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SLEEP MEDICINE PEARLS

Abnormal Pulse Oximetry Signal

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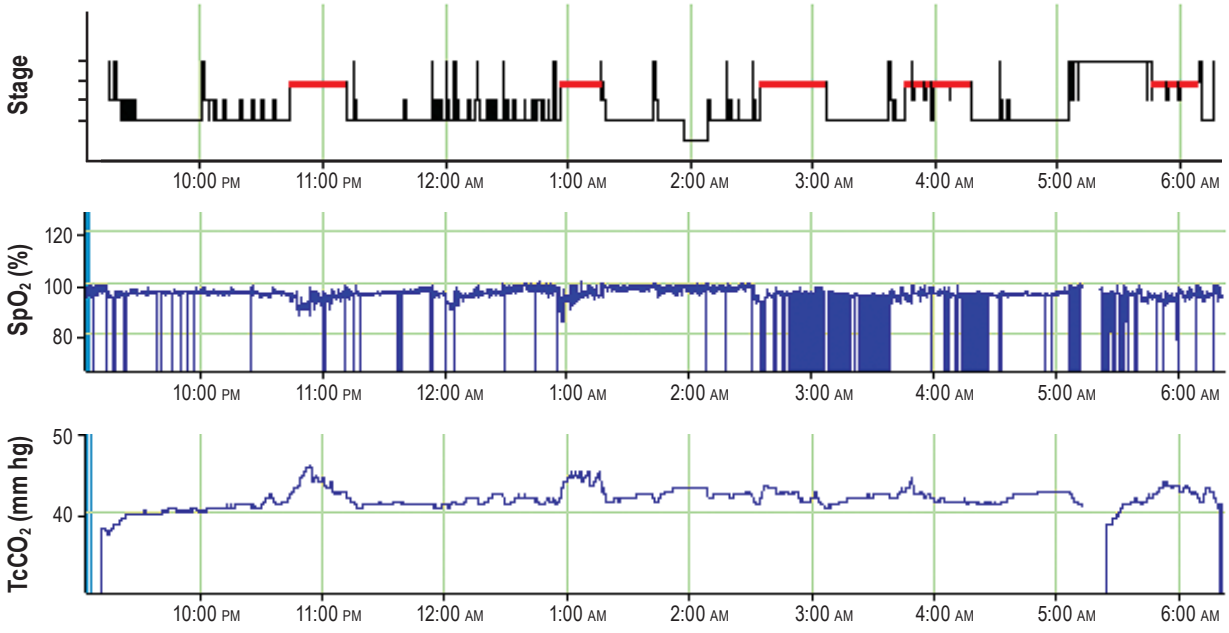
A 45-year-old male patient received a continuous flow left ventricular assist device (LVAD, HeartWare HVAD, Framingham, Massachusetts, United States) because of non-ischemic dilated cardiomyopathy (ejection fraction 10%) associated with long-standing hypertension and iron deficiency anemia. The LVAD was intended as bridge therapy in preparation for heart transplantation. A year later the patient was removed from the transplant list because of recurrent infections and nonadherence, particularly with weight loss.

Thirty months after implantation the patient was referred to a sleep medicine specialist because of loud snoring, witnessed apneas, and morning headaches that predated the LVAD. There were no previous sleep assessments or sleep diagnoses. He experienced fatigue regardless of sleep duration. Epworth Sleepiness Scale score was 9/24. The patient retired to bed at 10:30 PM and arose at 7:00 AM with approximately 3 awakenings. The patient had an urge to move his legs because of evening discomfort that was improved by stretching or movement. Mallampati score was 2, with large tonsils, neck circumference of 43 cm, and body mass index of 39 kg/m^2. Blood pressure could not be measured without Doppler ultrasound due to the LVAD.[1] LVAD speed was 2700 revolutions per minute. The patient had been encouraged to sleep supine to reduce compression of the LVAD components.

The assessment was probable sleep-disordered breathing plus a new working diagnosis of restless legs syndrome. Ferritin level was 28 ng/ml.

QUESTION: How valid is pulse oximetry (SpO_2) done as a component of polysomnography (PSG) for a patient with a LVAD (Figure 1)?

Figure 1—Hypnogram, SpO_2 and $TcCO_2$ data of a patient with a LVAD



LVAD = left ventricular assist device, SpO_2 = oxygen saturation, $TcCO_2$ = transcutaneous CO_2.

ANSWER: The SpO_2 tracing revealed more artifact than customary due to the reduced pulsatility. The lower SpO_2 readings matched the elevated $TcCO_2$ measures during REM sleep reflecting the patient's REM-dependent reduction in ventilation. This correlation confirmed that useful SpO_2 data can be obtained in spite of the artifact.

The patient's PSG revealed a sleep efficiency of 90%, total sleep time of 8.2 hours, arousal index of 25 events/h (57% periodic limb movement related), periodic limb movement index 63.5 events/h, snoring grade 2, and an apnea-hypopnea index of 4 events/h. His SpO_2 nadir was 83%. $TcCO_2$ revealed a mean baseline awake mean of 37 mm Hg with a maximum of 43 mm Hg. As he did not meet criteria for any type of sleep apnea or hypoventilation. The final assessment was that his sleep complaints were chiefly associated with restless legs syndrome. The patient was continued on iron supplementation, vitamin C and started on ropinirole. He was advised to lose weight and avoid sedating medications.

Patients with LVADs experience significantly reduced pulsatile blood flow so the validity of SpO_2 is debatable because its spectrophotometer measures the difference in the amplitude of cardiac pulsations to determine the proportion of oxyhemoglobin.[2] Emergency and critical care medicine use pulse oximetry in patients with LVAD but advise augmenting its measurements with arterial blood gases.[1,3] SpO_2 as a component of PSG for patients with LVAD could also be problematic because of the possibility that the mean arterial pressure is insufficient for reliable detection.[4–6] Citing unpublished experience Akkanti comments that this risk appeared to be theoretical and not confirmed in clinical practice.[4] Routinely employing SpO_2 and $TcCO_2$ monitoring (a measure independent of pulsatile blood flow and less invasive than arterial blood gases), during PSG tests in patients with LVAD increases confidence in the ventilatory assessment. Our case demonstrates, although the SpO_2 signal displayed evident aberrations, interpretation was still possible, validated by $TcCO_2$ data that also revealed a nonpathologic degree of REM-dependent hypoventilation in the absence of any type of sleep apnea.

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1. SpO_2 is dependent on cardiac pulsatility which is markedly reduced with LVAD placement.
2. Blood pressure cannot be measured using conventional tools with a LVAD for the same reason.
3. Patients with LVADs may have increased risk of obstructive sleep apnea because of the desirability of supine sleep, and central sleep apnea because of their underlying cardiac disease.
4. The pulse oximetry signal used during PSG may be detected but with artifact due to the reduction of pulsatility.
5. Adding $TcCO_2$ monitoring provides another noninvasive source of data not dependent on pulsatility that may help validate the SpO_2 results.

CITATION

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