas, high BP, BMI, age, neck circumference, and sex.[35] One study evaluated the utility of a modified version of the STOP-BANG by removing neck circumference and adding nocturnal oxygen desaturation ("STOP-BAG-O"); this modified version was found to be a valid tool for identifying poststroke OSA.[36]

The NoSAS questionnaire is a clinical screening tool for OSA that is based on a patient's BMI, age, neck circumference, sex, and whether they snore during sleep.[37] For an AHI cutoff of $\geq$ 5, in a study evaluating 221 patients with stroke, the NoSAS had an area under the curve of 0.831 (95% CI, 0.768–0.894; $P < .05$), which was superior to the results seen for the STOP-BANG and Berlin questionnaires.[33]

A limitation of all questionnaire studies is that as one chooses a cutoff that favors the sensitivity, there will typically be a reduction in the specificity (and vice versa). Moreover, although questionnaires have promise for predicting OSA following a stroke, given the atypical characteristics poststroke, objective measures are recommended to assess for OSA in this population.

In-Laboratory Polysomnography: Polysomnography is the current standard tool to diagnose OSA. However, there are barriers that impede the utility of polysomnography, including limited access and wait times, as well as patient unwillingness to sleep overnight in a sleep laboratory.[34]

Ambulatory Sleep Testing: Home sleep apnea testing (HSAT) has been increasingly used to diagnose OSA in stroke populations. Level 3 HSAT has been validated against in-laboratory polysomnography in adults with a high pretest probability of moderate to severe OSA, which includes patients with stroke.[38] HSAT has been found to be feasible for unattended use following a stroke.[39] HSAT can also be used in various settings, including inpatient hospital settings immediately poststroke, rehabilitation facilities, and also in ambulatory settings (eg, a patient's home). In patients with stroke/transient ischemic attack (TIA), an RCT that evaluated use of HSAT (compared with polysomnography) showed that HSAT could facilitate timely detection and management of OSA, which was associated with reduced poststroke/TIA sleepiness and improved functional outcomes, as well as offering a cost-effective approach.[40]

Treatment of OSA Following Stroke

CPAP: Given the negative consequences of untreated OSA, such as increased mortality and ongoing stroke risk, the treatment of OSA in the stroke and TIA population is critical. Currently, the gold standard for treating moderate to severe OSA in patients with stroke is CPAP. CPAP reverses the harmful impact of OSA by acting as a pneumatic splint that keeps the airway open during sleep.[41]

Randomized studies have shown that CPAP treatment can improve patients' nonvascular outcomes such as

cognitive function, sleep quality, and daytime sleepiness in patients with stroke (Table 1).[9,42-54] However, RCTs that have evaluated the effect of CPAP on vascular outcomes and mortality have had mixed results and have been largely negative (Table 2).[44,45,50-52,54,55] The Sleep Apnea Cardiovascular Endpoints (SAVE) study, which was the largest of these RCTs, found that there were no differences in cardiovascular outcomes after 3.7 years in the CPAP therapy arm compared with the usual care arm.[50] However, the average adherence to CPAP in that study was only 3.3 h per night. A preplanned subgroup analysis, which used a propensity score matching scheme that compared patients who adhered to CPAP therapy (ie, use of CPAP for $\geq$ 4 h per night) vs patients in the control arm who would have complied with CPAP therapy revealed a significantly lower risk of stroke in those using CPAP. A meta-analysis of three RCTs found that patients with known cardiovascular disease and OSA who used CPAP for $\geq$ 4 h per night had a significantly lower risk of major adverse cardiac or cerebrovascular events compared with those who used CPAP $<$ 4 h per night.[56]

Unfortunately, adherence to CPAP is typically low among patients with stroke. A meta-analysis showed that CPAP adherence rates in patients with stroke varied from 11% at the 5-year follow-up to 72% at 2-year follow-up, with a mean of 37%.[57] Moreover, only a small proportion of patients with stroke with OSA are treated with CPAP during the initial year following the stroke.[58] Earlier adherence to CPAP has been linked with long-term usage of CPAP in a stroke population.[59] Predictors of CPAP adherence among patients with stroke also include greater functional capacity and those who report less daytime sleepiness.[60] One way to encourage CPAP adherence and reduce dropout rates in patients with stroke could include interdisciplinary coaching during in-hospital rehabilitation and telemedicine monitoring.[61]

Alternative Treatment Options: There are also other interventions that have been developed which could be particularly useful for patients with poststroke OSA who cannot tolerate CPAP. These include positional therapy, mandibular advancement devices, oropharyngeal exercises, hypoglossal nerve stimulation, and pharmacologic agents.

Positional therapy, which involves the avoidance of the supine position during sleep, is a commonly used treatment option in clinical practice for patients with OSA that is aggravated by supine sleep. The prevalence of supine sleep in patients with stroke is high, especially in those who had more severe strokes.[62] Although not widely investigated, one RCT found that patients with stroke who used positional therapy (ie, a therapeutic pillow) had an average reduction of 19.5% of AHI.[62]

Mandibular advancement devices serve to keep the airway open and patent. Compared with CPAP, mandibular advancement devices are not as effective in treating OSA.[63] However, mandibular advancement devices could still be considered for patients who cannot tolerate CPAP, particularly those with mild and symptomatic OSA.

There is also potential for the use of hypoglossal nerve stimulation to treat poststroke OSA, given that patients with stroke have a high prevalence of hypoglossal nerve dysfunction.[64] In the general population, hypoglossal nerve stimulation has been found to be effective in select patients with a BMI $<$ 32 kg/m^2.[65] This approach is potentially promising for patients with stroke, given that patients with stroke with OSA tend to have lower BMIs (compared with patients with OSA in the general population). However, studies are needed to establish the feasibility, safety, and efficacy of hypoglossal nerve stimulation in patients with poststroke OSA.

Similarly to how CPAP acts as a pneumatic splint to keep the upper airway patent during sleep, the strength and function of the muscles in the airway may also play a key role in maintaining airway patency. This provides the basis for oropharyngeal exercises to train the upper airway muscles as a potential treatment option for OSA. Although two small RCTs have assessed the feasibility and efficacy of administering an oropharyngeal exercise regimen to patients with stroke with OSA, larger studies are needed.[66,67]

Currently, there are no pharmacologic options that have been widely studied for individuals with sleep apnea, including poststroke OSA. However, small trials examining combinations of atomoxetine and oxybutynin, as well as reboxetine and oxybutynin, reported reductions in OSA severity.[68,69] These studies highlight the potential use of pharmacologic treatments in OSA; however, none of these agents has been studied in poststroke OSA.

Evidence Review

Designing Future Clinical Trials

There has been an abundance of research in poststroke OSA that has led to important advances

TABLE 1] Randomized Controlled Trials on CPAP Use on Nonvascular Outcomes Poststroke

Study	Diagnostic Criteria for OSA	N (% of Female Sex)	Method Used to Detect OSA	Mean CPAP Adherence (h/d)	Timing of CPAP Initiation Poststroke	Follow-Up Period	Conclusions
Sandberg et al, 2001[42]	AHI ≥ 15	63 (54%)	PSG	4.1	2-4 wk	28 d	↓ depressive symptoms No change in cognition
Hsu et al, 2006[43]	AHI ≥ 30	30 (32%)	Ambulatory sleep test	1.4	2-3 wk	6 mo	No change in depression or cognition
Bravata et al, 2011[44]	AHI ≥ 5	55 (33%)	Ambulatory sleep test	...	Majority within 24-48 h	30 d	↑ neurologic recovery with ↑ CPAP adherence and < 48 h to CPAP initiation
Parra et al, 2011[45]	AHI ≥ 20	140 (29%)	Ambulatory sleep test	5.3	3-6 d	1 mo	↑ neurologic recovery
Ryan et al, 2011[46]	AHI ≥ 15	44 (20%)	PSG	4.96	3 wk	1 mo	↑ neurologic recovery ↓ daytime sleepiness ↑ motor outcomes
Minnerup et al, 2012[47]	AHI ≥ 10	50 (62%)	Ambulatory sleep test	4.2	24 h	8 d	↑ neurologic recovery with ↑ CPAP adherence
Brown et al, 2013[48]	AHI ≥ 5	32 (44%)	PSG	4.5	7 d	3 mo	No change in depressive symptoms
Aaronson et al, 2016[9]	AHI ≥ 15	36 (39%)	Ambulatory sleep test	2.5	1-16 wk	1 mo	No change in neurologic function ↑ cognition
Khot et al, 2016[49]	AHI ≥ 5	40 (45%)	PSG or ambulatory sleep test	3.7	10 d	28 d	↑ cognition
McEvoy et al, 2016[50]	Oxygen desaturation ≥ 12	2687 (19%)	Ambulatory sleep test	3.3 (entire intervention sample, not poststroke subset)	...	3.7 y	↓ snoring and daytime sleepiness ↑ health-related quality of life and mood (in entire intervention group, not stroke subset)
Gupta et al, 2018[51]	AHI ≥ 15	116 (19%)	PSG	4.2	< 6 mo	1 y	↑ functional status ↓ daytime sleepiness No change in cognition
Bravata et al, 2018[52]	AHI ≥ 5	252 (41%)	Ambulatory sleep test	3.9	1-4 wk	6-12 mo	↑ neurologic recovery ↑ functional status
Kim et al, 2019[53]	AHI ≥ 20	40 (28%)	Ambulatory sleep test	4	4.6 ± 2.8 d following admission to stroke rehabilitation	3 wk	↑ cognition ↓ daytime sleepiness ↑ sleep quality
Bernasconi et al, 2020[54]	AHI ≥ 20	41 (22%)	PSG	4	3 mo	18 mo	No change in functional status

AHI = apnea-hypopnea index; PSG = in-laboratory polysomnography (level 1).

TABLE 2] Randomized Controlled Trials on CPAP Use on Vascular Outcomes Poststroke

Study	Diagnostic Criteria for OSA	N (% of Female Sex)	Method Used to Detect OSA	Mean CPAP Adherence (h/d)	Timing of CPAP Initiation Poststroke	Follow-Up Period	Conclusions
Bravata et al, 2011[44]	AHI ≥ 5	55 (33%)	Ambulatory sleep test	…	Majority between 1 and 2 d	30 d	↓ recurrent events (not statistically significant)
Parra et al, 2011[45]	AHI ≥ 20	140 (29%)	Ambulatory sleep test	5.3	3-6 d	2 y	No change in cardiovascular event-free survival
Parra et al, 2015[55]	AHI ≥ 20	126 (29%)	Ambulatory sleep test	5.3	3-6 d	5 y	↑ cardiovascular event-free survival
McEvoy et al, 2016[50]	Oxygen desaturation ≥ 12	2,687 (19%)	Ambulatory sleep test	3.3	…	3.7 y	No change in recurrent events No change in mortality
Gupta et al, 2018[51]	AHI ≥ 15	116 (19%)	PSG	4.2	< 6 mo	1 y	No change in recurrent events
Bravata et al, 2018[52]	AHI ≥ 5	252 (41%)	Ambulatory sleep test	3.9	1-4 wk	6-12 mo	No change in recurrent events
Bernasconi et al, 2020[54]	AHI ≥ 20	41 (22%)	PSG	4	3 mo	18 mo	No change in recurrent events

AHI = apnea-hypopnea index; PSG = in-laboratory polysomnography (level 1).

in this field. Even though the present literature suggests that leaving OSA untreated in patients with stroke is associated with negative vascular outcomes, there is no convincing evidence that treating OSA following a stroke reduces vascular events or mortality risk. However, there are several limitations to the current literature, and novel approaches to designing future RCTs in poststroke OSA are needed.

CPAP, Non-CPAP Interventions, and Timing of Interventions: As discussed earlier, there are several treatment strategies for OSA that have yet to be trialed in poststroke OSA. These modalities used alone or in combination with CPAP (or another modality) may prove beneficial in poststroke OSA. If CPAP is to be used in future trials, adequate adherence to CPAP needs to be achieved. Prior studies have shown that the benefits of CPAP therapy become apparent with at least 4 h of use per night.[56] Use of a run-in period prior to randomization may help address issues around CPAP compliance in trials evaluating patients with poststroke OSA.[23] We also recommend that future trials consider early treatment of OSA (ideally within the first 48 h of stroke) and long-term follow-up calculated in accordance with sample size to ensure adequate power.

Tailoring Interventions According to OSA Endotypes and Phenotypes: The recent literature has provided strong evidence for the presence of four physiologic traits that are thought to significantly contribute to the development of OSA: (1) upper airway collapsibility; (2) increased arousability; (3) heightened loop gain; and (4) reduced dilator muscle responsiveness.[70] However, current diagnostic methods do not facilitate measurement of OSA endotypes and phenotypes, and there is the need to simplify these traits possibly through the use of automated techniques or novel diagnostic techniques. Limited work has explored endotypes and phenotypes in poststroke OSA, and whether these factors influence long-term outcomes. Future trials could consider offering therapies according to the OSA endotypes or phenotypes of participants with poststroke OSA; further research in this area is greatly needed.[71]

Studying Patients With Severe Sleep Apnea and Excessive Daytime Sleepiness: Future trials should study patients with moderate to severe OSA and

excessive daytime sleepiness as evolving evidence in nonstroke populations suggests that patients with excessive daytime sleepiness and an AHI $\geq$ 20 are at greatest risk of all-cause mortality.[72] Although the vascular benefits have yet to be proven, CPAP is an effective therapy for excessive daytime sleepiness in the general population. Although a CPAP trial would require a patient with poststroke OSA and excessive daytime sleepiness to be a part of the control arm, we think this is justifiable from an ethical standpoint given the clinical equipoise in this area.[23]

Alternative Metrics of OSA Severity: As discussed earlier, in isolation, the AHI may be a limited measurement tool on its own due to the multitude of physiologic elements that occur in a patient with sleep apnea (Fig 1).[73] The AHI does not fully take into consideration factors such as duration of respiratory events, arousal threshold, sleep fragmentation, and other pathophysiologic issues. Based on these potential mechanisms, there is potential for other metrics to assess OSA severity beyond the AHI. Targeting patients for CPAP therapy based on these physiologic markers may be helpful when developing novel RCTs.[74] These include pulse rate (heart rate) variability, hypoxic burden, odds ratio product, ventilatory burden, and cardiopulmonary coupling. In particular, cardiopulmonary coupling analysis within 30 days of stroke onset was found to predict severe functional impairment poststroke.[75]

Future Directions

OSA is an independent risk factor for stroke and is associated with multiple vascular risk factors. Conversely, OSA is prevalent following a stroke, and observational studies have shown that untreated poststroke OSA is linked with poorer neurologic and cognitive outcomes, longer hospitalizations, and an increased risk of recurrent stroke and mortality. The current gold standard for treating poststroke OSA is CPAP therapy. RCTs have shown that treatment of poststroke OSA using CPAP improves nonvascular outcomes such as cognition, neurologic recovery, mood, and daytime sleepiness. However, RCTs that have evaluated the effect of CPAP on recurrent stroke risk and mortality have been largely negative. There is an urgent need for high-quality RCTs in poststroke OSA, which may show the utility of CPAP (and/or other treatment modalities) in reducing recurrent vascular events and mortality.

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