COMMENTARY

Sleep Disordered Breathing in Heart Failure: A Contemporary Assessment


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Heart failure (HF) is a public health problem of epidemic proportions, with a prevalence of more than 5 million in the United States, a figure that underestimates the true burden of disease, since a substantial portion of HF in the general population may be subclinical and remain undiagnosed. Despite an annual cost of nearly $30 billion dollars per year in the United States and increasingly sophisticated drug and device therapy, the death rate from HF continues to increase, a trend which fuels interest in comorbid disease associations which may offer alternative therapeutic avenues.

Obstructive sleep apnea (OSA) and central sleep apnea (CSA), collectively referred to as sleep disordered breathing (SDB), are both tightly linked to HF and, for various reasons, may be pathophysiologically important in HF. These relationships have attracted much attention in part because of the perception that SDB and HF frequently coexist. Indeed, available epidemiologic data suggests that the prevalence of SDB in HF may approach 50%. However, the literature to date has been dominated by relatively small case series or originate from sleep laboratory referral populations. By their nature, such studies may artificially magnify the occurrence of disease, but the current report by Macdonald et al provides stronger evidence that such a high prevalence of SDB in HF truly exists.

Among the first and largest to systematically examine the occurrence of both OSA and CSA in a HF population, Macdonald and colleagues found that nearly two-thirds of subjects had either OSA or CSA (a figure which may be underestimated since those with primary diastolic dysfunction or valvular heart disease were excluded). While confirming previously established features of OSA in HF, the paper offers some novel and interesting twists from the standpoint of CSA and Cheyne-Stokes respiration.

Because CSA is generally thought to occur as a consequence of HF, the first approach to CSA treatment is often medical optimization of HF, which includes β-blocker administration as standard therapy. Most of the existing epidemiologic literature pre-dated the routine use of β-blockers in HF, prompting some to assert that previous estimates of CSA prevalence no longer applied in the modern era of HF treatment, assuming that more contemporary analyses would prove that CSA has become less common over time. On the contrary, Macdonald et al have demonstrated that CSA remains highly prevalent in HF despite nearly universal β-blocker usage.

The authors have also taken an important step in the quest to identify appropriate metrics to quantify CSA severity. By convention and ease of application, but without sound evidence for validation, clinicians and researchers alike often apply the apnea-hypopnea index (AHI) to CSA as they would to OSA, a disorder which, while sometimes comorbid with CSA, is pathophysiologically distinct. Although the AHI has been well established to correlate with outcomes in OSA, similar validation data do not exist in the setting of CSA, as outlined in the latest American Academy of Sleep Medicine Scoring Manual. The use of the Cheyne-Stokes respiration (CSR) time metric in the current paper seems intuitive and relatively easy to apply, warranting further exploration as a potential marker of CSA severity.

The investigation by Macdonald and coworkers is not without limitations. Most notably, the validity of unattended portable monitoring in patients with HF is unknown, particularly since such patients have been shown to have shorter sleep times and reduced sleep efficiency compared with control subjects in a community sample. On the other hand, portable monitoring may be better suited for detection of CSA, which can occur during wakefulness as well as sleep, and may be more readily identifiable than OSA by simple pattern recognition of typical crescendo-decrescendo ventilation. What is clear is that further research like that from Macdonald and colleagues is sorely needed if we hope to learn more about the complex interactions between sleep, breathing, and the failing heart.

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