ECG Quiz 49

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
A SPECIAL INTEREST GROUP OF SA HEART ASSOCIATION

ANSWER

OVERVIEW OF THE ECG

The rate is regular and slow – 54bpm, the QRS complexes are wide and the QT interval is prolonged.

MORE DETAILED ANALYSIS OF THE ECG

Before proceeding to a systematic analysis of the ECG, one should consider the clinical context. The details given are limited, but it is clear that the man is not well, having been found semi-conscious. Bradycardia of 54bpm cannot explain a depressed level of consciousness in a young man. He therefore falls into the category of what I have termed "sick bradycardia", by which I mean conditions which cause bradycardia, as opposed to a primary arrhythmia. Neither (c) Sinus bradycardia with right bundle branch block, (d) Long QT syndrome, nor (e) Brugada syndrome would account for his condition.

Causes of "sick bradycardia":

- Hyperkalaemia
- Hypothermia
- Hypothyroidism
- Hypoxia
- Hypercarbia
- Head injury or other intracranial pathology
- Hyperautonomia ("vasovagal")
- Myocardial infarct
- Drugs (e.g. calcium channel blocker + beta blocker)

The rhythm is regular at 54bpm (R-R 1160ms). The QRS is wide (just over 140ms) and is not typical for either right or left bundle branch block, which raises the possibility that it is a ventricular escape rhythm. However, there are rather small P waves preceding each QRS and no sign of any non-conducted P waves. The PR interval is prolonged at around 240ms and is constant, thus excluding AV dissociation.

The wide QRS complexes have a rapid upstroke, small septal Q waves in V5 and V6 with normal R wave progression in the precordial leads, suggesting normal initial ventricular depolarisation and excluding a ventricular origin. It is the slurred terminal part of the QRS which is responsible for the width. Unlike bundle branch block, this deflection is positive in all leads, except aVR and V1. It appears as if tagged on to an otherwise normal QRS. This is a J wave or Osborn wave and, with the added features of sinus bradycardia and a prolonged QT interval, is typical for hypothermia in this clinical context.

The QT interval is considerably prolonged at 650ms, the QTc being 605ms. The T waves are inverted in III and aVF. J waves are not a feature of the drug-induced or congenital long QT syndrome.

While hyperkalaemia causes flattening or disappearance of P waves, and prolongs the PR interval and QRS duration, it causes diffuse slowing of intraventricular conduction (not present in this ECG) and does not usually cause J waves (Figure1). It does not prolong the QT interval.

Acute intracranial pathology, such as subarachnoid haemorrhage, may cause marked bradycardia, QT prolongation and T wave inversion (see SA Heart J 2015;12:88 ECG quiz 38). It does not usually cause QRS widening or J waves.

Drug overdose must be considered. Depending on the drug, there may be bradycardia, heart block, QRS widening and QT prolongation. Drugs which affect intraventricular conduction and repolarisation, such as tricyclics and Class I antiarrhythmics usually prolong both the initial and terminal parts of the QRS and do not cause J waves. Some drugs (e.g. tricyclics) may produce a pattern mimicking the Brugada pattern (Figure 2).

The correct answer is therefore (a): Metabolic derangement – in this case, hypothermia. His temperature was recorded as 29°C. Hypercalcaemia can also cause J waves, but is unlikely to cause a depressed level of consciousness. The prolonged QT interval also precludes this diagnosis.

While those who are aware of the ECG manifestations of hypothermia may have recognised this instantly, it is important nevertheless to analyse the ECG carefully before coming to a definite conclusion. In the Emergency Unit, recognition of these features assists in the making of a rapid diagnosis and choice of appropriate treatment and helps to avoid the reflex response to bradycardia of requesting insertion of a temporary pacemaker.

DISCUSSION

The J wave of hypothermia was described in 1938 by Tomasjewski,[1] and studied by Osborn, with whose name it is now associated, in 1953.[2] J waves are not confined to hypo-

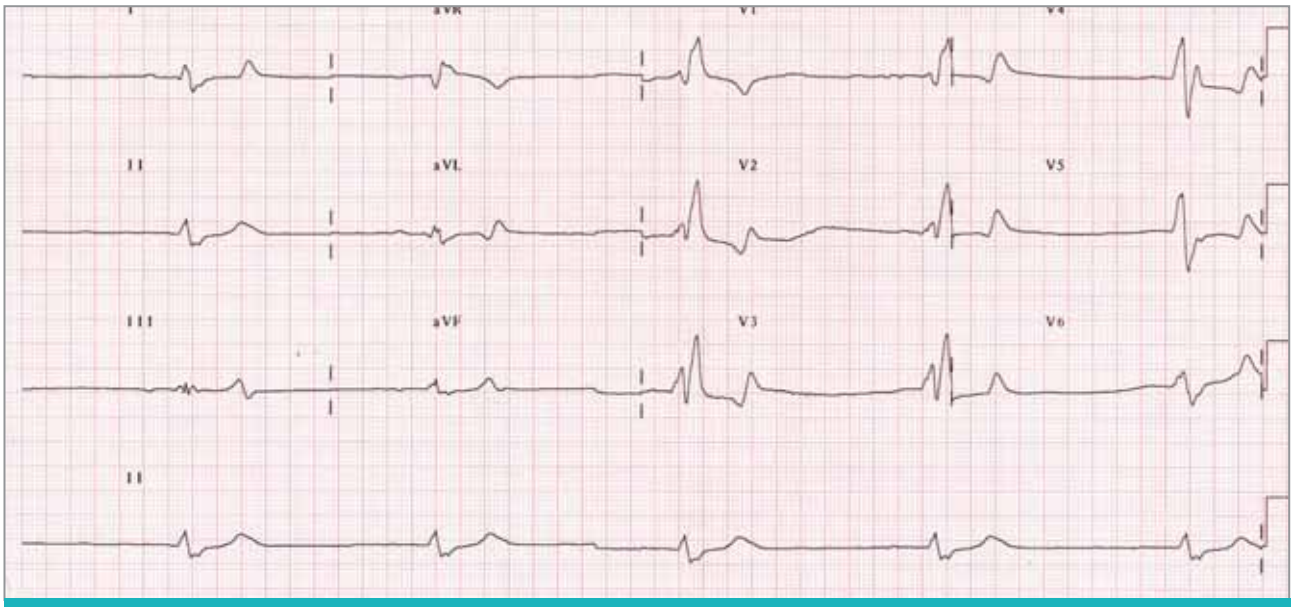

FIGURE 1: ECG from a patient with a serum K+ of 8.8mmol/L, showing small, flat P waves, prolonged PR interval, a wide, diffusely slurred QRS, and peaked T waves.

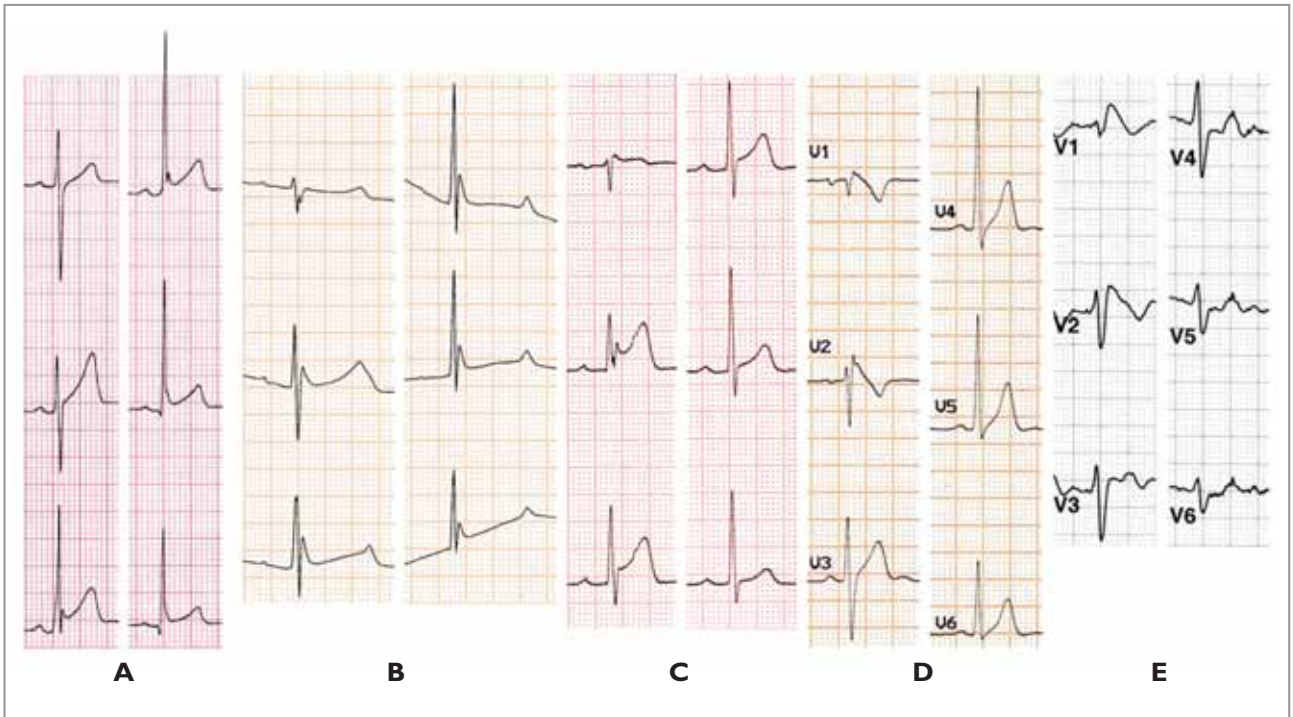

FIGURE 2: Some patterns of early repolarisation (leads V1 to V6)
A: early repolarisation in an otherwise normal, asymptomatic man. B: severe hypothermia (current patient). C: Type II Brugada pattern in an asymptomatic man. D: Type I Brugada pattern. E: pattern resembling Type I Brugada in a young woman with an overdose of a tricyclic.

thermia, but occur in early repolarisation syndromes and may be part of the Brugada pattern[3] (Figure 2).

The mechanism of the J wave has been well studied by Antzelevitch.[4] It is related to a large phase I of the action potential in the epicardial and M cells, but not endocardial cells. This is due to an exaggerated transient outward current (Ito) in the epicardial layers, particularly of the right ventricle (Figure 3). Hypothermia augments this action potential notch and thus increases the transmural voltage gradient and the amplitude of the J wave.[5]

The other ECG features of hypothermia include sinus bradycardia, increased PR and QRS intervals, QT prolongation, junctional rhythms (although shivering artefact may obscure the P waves), atrial fibrillation and ventricular fibrillation.[6] The latter becomes common at temperatures below 25°C, as does asystole.

A number of J wave syndromes are now recognised.[3] Some examples are shown in Figure 2. There are probably common mechanisms underlying the disparate causes. The early repolarisation pattern has long been recognised in otherwise normal young people, with a predominance of males. It is most common in athletes and young black men. More recently, J waves have been associated with otherwise unexplained episodes of ventricular fibrillation, the risk being greatest in those with J waves in the inferior leads, associated with flat or downsloping ST segments. The mechanism is thought to be phase II re-entry, due to dispersion of repolarisation related to different repolarisation times between epicardium and endocardium. While the relative risk is increased, the absolute risk remains small. Interestingly, hypothermia may provoke ventricular fibrillation in patients with the early repolarisation pattern, presumably by further accentuating the dispersion of repolarisation.

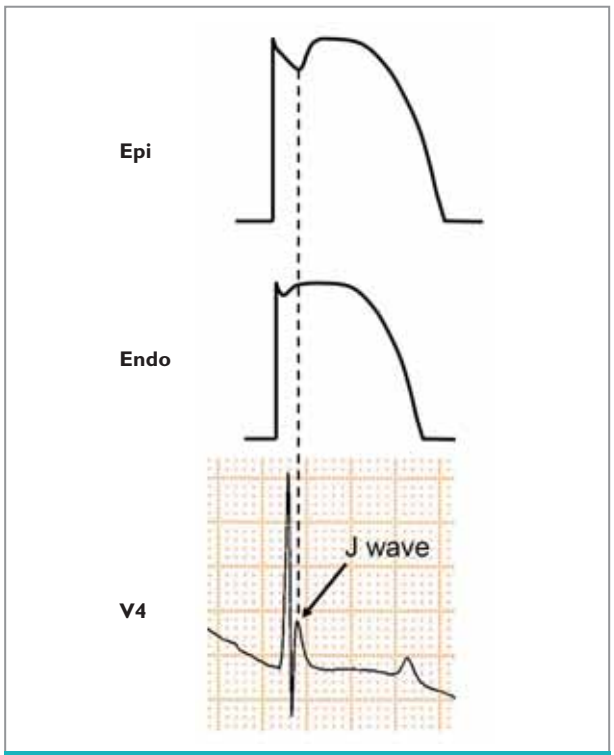

FIGURE 3: J wave resulting from exaggeration of the phase I notch in the action potential of epicardial cells due to hypothermia.

LESSONS AND CONCLUSIONS

- Bradycardia alone is usually well tolerated. If the patient has impaired consciousness, is shocked, or otherwise unwell, consider one of the causes of bradycardia listed above.
- The ECG of hypothermia is characterised by bradycardia, prominent J waves (Osborn waves) and a prolonged QT interval. Atrial and then ventricular fibrillation commonly supervene at lower temperatures.
- J waves occur in a variety of other conditions, including normal people.

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Conflict of interest: none declared.

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