Rehabilitation of Cardiovascular Disorders and Sleep Apnea

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KEYWORDS

• Obstructive sleep apnea • Sleep disordered breathing • Cardiac rehabilitation
• Stroke rehabilitation • Cardiovascular disease • Stroke

KEY POINTS

- Obstructive sleep apnea (OSA) is a common disorder in patients entering cardiac rehabilitation units.
- Sleep disordered breathing is underdiagnosed in the poststroke period.
- Early screening and treatment of OSA are important in the management of the poststroke period or after cardiac events.
- Failure to treat OSA in cardiac patients and patients with stroke can have negative impacts on outcomes during rehabilitation.

INTRODUCTION

Obstructive sleep apnea (OSA) syndrome is a disorder affecting 2% to 4% of the general population in the United States.[1–3] It is characterized by recurrent episodes of upper airway collapse during sleep. This condition presents in more than half of patients with acute coronary heart disease who are eligible for enrollment in cardiac rehabilitation programs.[4] Because of the hemodynamic fluctuations associated with OSA, those patients with concurrent OSA who enter cardiac rehabilitation programs may be placed at greater risk for arrhythmias and exercise-related complications.[5] This risk can lead to an increase in major adverse cardiac events such as revascularization, heart failure (HF), hospital readmission, functional limitation, and reduced quality of life in cardiac patients.

In contrast, stroke, with an incidence of 2 to 18 per 1000 per year, is the second leading cause of death worldwide and more than 50% of survivors have mental and physical impairment.[6,7] Although sleep disordered breathing (SDB) has been recognized in patients with stroke since the early nineteenth century,[8] over the last 2 decades it has been emerging as an important cardiovascular risk factor.

In the poststroke period, patients with OSA have greater disability and higher mortality than patients without OSA.[9,10] However, OSA is under-recognized during the poststroke period because of a lack of symptoms or lack of gross obesity, despite evidence that managing this risk factor may benefit those patients.[11] There is growing evidence that treatment of coexisting OSA with continuous positive airway pressure (CPAP) or mandibular advancement devices can successfully treat OSA, resulting in improved rehabilitation outcomes and improved health-related quality of life.[12–15]

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PREVALENCE OF OBSTRUCTIVE SLEEP APNEA IN PATIENTS ENTERING CARDIAC REHABILITATION PROGRAMS

It is well known that patients with different types of cardiovascular diseases have higher prevalence of SDB.[16–19] Several studies have shown that OSA is an independent risk factor for development of hypertension and has a dose-response relationship between severity of OSA and incidence of hypertension.[18,20,21] SDB is also estimated to have a prevalence of 30% to 69% in patients with coronary artery disease (CAD)[22–24] and 50% to 70% in patients with systolic HF.[25,26] However, fewer data are available in HF with preserved ejection fraction (EF) than in those with reduced EF. The largest study on this group was done on 244 consecutive patients: 97 patients (39.8%) presented with OSA and 72 patients (29.5%) with central sleep apnea (CSA).[27]

In the postdischarge period after coronary revascularization procedures, 52% to 64%[28,29] of patients had moderate to severe OSA. A recent study suggested that, overall, 53% of patients entering cardiac rehabilitation programs are at high risk, or are previously diagnosed with OSA.[4] However, the experts believe that the prevalence of OSA is substantially higher and is largely under-recognized in patients entering cardiac rehabilitation programs.[30]

PREVALENCE OF OBSTRUCTIVE SLEEP APNEA IN THE POSTSTROKE PERIOD

In the past decade, longitudinal studies have shown that people with SDB have a greater risk for stroke.[31–35] The reported prevalence in the literature is very high, ranging from 40% to 90%,[9,10,34,36–40] likely reflecting different study designs and definitions of SDB.

Yaggi and colleagues[41] followed 1022 patients over 6 years and found that OSA is associated with an increased risk for stroke, transient ischemic attack, and death. This finding was true even after adjusting for age, gender, body mass index (BMI), arterial hypertension, diabetes, atrial fibrillation, hyperlipidemia, and smoking habits. Patients with severe SDB had a higher chance of having stroke (hazard ratio [HR], 3.3; confidence interval [CI], 1.7–6.3). These results were confirmed by other investigators as well.[35,42–44]

It has been shown that many patients experience a recurrent stroke within 5 years of their first attack[45] and those with recurrent stroke have a higher chance of having SDB compared with first-time stroke victims.[46] Note that presence of an apnea-hypopnea index (AHI) greater than 10 per hour is an independent risk factor for stroke recurrence.[46]

The prevalence of sleep apnea is highest early following stroke and may decrease during recovery,[34,47] reflecting a decrease in sleep apnea in most patients, although it can also be caused by a higher mortality in those with more severe form of disease. Although SDB tends to improve spontaneously several weeks after stroke, approximately 50% of patients still have OSA 3 months after the acute event.[31,32,34] Szücs and colleagues[48] reported that the frequency and severity of sleep apnea were unchanged 3 months later in most patients with ischemic stroke, whereas it was greatly improved in patients with hemorrhagic stroke. Moreover, hemorrhagic strokes lead more often to central apneas.[49]

Both OSA and, less frequently, CSA have been identified as risk factors for stroke.[10] However, only OSA is recognized as a mortality risk for ischemic stroke.[9,48,49] Periodic breathing (PB) may also develop in patients with stroke in the absence of cardiopulmonary disease, disturbed consciousness, or HF.[50] During the acute phase of stroke, obstructive apnea is the most predominant type. Central apneas are associated with altered mental status, brain edema, and ischemia of brainstem and their incidence diminishes during recovery after stroke.[51] Rowat and colleagues[52] investigated the impact of PB in the awake state on mortality in the immediate phase after stroke. They investigated 138 patients with stroke at a median of 4 hours after stroke and found that those with PB were more likely to have severe stroke and more likely to be dead or dependent at 3 months. In contrast, another study showed that CSA was not related to early death among the patients with stroke. Only patients with OSA (AHI >10) had an increased risk of early death.[9]

In a prospective study on 161 patients admitted to a stroke unit, no relationships were found between sleep-related respiratory events and the topographic location of the neurologic lesion or vascular involvement.[34]

WHY OBSTRUCTIVE SLEEP APNEA IS UNDER-RECOGNIZED IN STROKE AND CARDIAC REHABILITATION UNITS

OSA is rarely considered in rehabilitation units despite evidence that managing OSA, when present, may benefit patients with cardiovascular diseases. It has been shown that, despite higher prevalence of SDB among patients with stroke, none of them had been referred for diagnostic studies because SDB had not been suspected clinically because of less sleepiness and lower BMI.