

LASZLO LITTMANN, MD, PhD
Department of Internal Medicine, Carolinas
Medical Center, Charlotte, NC, and Professor
of Medicine, University of North Carolina,
Chapel Hill

The Clinical Picture

Wide QRS complex rhythm with pulseless electrical activity

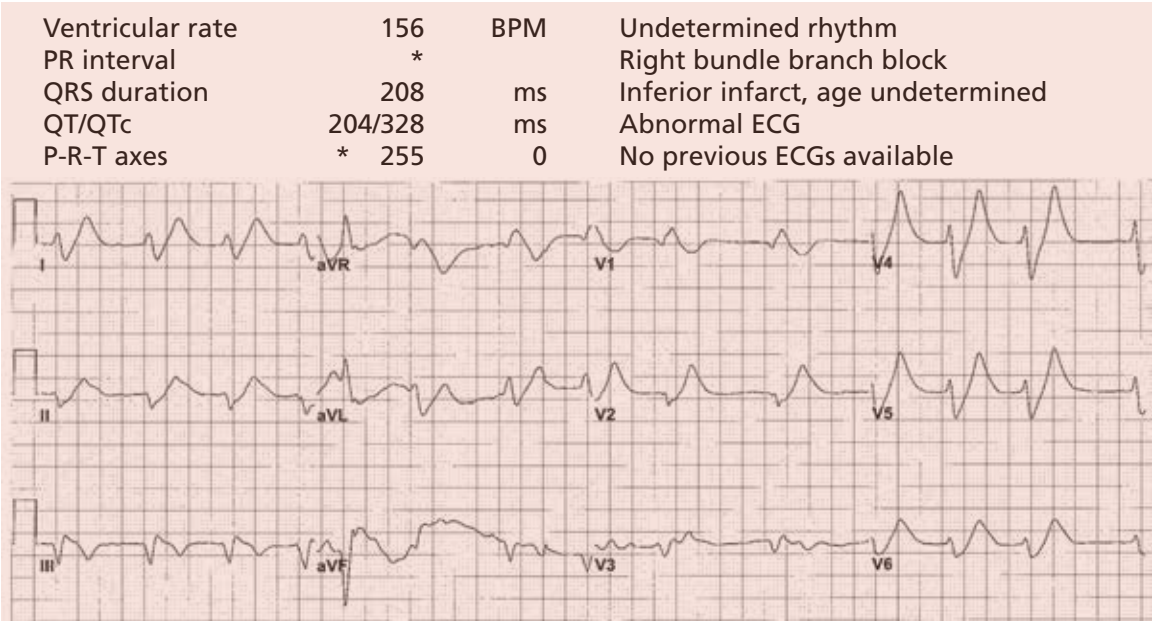


FIGURE 1. At presentation, the standard 12-lead electrocardiogram demonstrated an irregular wide-QRS-complex rhythm with features of right-superior-axis deviation and right bundle branch block. Although the interpretation software indicated a ventricular rate of 156 bpm, inspection reveals the rate to be only half that (ie, “double counting” of the heart rate). The serum potassium concentration at the time of this test was 8.9 mmol/L.

A 64-YEAR-OLD MAN with chronic kidney disease and recent upper gastrointestinal hemorrhage suffered pulseless electrical activity and cardiac arrest. Cardiopulmonary resuscitation was started, with three attempted but failed electrical cardioversions. Return of spontaneous circulation required prolonged resuscitation efforts, including multiple rounds of epinephrine, calcium, and sodium bicarbonate. The standard 12-lead electrocardiogram (FIGURE 1) showed an irregular wide-QRS-complex rhythm, with right bundle

branch block and right-superior-axis deviation. What was the cause of the pulseless electrical activity and the features on the electrocardiogram? The presentation of cardiac arrest with pulseless electrical activity usually has a grave prognosis, and in the acute setting, the cause may be difficult to establish. However, several conditions that cause this presentation have treatments that, applied immediately, can lead to quick and sustained recovery.¹ Electrocardiography can be a powerful tool in the urgent evaluation of pulseless electrical activity.^{2,3} Narrow-QRS-complex pulseless electrical activity is often

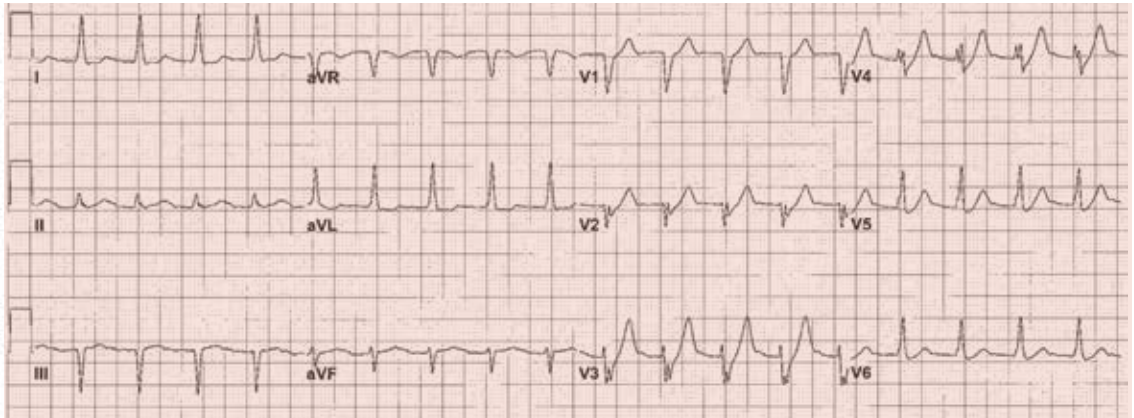


FIGURE 2. After aggressive treatment of the hyperkalemia, the QRS complexes narrowed and the QRS axis normalized, but the chest leads demonstrated J-point depression followed by upsloping ST segments, culminating in tall, peaked T waves. This pattern is also occasionally seen in acute occlusion of the left anterior descending coronary artery. The serum potassium at this time was 7.1 mmol/L.

ECG can be a powerful tool in the urgent evaluation of pulseless electrical activity

caused by mechanical factors such as cardiac tamponade, tension pneumothorax, pulmonary embolism, and major hemorrhage.³ Pulseless electrical activity associated with a wide QRS complex and marked axis deviation, as in this patient, is usually the result of a metabolic abnormality, most often hyperkalemia³; additional indicators of severe hyperkalemia include ST-segment elevation in the anterior chest leads (including the Brugada pattern⁴) and, as in this patient, “double counting” of the heart rate by the interpretation software (FIGURE 1).^{5,6}

Based on the suspicion of a metabolic cause, the serum potassium was tested and was 8.9 mmol/L (reference range 3.5–5.0). The patient was given intravenous calcium, sodium

bicarbonate, glucose, and insulin, and 2 hours later the serum potassium had decreased to 7.1 mmol/L. At that time, the electrocardiogram (FIGURE 2) showed a regular rhythm with ectopic P waves, probably an ectopic atrial tachycardia. There were now narrow QRS complexes with J-point depression, upsloping ST segments, and tall, hyperacute T waves in the chest leads—a pattern recently described in proximal left anterior descending coronary artery occlusion.⁷ The electrocardiographic similarities in hyperkalemia and acute myocardial infarction are probably the result of potassium accumulation in the ischemic myocardium associated with acute coronary occlusion.⁷

The patient had a full recovery, both clinically and on electrocardiography. ■

REFERENCES

1. Saarinen S, Nurmi J, Toivio T, Fredman D, Virkkunen I, Castrén M. Does appropriate treatment of the primary underlying cause of PEA during resuscitation improve patients' survival? *Resuscitation* 2012; 83:819–822.
2. Mehta C, Brady W, Haley MW. Pulseless electrical activity in cardiac arrest: electrocardiographic presentations and management considerations based on the electrocardiogram. *Am J Emerg Med* 2012; 30:236–239.
3. Littmann L, Bustin DJ, Haley MW. A simplified and structured teaching tool for the evaluation and management of pulseless electrical activity. *Med Princ Pract* 2014; 23:1–6.
4. Littmann L, Monroe MH, Taylor L 3rd, Brearley WD Jr. The hyperkalemic Brugada sign. *J Electrocardiol* 2007; 40:53–59.
5. Littmann L, Brearley WD Jr, Taylor L 3rd, Monroe MH. Double counting of heart rate by interpretation software: a new electrocardiographic sign of severe hyperkalemia. *Am J Emerg Med* 2007; 25:584–586.
6. Tomcsányi J, Wágner V, Bózsik B. Littmann sign in hyperkalemia: double counting of heart rate. *Am J Emerg Med* 2007; 25:1077–1078.
7. de Winter RJ, Verouden NJ, Wellens HJ, Wilde AA; Interventional Cardiology Group of the Academic Medical Center. A new ECG sign of proximal LAD occlusion. *N Engl J Med* 2008; 359:2071–2073.

ADDRESS: Laszlo Littmann, MD, PhD, Department of Internal Medicine, Carolinas Medical Center, PO Box 32861, Charlotte, NC 28232; e-mail: Laszlo.Littmann@carolinashealthcare.org