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The diagnostic use of respiratory artifact

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Abstract Respiratory artifact in the electrocardiogram usually indicates labored breathing due to compromised cardiac or pulmonary function. This case series illustrates that correlating the presence and characteristics of respiratory artifact with the ECG morphology and with changes in heart rate frequently allows us to reach specific clinical diagnoses.

Keywords: Respiratory artifact; Emphysema; COPD; Central sleep apnea; Obstructive sleep apnea; Electrical alternans; Cheyne-Stokes breathing

We have recently shown that in hospitalized patients, 12-lead electrocardiograms (ECGs) and monitor recordings frequently demonstrate repetitive microoscillations.[1] These were noted to be respiratory artifact whose presence signified increased work of breathing and a high-risk state often necessitating ventilatory support. When present, the respiratory artifact constituted the second vital sign in the ECG: it allowed for the precise tracking of the respiratory rate. The respiratory rate can be approximated by counting the number of microoscillations in a 10-second recording and multiplying it by 6. A more accurate rate assessment is achieved by measuring 1 or more respiratory artifact cycles in millimeters and dividing it into 1500 or its multiples.[1] In general, most patients with respiratory artifact had diseases of the respiratory or circulatory systems. In this series, we would like to show that correlating the presence and characteristics of respiratory artifact with the heart rate and ECG morphology frequently enables us to reach a more specific clinical diagnosis.

Chronic obstructive pulmonary disease exacerbation

The ECG in panel A (Fig. 1) was recorded from a 63-year-old man who presented with acute respiratory distress. It demonstrated sinus tachycardia with tall and peaked P waves in the inferior leads, negative P waves in lead aVL, poor R wave progression in the chest leads, and what appeared to be a flat line in lead I. A flat line appearance in lead I ("lead I sign")[2] and negative P waves in aVL[3] signify emphysema. Both are believed to be the result of a downward displacement of the diaphragm and the consequent verticalization of the P, QRS, and T wave axes.[2,3] The rhythm strip at the bottom of panel A revealed repetitive microoscillations consistent with respiratory artifact indicating increased work of breathing.[1] The heart rate was 110 beats/min and the respiratory rate, as calculated from the rate of the respiratory artifact, was 38 breaths/min. This ECG, therefore, suggested severe emphysema, labored breathing, and tachycardia with marked tachypnea.

The ECG in panel B was recorded from a 76-year-old woman who also presented with respiratory distress. The morphologic features including the negative P waves in aVL, the flat line appearance in lead I, the poor R wave progression in the chest leads, and the presence of respiratory artifact as well as the tachycardia and tachypnea were essentially identical to the findings in panel A.

A flat line appearance in lead I[2] and negative P waves in aVL[3] combined with tachycardia and rapid respiratory artifact[1] should be considered to be the ECG footprints of chronic obstructive pulmonary disease exacerbation.

Central sleep apnea

The illustration (Fig. 2) demonstrates telemetry recordings obtained from a 54-year-old man who was hospitalized for acute decompensated heart failure. Panel A is the nocturnal heart rate trend curve. It shows an overall normal heart rate with episodic fluctuations in the heart rate as indicated by the

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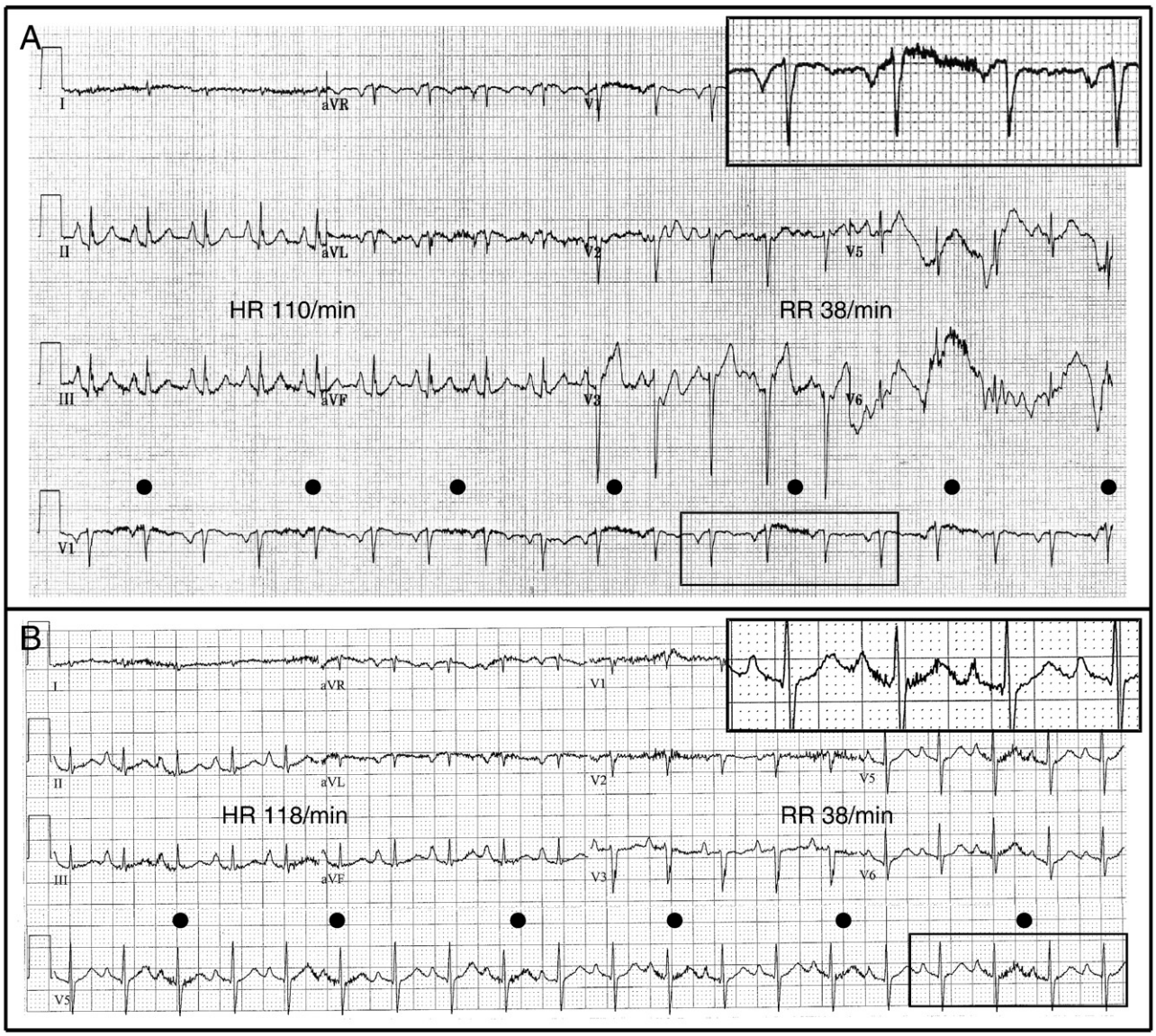

Fig. 1. Two cases of chronic obstructive pulmonary disease exacerbation. In both panels, bullets above the rhythm strips in the bottom indicate respiratory artifacts. The inserts in the upper right corner of each panel are enlargements of the bracketed portions of the respective rhythm strips. HR indicates heart rate; RR, respiratory rate. See text.

circles. Panels B and C are representative examples of numerous similar continuous 20-second rhythm strips recorded during the periods of heart rate turbulence. In both panels, marked sinus bradycardia, with junctional escape complexes in panel B, was interrupted by a sudden increase in the heart rate. On both occasions, the abrupt increase in the sinus rate coincided with the emergence of rapid respiratory artifact consistent with increased work of breathing and tachypnea.[1] Central sleep apnea is a common finding in patients with advanced heart failure.[4] It is characterized by the periodic cessation of breathing with no respiratory effort during which sympathoinhibitory mechanisms predominate and the heart rate is usually slow. Upon awakening from apnea, there is a sudden increase in respiratory rate and respiratory muscle tone with gasping episodes and the onset of postvagal tachycardia.[5] The presence of rapid cyclic microoscillations in the bottom strips of panels B and C represented gasping tachypnea on arousal from apnea. A sleep study revealed severe sleep-disordered breathing with a predominance of central sleep apnea.

Previous studies using computerized signal processing have shown that analysis of the respirophasic modulation of surface ECG measurements may provide information about the respiratory rate and patterns of sleep-disordered breathing.[6] Our current study demonstrates that simple visual observation of ECG rhythm strips alone, without sophisticated signal processing, can sometimes offer diagnostic clues to the presence and type of sleep-disordered breathing. In telemetry recordings, periodic nocturnal heart rate turbulence that is characterized by repeat episodes of slow heart rates interrupted by the simultaneous emergence of sudden sinus acceleration and rapid respiratory artifact should raise the clinical suspicion of central sleep apnea.