Electrocardiogram in an Attempted Suicide

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A 54-year-old man attempted suicide with a gunshot to the right external auditory canal. In the emergency department, he was alert and combative, could not recall details of the event, and his Glasgow coma scale was 15. Computed tomography of the brain showed longitudinal right petrous bone fractures and comminution of the middle ear. Bone fragments were intracerebral just above the petrous ridge. Tiny lead particles were scattered throughout the brain, primarily in the posterior portion of the right temporal lobe and the right lateral ventricular atrium, with a few having crossed the midline to lodge in the left parietal and occipital lobes. There were also subarachnoid, intraventricular, and subdural hemorrhages, scattered pneumocephalus, and mild uncal herniation. The patient had intermittent episodes of systemic arterial hypertension (as high as 175 mmHg systolic and 101 diastolic) and bradycardia (as slow as 43 beats per minute) during the first five days of hospitalization. The patient’s first electrocardiogram showed a gradually slowing sinus rate and marked sinus arrhythmia with R-R intervals varying from 0.63 to 1.26 seconds. Two days later, the fourth hospital day, the electrocardiogram shown below was recorded.

Figure: Electrocardiogram recorded on the fourth hospital day. See text for explication.

What is your diagnosis?
Explication is on p. 167
ECG of the Month
Presentation is on p. 166

DIAGNOSIS: Sinus bradycardia with an atrial escape complex, followed by a junctional escape rhythm with isorhythmic atrioventricular dissociation; voltage criteria for left ventricular hypertrophy.

The first P wave is upright in leads I, II, and III and is of sinus node origin. The second P wave has a different morphology in leads II and III and is probably an ectopic atrial escape complex. Thereafter, similar narrow QRS complexes occur with minimal irregularity at a rate of 46 complexes per minute. P waves seemingly have disappeared, but close inspection of the lead II rhythm strip reveals an upright P wave to be buried in each of the latter six QRS complexes, as manifested by their having a slightly wider base than either of the first two QRSs and by a slight notch occurring at the end of each of the latter six R waves (best seen in the last and third from last QRSs). Thus, there is sinus bradycardia with a junctional escape rhythm and isorhythmic atrioventricular dissociation. The sum of $SV_1 + RV_5$ just exceeds 35 mm (3.5 mV) and therefore meets one of the criteria of Sokolow and Lyon for left ventricular hypertrophy.¹

When increased intracranial pressure diminishes perfusion of the brainstem, the sympathetic nervous system is activated, causing an increase in heart rate and blood pressure. The arterial hypertension stimulates the carotid baroreceptors, and the sinus rate is reflexly slowed, which in this patient allowed the junctional escape rhythm to emerge.

Thus, the patient with increased intracranial pressure frequently presents with high blood pressure, bradycardia, and occasionally a slow respiratory rate, which often is a harbinger of death. This phenomenon was first described in 1901 by Harvey Cushing, the founder of neurosurgery in America.²

In the latter portion of the electrocardiogram displayed, there is neither atrioventricular conduction with capture of the ventricles nor ventriculoatrial conduction with atrial captures because the atrioventricular node is refractory due to impulses arriving simultaneously from the atria above and the junction below. This synchronization of dissociated Ps and QRSs may be brief and simply happenstance. When it is present over a protracted period, the coupling is thought to be due to some biologic stimulus, such as electrotonus.³

REFERENCES


Dr. Glancy is a Professor and Dr. Chen is a Fellow in the Sections of Cardiology, Departments of Medicine, Louisiana State University Health Sciences Center and LSU Interim Public Hospital in New Orleans.

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