Sleep-Disordered Breathing and Heart Disease
Is it One Big, Vicious Loop?

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Clinically important sleep-disordered breathing (SDB) affects ∼2% to 4% of the general adult population, and its incidence is highest in the older segment of the population.1 Atherosclerotic heart disease is also largely a disease of older people, and often declarates itself by an ischemic event or the onset of heart failure (HF). SDB causes multiple physiological perturbations, promoting stress and intermittent hypoxia and negatively affecting cardiac function.2 Predictably, untreated SDB may promote atherosclerosis and aggravate cardiac failure.3

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Emerging evidence indicates that reverse causation may also be true. A recent study of patients with HF and proven SDB indicates that, during sleep, fluid from the lower extremities shifts to the neck region of the body, causing mechanical constraints in the upper airway and worsening their SDB.4 Moreover, patients with significant cardiovascular disease can develop ventilatory instability as a result of pulmonary congestion and circulatory delays.5 Although it is well known that ventilatory instability is an important risk factor for central sleep apnea and Cheyne-Stokes respiration,6 recent data indicate that it is also a risk factor for severe obstructive sleep apnea.5–7 With these plausible mechanisms in place, it would be prudent to ask whether, if an individual were to develop HF or experience a myocardial infarction (MI), he or she would develop more severe obstructive sleep apnea. Evidently, HF and MI are interconnected because a third of patients who have suffered from a MI will eventually develop HF within 5 years. Briefly, >6400 men and women ≥40 years of age who were predisposed to heart disease were recruited in 7 large cohorts. A large number of subjects also had polysomnography at baseline and at the fifth year of follow-up. Approximately 40% of the cohort used in this study had an apnea-hypopnea index of >5 episodes per hour, qualifying them for the diagnosis of SDB. This number is much higher than that seen in the general population,1 and although the prevalence of SDB in those with HF is likely higher, the exact figure is not known with certainty.11 The authors have demonstrated that, although an incident MI or HF was associated with a worsening of SDB in the cohort during follow-up, these events did not cause the de novo development of clinically relevant SDB. Even though the average changes in apnea-hypopnea index were relatively small, the changes would be of clinical importance in some subjects.

The Chami et al study has major strengths. The sample size in the study was robust, and polysomnographic measurements were done with stringent quality assurance; therefore, the data were of high quality, and were credible. A major limitation of the study is that, although the SHHS cohort provided community-based sampling of ethnically diverse subjects, the cohort in this study included a high proportion of subjects with SDB. The application of the findings to the general population, and even to the patients with heart disease, has to be done with a great deal of caution. Other limitations inherent to observational studies also apply, and causality cannot be firmly established. Nevertheless, in the presence of plausible mechanism(s) linking fluid shift and ventilatory instability to obstructive sleep apnea, the findings cannot be ignored when caring for patients with recent onset of HF or MI.

Is the vicious loop of cardiac disease and SDB complete? We do not have a definitive answer, but chances are that, in some patients, heart disease and SDB may well form a
vicious loop, with one condition aggravating the other. In large part, we do not have data that are entirely specific to the patient population. The evidence to support the widespread use of sleep studies to diagnose SDB in everyone with recent onset of HF or heart attack is certainly not substantiated; this approach also is not practical. The primary management of these patients should be directed to the underlying heart condition; however, it is reasonable to consider the diagnosis of SDB in patients who have also demonstrated clinical features suggestive of SDB, particularly if they complain of excess daytime sleepiness or fatigue that is disproportionate to the severity of the underlying cardiac disease. We should also consider the diagnostic possibility of SDB in those patients who have not responded to optimal treatment for their underlying cardiac disease. Furthermore, in each individual patient, the clinician should be aware of the consequences of untreated SDB on heart disease, and adequate treatment of their sleep disorders will break the vicious loop of SDB and heart disease.

Disclosures
None.

References

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