Country cardiograms case 44: Answer

Sara F.M. Awad, MBBS
Ahmad Raed Tarakji, MD, MSPH, FRCPC, FACP, FASN
Ihsan Rafie, MB ChB, MRCP(UK), CCT (Cardiology)
Department of Medicine, Yarmouth Regional Hospital, Yarmouth, NS

Initial electrocardiography (ECG) shown in Figure 1 (on page 110) shows normal sinus rhythm with ST–T changes compatible with chronic repolarization abnormalities rather than acute cardiac ischemia, especially considering that the patient did not report chest pain.

Her cardiac telemetry strips (Fig. 2, on page 111) show sinus bradycardia, changing to nodal beats and then asystole due to sinus node arrest. The sinus node arrest is shown as a flat straight line with no P wave or escape junctional or ventricular QRS complexes seen. The patient suddenly gasped and woke up. The strip showed that the rhythm gradually reverted back to sinus rhythm, her heart rate increased to more than 50 beats/min, the pause disappeared and the patient denied any symptoms.

She was admitted to the intensive care unit. Results of laboratory investigations, including complete blood count, electrolytes and thyroid function, were all normal. Troponin T levels were indeterminate on multiple tests, probably as a result of decreased coronary perfusion during the asystole episodes. Overnight, she had several episodes of the same rhythm of sinus bradycardia, progressing to nodal beats, then prolonged sinus node arrest with the longest lasting about 13 seconds (first strip panel in Fig. 2). The β-blocker she was taking for her hypertension was discontinued. A temporary transvenous pacemaker was inserted. Repeat ECG showed 2 paced beats after the sinus beats (Fig. 3). At a referral hospital, a permanent transvenous single-chamber pacemaker was inserted with VVIR (ventricular pacing, ventricular sensing, inhibition response and rate-adaptive) mode.[1] No further asystole episodes were witnessed; the patient felt well and was discharged home.

The clinical presentation and the ECG findings were suggestive of malignant vasovagal syndrome.[2] The cardioinhibitory form of vasovagal or neurocardiogenic syncope is defined by a ventricular rate during syncope of less than 40 beats/min for longer than 10 seconds or asystole for longer than 3 seconds.[3] Malignant vasovagal syndrome occurs when there is a predominant asystolic–bradycardic component (cardioinhibitory type) in association with injurious and disabling frequent syncope.[3] This is usually diagnosed using the head-up tilt-table test, but the test wasn't required to confirm our patient's diagnosis. In malignant vasovagal syndrome the vagal tone increases during sleep and on bending forward. In our patient, these increases were exaggerated and caused her excessive bradycardia and sinus node arrest, with junctional and ventricular escape suppression.

Often this condition is mistaken for epilepsy, as acute brain hypoxia that occurs during prolonged asystole could trigger seizure-like activities. Conse-

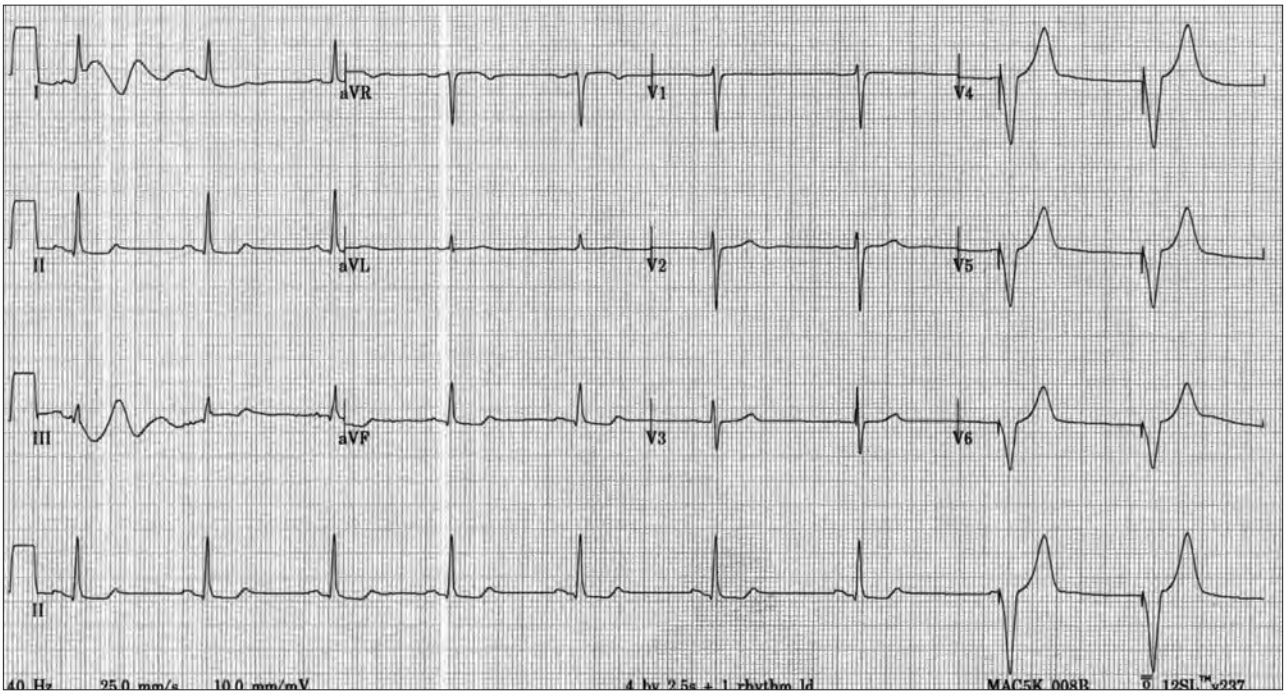

Fig. 3. Twelve-lead electrocardiogram after insertion of a temporary transvenous pacemaker.

quently, patients may be treated inappropriately with antiepileptic medications. The evidence is limited with respect to the management of malignant vasovagal syndrome, and the treatment should be tailored to the condition's severity and the patient's response to treatment.[2–4] The treatment can be in the form of pharmacologic management, such as discontinuing rate-limiting medications, or permanent pacing.[3,5,6] However, most cardiologists still prefer to implant a permanent pacemaker for patients with this syndrome, especially when the sinus node arrest is prolonged, frequent and causes injury,[4] as in our patient. Cardiologists often prefer a pacemaker with a rate-drop response algorithm.[3] Hayes reports that this feature of the pacemaker is

> designed to minimize symptoms of neurocardiogenic syncope. If a patient's heart rate drops at a rapid rate or falls below a specific level (both adjustable parameters), a response is triggered in which the pacemaker will pace the heart at an accelerated rate (e.g., 100 beats/min) for a defined period of time (e.g., two minutes).[1]

This treatment will increase the cardiac output, counter the hypotensive effect of vagal stimulation and minimize the symptoms.

For the question, see page 110.

Competing interests: None declared.

REFERENCES

1. Hayes D. Modes of cardiac pacing: nomenclature and selection. Available: www.uptodate.com/contents/modes-of-cardiac-pacing-nomenclature-and-selection (accessed 2012 Jan. 17).
2. Gan HW, Lim BC, Teo WS. Electrocardiographical case. Young woman with frequent syncopal attacks. *Singapore Med J* 2007;48:1061-3.
3. Parry SW, Kenny RA. The management of vasovagal syncope. *QJM* 1999;92:697-705.
4. Petersen ME, Chamberlain-Webber R, Fitzpatrick AP, et al. Permanent pacing for cardioinhibitory malignant vasovagal syncope. *Br Heart J* 1994;71:274-81.
5. Sutton, R. Vasovagal syndrome — Could it be malignant? *Eur J Cardiac Pacing Electrophysiol* 1992;2:89.
6. Pentousis D, Cooper J P, Cobbe SM. Prolonged asystole induced by head up tilt test. Report of four cases and brief review of the prognostic significance and medical management. *Heart* 1997;77:273-5.