Heart Failure and Sleep Apnea

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Sleep apnea has an association with various cardiovascular diseases and generally, it is classified as either obstructive sleep apnea (OSA) or central sleep apnea (CSA). OSA is a well-known trigger of cardiovascular diseases, whereas CSA is associated with cardiac pathologies that could further worsen prognosis. CSA, rather than OSA, is common in patients with heart failure. However, OSA and CSA often coexist in such patients. OSA is estimated to occur in approximately 10–40% of heart failure patients.1–4 Previous studies have reported that common characteristics of patients with OSA and heart failure are obesity, snoring or old age. Sleep-related upper airway collapse is the pathophysiology of OSA. The inspiratory effort against the occluded upper airway during sleep increases the negative intrathoracic pressure, which leads to an increase of preload (venous return) and transmural ventricular pressure. Intermittent hypoxia and sympathetic nerve activation during sleep also cause deterioration of cardiac function. These factors play a role in worsening heart failure.

CSA occurs in approximately 30–50% of heart failure patients,5–6 and is a factor in increased mortality of patients with chronic heart failure (CHF). The activation of the sympathetic nerve system leads to enhanced central chemosensitivity to CO2, which is a major determinant of negative intrathoracic pressure in CHF patients.7,8 The optimum therapy for CHF appears to have a mitigating effect on sleep apnea, and other measures, such as home oxygen therapy (HOT) or continuous positive airway pressure (CPAP), are used to further control the respiratory disturbance. HOT, CPAP and adaptive servo-ventilation (ASV) therapies will improve CSA.9–11 In addition to optimized medical treatment for heart failure, such as cardiac resynchronization therapy, defibrillators or β-blockers, which is indispensable.12 It has been believed that CSA is related to the severity of heart failure, but several reports have demonstrated that the severity of CSA is related to neither the level of brain natriuretic peptide nor left ventricular ejection fraction. Although systematic evidence is insufficient, optimal treatment of heart failure probably improves hemodynamics, which may lead to an improvement or even termination of CSA. Abe et al suggest that optimal medical treatment in patients with heart failure is the most powerful tool for improving CSA.13 They found that surgical correction in a subset of patients with valvular disease resulted in a remarkable reduction in CSA and their study results support a previously proposed theory that hemodynamic improvement following medical treatment is also related to a significant improvement of CSA. Accordingly, the optimization of medical therapy with a reduction of cardiac filling pressures should be required as an initial step in CSA treatment. Valve repair is the most appropriate treatment in patients with valvular disease. Although device therapies, including HOT, CPAP or ASV, seem to be beneficial, they are not currently recommended in clinical practice because they have no proven effect on mortality and morbidity in such patients. Before introducing device therapy for a CSA patient with heart failure, we need to evaluate whether the patient has received optimal medical therapy, including drug treatment or valve replacement.

Polysomnography (PSG) is the gold standard for diagnosing sleep-disordered breathing, but is not popular among Japanese cardiologists. Instead of PSG, a portable device is widely used for the evaluation of sleep-disordered breathing in heart disease patients. The American Academy of Sleep Medicine14 makes the following recommendations: unattended portable monitoring for the diagnosis of OSA should be performed only in conjunction with a comprehensive sleep evaluation; a portable device is not appropriate for the diagnosis of OSA in patients with significant comorbid medical conditions that may degrade the accuracy of portable monitoring; and portable monitoring is not appropriate for the diagnostic evaluation of patients suspected of having comorbid sleep disorders.15 In the study by Abe et al, PSG recordings were obtained from 150 patients with valvular disease, and PSG technologists at the sleep center laboratory scored the recordings. Their study is one of the largest studies of heart failure patients performed in Japan using PSG instead of a portable device. Almost all Japanese cardiologists are well aware of the time needed to use PSG to examine patients with sleep-disordered breathing. However, routine clinical practice is busy and it can be difficult find the time for this examination. Furthermore, many patients with cardiac disease are not aware of the symptoms of sleep-disordered breathing, such as sleepiness. It is necessary for us to have experience of PSG or device therapies in order to comprehend the clinical measures that are well accepted worldwide. According to the results of this study, it is vital for cardiologists to take the accuracy of PSG into account when they evaluate sleep-disordered breathing in heart disease patients. In the future, many cardiologists may find it better to use PSG instead of a portable device to evaluate sleep-disordered breathing in asymptomatic patients with heart disease.
References


