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SLEEP MEDICINE PEARLS

A Change of Heart

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A 65-year-old man was referred for snoring, witnessed apneas, and fatigue. He had ischemic cardiomyopathy (ejection fraction 21%), New York Heart Association class IV symptoms, and American Heart Association Stage D heart failure (HF) requiring continuous intravenous dobutamine.

Diagnostic polysomnography (PSG) showed an apnea-hypopnea index (AHI) of 71 events/h with greater than 50% central sleep apnea (CSA) events and Hunter–Cheyne-Stokes breathing (HCSB) during supine non-rapid eye movement sleep (**Figure 1**). Titration PSG showed worse HCSB with continuous positive airway pressure (CPAP) and bilevel ST therapy (**Figure 2**). Before further testing or therapy could be initiated, the patient underwent placement of a third-generation (HeartMate 3, Abbott, Abbott Park, Illinois, United States) left ventricular assist device (LVAD) as destination therapy. He presented to the sleep provider clinic post-LVAD to discuss his sleep study results and therapy options.

QUESTION: What therapy or further evaluation would you recommend for this patient?

ANSWER: Sleep-disordered breathing can change along with changes in cardiac function. PSG should be repeated post-LVAD to assess current pathology and identify optimal treatment settings.

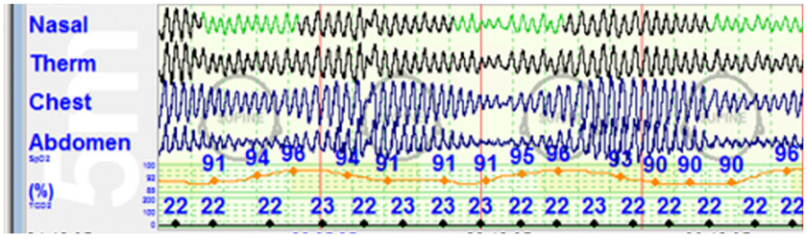
DISCUSSION

Patients with HF may exhibit multiple forms of sleep-disordered breathing.[1,2] Among stable outpatients with self-reported HF in the Sleep Heart Health Study, OSA was most common (55% patients), 12% had comorbid CSA, and only 4% had CSA with HCSB.[1] HCSB was more common among older men with lower body mass index.[1]

As in this clinical vignette, patients with HF often display a subtype of CSA known as HCSB. HCSB is characterized by high loop gain (ratio ≥ 1) and respiratory instability[3] due to altered chemoreceptor and stretch receptor responsiveness (controller gain), cardiac circulatory times (mixing gain), and respiratory efficiency (plant gain).[4] SERVE-HF and other recent studies have raised doubt about the most effective treatment of HCSB in patients with HF and reduced ejection fraction.[5] A subset of patients may respond to CPAP, whereas others may respond to oxygen, lateral sleep position, and carbon dioxide retention to attenuate loop gain.[6] Treatment of HF remains the central focus. LVAD is an established option as a bridge to transplantation or destination therapy.[2]

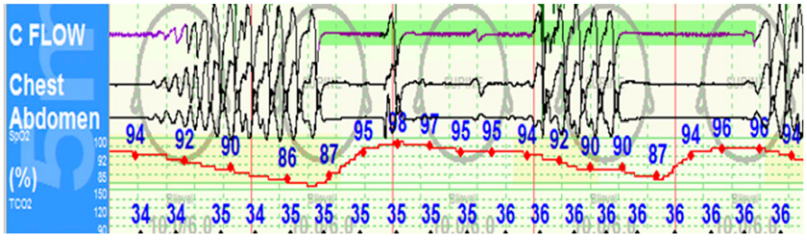
LVAD should improve HCSB by shortening circulatory time and thus reducing loop gain. Evidence is limited to case reports, usually in early LVAD models.[7,8] Early LVADs utilized pulsatile and continuous flow (bearing suspended rotary) pumps. The current third-generation LVADs use a responsive, frictionless magnetic or hydrodynamic rotary levitation system with wide blood-flow passages to simulate a physiologic pulse.[7] There are two reasons to repeat sleep testing post-LVAD in patients with known sleep apnea. First, the form of sleep-disordered breathing may change dramatically with improved cardiac output. Second, persistent sleep apnea events may elevate nocturnal pulmonary vascular resistance and impair LVAD pulsatility index and flow.[8]

Figure 1—Diagnostic polysomnography.



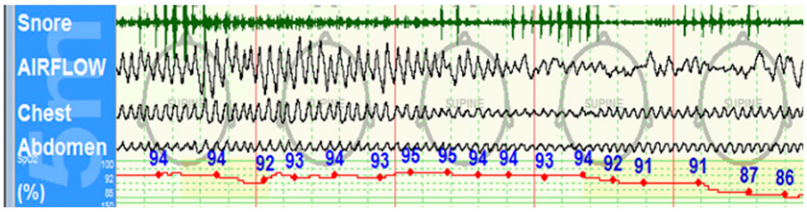
Pre-left ventricular assist device diagnostic polysomnography showing Hunter–Cheyne-Stokes breathing during supine non-rapid eye movement sleep.

Figure 2—Titration polysomnography.



Pre-left ventricular assist device titration polysomnography showing worse Hunter–Cheyne-Stokes breathing on positive airway pressure during supine non-rapid eye movement sleep.

Figure 3—Repeat diagnostic polysomnography.



Post-left ventricular assist device diagnostic polysomnography showing obstructive sleep apnea.

Our patient's hemodynamics improved post-LVAD. Repeat diagnostic PSG showed an AHI of 28 events/h, with obstructive hypopneas and no HCSB (**Figure 3**). Auto-titrating CPAP therapy was initiated, with download showing a residual AHI of 3.2 events/h.

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1. PSG should be repeated post-LVAD because sleep-disordered breathing may change with improved cardiac function and persistent sleep apnea events may impair LVAD function.
2. LVAD should reduce high loop gain via improved cardiac circulatory times and reduced delays in respiratory receptor signaling and response.

CITATION

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ABBREVIATIONS

CPAP, positive airway pressure
CSA, central sleep apnea
HCSB, Hunter–Cheyne-Stokes breathing
HF, heart failure
LVAD, left ventricular assist device
OSA, obstructive sleep apnea
PSG, polysomnography

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DISCLOSURE STATEMENT

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