CASE REPORT

“Awakenings”: Electrocardiographic Findings in Central Sleep Apnea

Laszlo Littmann, M.D., Ph.D., Ross M. Nesbit, M.D.,
and J. Matthew Blackwell, M.D.

From the Department of Internal Medicine, Carolinas Medical Center, Charlotte, NC

Central sleep apnea is an important but frequently missed clinical diagnosis. The purpose of this clinical case series is to demonstrate that in a subset of patients with central sleep apnea, inpatient telemetry ECG recordings may reveal a consistent relationship between changes in sinus rate, AV conduction, and the presence and rate of respiratory artifact that should raise the clinical suspicion of central sleep apnea. In the three presented cases, marked sinus bradycardia or AV block was followed by the simultaneous occurrence of abrupt acceleration of heart rate and the appearance of rapid micro-oscillations consistent with respiratory artifact. These changes suggested central sleep apnea characterized by bradycardia during the apneic spells followed by awakening of the breathing center and postvagial tachycardia. In each case, central sleep apnea was confirmed by visual observation of the patient, documentation of arterial desaturations during episodes of bradycardia, and in two, by a subsequent sleep study. Physicians should be aware of the potential and significance of these electrocardiographic disturbances in patients with central sleep apnea.

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Sleep apnea syndrome is a common and frequently undiagnosed disorder associated with significant cardiovascular morbidity and mortality.¹ The recognition of central sleep apnea (CSA) is particularly difficult because patients with CSA do not usually snore.² The gold standard method for the diagnosis of sleep-disordered breathing is the overnight polysomnogram (“the sleep study”). The purpose of this case series is to demonstrate that in some patients, untreated CSA may lead to unique electrocardiographic disturbances in telemetry rhythm strips which, if recognized, may raise awareness of the possibility of CSA even when other classic signs and symptoms of the disorder are not immediately apparent.

CASE PRESENTATIONS

Case 1

A 46-year-old man with history of hypertension, chronic renal failure, and cocaine abuse was hospitalized for hypertensive emergency with pulmonary edema. His initial blood pressure was 240/140 mmHg, and the urine drug screen was positive for cocaine metabolites. On the day of admission, overnight telemetry demonstrated an episode of paroxysmal AV block resulting in asystole of almost 8 seconds in duration (Fig. 1). Subsequent personal observation of the sleeping patient by a physician revealed numerous prolonged episodes of apnea. Each apneic spell was abruptly interrupted by marked hyperventilation and gasping

Address for correspondence: Laszlo Littmann, M.D., Ph.D., Department of Internal Medicine, Carolinas Medical Center, P.O. Box 32861, Charlotte, NC 28232. Fax: +1-704-355-7626; E-mail: Laszlo.Littmann@carolinashealthcare.org

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Figure 1. Case 1: Paroxysmal AV block resulting in a prolonged episode of asystole. Continuous recording.

tachypnea at fast respiratory rates. Review of telemetry showed that each observed event coincided with an episode of paroxysmal AV block with characteristics demonstrated in Figure 2. In this representative illustration, the 30-second continuous rhythm strip panels both demonstrate gradual sinus deceleration with progressive prolonga-

tion of the PR intervals culminating in transient complete heart block and asystole (top strips). The episodes of asystole were followed by acceleration of the heart rate, normalization of the PR intervals, and the appearance of repetitive micro-oscillations consistent with respiratory artifact (middle strips).³ The respiratory rate, as calculated from the

Figure 2. Case 1: Two 30-second continuous telemetry strips demonstrate gradual sinus deceleration and progressive prolongation of the PR intervals culminating in paroxysmal AV block. This is followed by normalization of the PR intervals with emergence of rapid respiratory artifact indicative of extreme tachypnea. Bullets below the strips indicate the presence of respiratory artifact. RR indicates respiratory rate. See text.

Figure 3. Case 2: Five 13-second lead II rhythm strips selected from a single overnight telemetry recording. In each panel, profound sinus bradyarrhythmia is interrupted by the sudden acceleration of heart rate and the simultaneous emergence of respiratory artifact, marked by bullets. The enlargement of the bracketed portion of the top panel demonstrates one of the micro-oscillations indicative of respiratory artifact. Please note that interpretation software labeling of some of the QRS complexes is inaccurate. HR indicates heart rate. See text.

respiratory artifact, ranged from 54 to 76 breaths/min, indicating extreme tachypnea.³ The respiratory artifact then gradually disappeared and both the heart rate and PR intervals stabilized (lower strips). These findings are consistent with CSA that, in addition to the visual observation of the patient, was further confirmed by pulse oximetry demonstrating marked arterial desaturations during the episodes of AV block, and by a subsequent sleep study.

Case 2

A 36-year-old man with idiopathic dilated cardiomyopathy was admitted for heart failure exacerbation symptoms. Overnight telemetry strips revealed multiple episodes of profound sinus bradyarrhythmia followed by sudden acceleration of

the sinus rate, as demonstrated in Figure 3. The abrupt doubling of the heart rate always coincided with the appearance of repetitive micro-oscillations consistent with respiratory artifact.³ Based on the characteristic findings of marked sinus bradycardia followed by the abrupt and simultaneous occurrence of sinus acceleration and the appearance of rapid respiratory artifact, the diagnosis of CSA was suspected. A subsequent sleep study confirmed severe sleep-disordered breathing with a predominance of CSA.

Case 3

A 63-year-old man with a long history of poorly controlled hypertension was hospitalized for hypertensive emergency with pulmonary edema. Overnight telemetry revealed numerous episodes

Figure 4. Case 3: Two continuous telemetry rhythm strips demonstrate sinus bradycardia and junctional escape rhythm followed by the abrupt increase in heart rate and the emergence of respiratory artifact indicating increased work of breathing and tachypnea. The respiratory artifact is marked by bullets. HR indicates heart rate; RR indicates respiratory rate. See text.

of profound sinus bradycardia and junctional escape rhythm as shown in the two continuous rhythm strip panels in Figure 4. On each occasion, the bradycardic period was interrupted by the abrupt doubling of the heart rate and the simultaneous appearance of repetitive micro-oscillations indicating increased work of breathing and tachypnea.³ Based on these telemetry findings, CSA was suspected. Overnight pulse oximetry confirmed sleep apnea by documenting arterial desaturations during the episodes of bradycardia. This patient was lost to follow-up and therefore did not undergo a sleep study.

DISCUSSION

CSA is a common finding in patients with heart failure.⁴ It is characterized by the periodic cessation of breathing with no respiratory effort during which sympathoinhibitory mechanisms predominate, usually leading to sinus bradycardia or AV block.^{2,4} Upon awakening from apnea, there are sudden increases in the heart rate, respiratory rate,

and respiratory muscle tone. CSA can be suspected when family members or other observers report periods of cessation of breathing followed by abrupt hyperpnea. In the absence of such direct observation, the diagnosis of CSA may remain elusive. This case series demonstrates that in a subset of patients with CSA, simple review of telemetry rhythm strips may raise awareness to the possibility of this disorder.

Previous studies using Holter monitoring have found cyclic variations in the heart rate of ambulatory patients with sleep apnea.^{5,6} Our study extends these observations to patients hospitalized with acute decompensated heart failure by demonstrating that in CSA, the sudden heart rate acceleration that follows the bradycardic/apneic episode may also be associated with the emergence of repetitive micro-oscillations. These micro-oscillations represent respiratory artifact whose presence indicates increased work of breathing.³ The respiratory artifact, when present, also allows the calculation of the respiratory rate. In each of the presented cases, awakening of the

breathing center was associated with marked tachypnea.

In case 1, telemetry recording of paroxysmal AV block led to the visual observation of the patient during sleep, the discovery of periods of apnea and then, to the described characteristic ECG findings. In cases 2 and 3, the typical telemetry pattern of bradycardia followed by tachycardia and tachypnea actually led us to suspect CSA. A fourth similar case where telemetry findings pointed to the possibility of CSA has recently been described.⁷

The most appropriate treatment of CSA in patients with heart failure is a subject of controversy.^{2,8,9} On the one hand, our study suggests that extreme cases of CSA may be associated with episodes of profound bradyarrhythmia including paroxysmal AV block, but typically, there should be no rush to proceed to aggressive treatment measures such as a temporary pacemaker. On the other hand, the demonstrated episodic severe bradyarrhythmia may provide some explanation why patients with CSA may benefit from chronic pacemaker therapy.^{10,11}

In summary, physicians should be aware of the electrocardiographic disturbances seen in some patients with CSA. These disturbances often manifest as heart rate turbulence characterized by episodic sinus deceleration or AV block followed by abrupt heart rate acceleration and the simultaneous emergence of rapid respiratory artifact. Given the appropriate clinical scenario, further extensive cardiac testing to explain the bradycardia

or AV block is often unnecessary, and treatment initially should be directed at the underlying condition including treatment of suspected sleep apnea. Furthermore, the presence of these characteristic telemetry findings in a previously undiagnosed patient should prompt consideration for overnight polysomnography.

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