ECG Quiz 47

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

ANSWER

(1) OVERVIEW OF THE ECG

There is a slightly irregular bradycardia at 54bpm with 2 different QRS morphologies.

More detailed analysis of the ECG

Each QRS is preceded by a P wave, but the PR intervals before complexes 2, 6 and 9 are 170ms, whereas those before the other QRS complexes are shorter (125ms). The P waves are all the same and are consistent with a sinus node origin (upright in II, inverted in aVR).

QRS complexes 2, 6 and 9 are narrow (100ms), with a normal rS pattern in V1; the others are wide (140ms) with an abnormal Rs (dominant R wave) pattern in V1. In V6, the narrow QRS is normal with an initial septal q wave and a brisk, normal-sized R wave. In contrast, the wide QRS in V6 has an initial iso-electric component. The R in V4 is slurred and taller than that in the narrow QRS.

The T waves following the narrow complexes are normal, but are inverted in V1 after the wide complexes.

Interpretation

The narrow QRS complexes are normal sinus beats. What, then, is the mechanism of the wide beats?

The pattern in V1, with an initial dominant R wave[1] and terminal S wave is not compatible with right bundle branch block (in which there is a broad terminal R wave). Premature ventricular complexes (PVCs) or an accelerated idioventricular rhythm (IVR) could have this pattern. However, they each follow a constant short PR interval, excluding IVR which should have evidence of AV dissociation. It is unlikely for PVCs to occur repeatedly just before a P can conduct to the ventricles, especially in the presence of bradycardia, although an occasional PVC can interrupt a normal PR.

The pattern of constant short PR and a wide QRS with a slurred upstroke (delta wave) is characteristic of pre-excitation (Wolff-Parkinson-White pattern).

The next question is: why do some of the complexes not have delta waves?

The WPW pattern of a shortened PR interval and wide QRS is a result of the fusion of 2 wave fronts depolarising the ventricles (Figure 1).

The electrical characteristics of the accessory pathway (AP) differ from those of the AV node. The AP conducts faster than the AV node, but its refractory period may be shorter, the same, or longer. In addition, the antegrade and retrograde refractory periods may differ. About 40% of APs are concealed, in that there is no delta wave visible in sinus rhythm, but the pathway may be able to conduct retrogradely and result in orthodromic atrioventricular re-entry tachycardias.

This patient's AP has a long antegrade refractory period, in the region of 1 200ms, such that it only conducts intermittently at slower heart rates and not at all at faster heart rates. On another ECG at a similar heart rate (54bpm) to the one shown, all the QRS complexes were pre-excited, whereas another with a heart rate of 60 was normal.

The correct answer is therefore (b): Sinus rhythm with intermittent pre-excitation (WPW).

(2) MANAGEMENT

The management of a patient with the Wolff-Parkinson-White pattern on the ECG depends on:

- The presence of symptoms (WPW syndrome)
- The potential risk of sudden death

Symptoms from pre-excitation are due to paroxysmal tachycardias (Figure 2). Most commonly, these are orthodromic atrioventricular tachycardias (AVRT) – narrow QRS, regular tachycardias with retrograde P waves, usually visible in the ST segment, just after the QRS complex. The mechanism is macro re-entry, with the AP forming the retrograde limb of the circuit and the AV node the antegrade limb. Rarely, the circuit operates in the opposite direction – antidromic AVRT. In this case, there is a regular wide QRS tachycardia which may be indistinguishable from ventricular tachycardia, because the ventricles are activated exclusively via the AP with slow, cell to cell conduction.

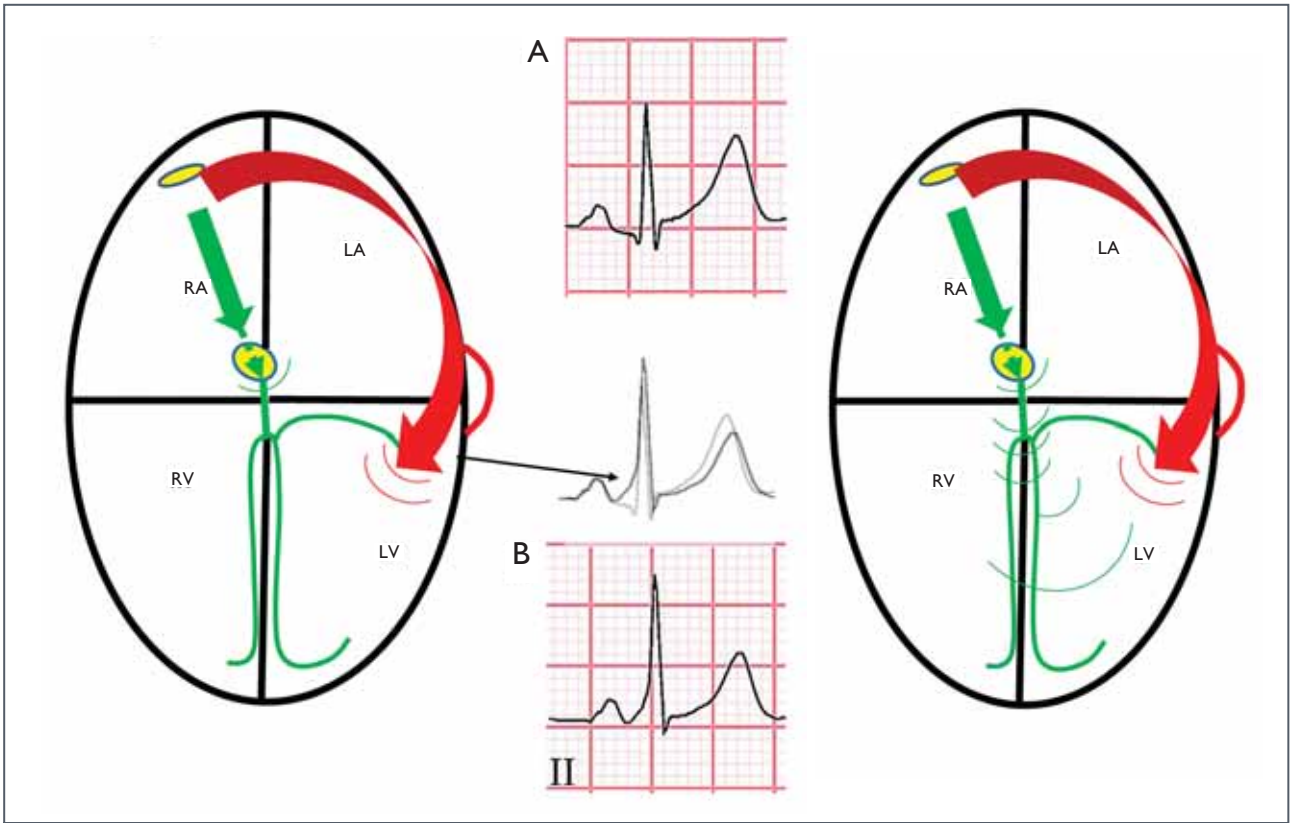

FIGURE 1: Mechanism of the delta wave in WPW pattern.

The diagram on the left shows early activation of the base of the ventricle via the rapidly conducting AP. This impulse conducts slowly through muscle to muscle transmission. The atrial impulse is delayed in the AV node. The right diagram shows rapid His-Purkinje conduction once the impulse traverses the AV node. This rapid activation then depolarises the rest of the ventricles, resulting in a fusion complex.

A. Normal complex from lead II. The PR is 180ms and the QRS duration 80ms. Total time from P wave onset to end of QRS = 260ms.

B. Pre-excited complex from lead II. The PR has shortened to 120ms and the QRS has widened to 140ms. The total time from P wave onset to end of QRS is still 260ms.

The superimposed complexes illustrate the fusion of the delta wave (arrow) with the normally inscribed latter part of the QRS.

AVRT is the most common mechanism of tachycardia in neonates and infants up to the age of one year, but symptoms usually start in late childhood or adolescence. They may, however, start at any age.

The risk of sudden death in WPW syndrome is real but low – 0.25% per year or 3 - 4% over a lifetime.[2] The mechanism is atrial fibrillation (AF) conducting sufficiently rapidly to the ventricles to induce ventricular fibrillation. AF is surprisingly common in patients with WPW syndrome. Around 20% will experience it at some time or another, even children as young as 10 years. It is usually triggered by an episode of AVRT. It is characterised by a very rapid, irregular wide QRS rhythm in which the QRS morphology is not compatible with right or left bundle branch block. The QRS morphology often varies from beat to beat, with occasional narrower complexes when an impulse happens to traverse the AV node ahead of that via the AP.

This patient has no symptoms of palpitations and therefore does not warrant treatment on that score. Is he at risk of sudden death should he develop AF? For the AP to conduct

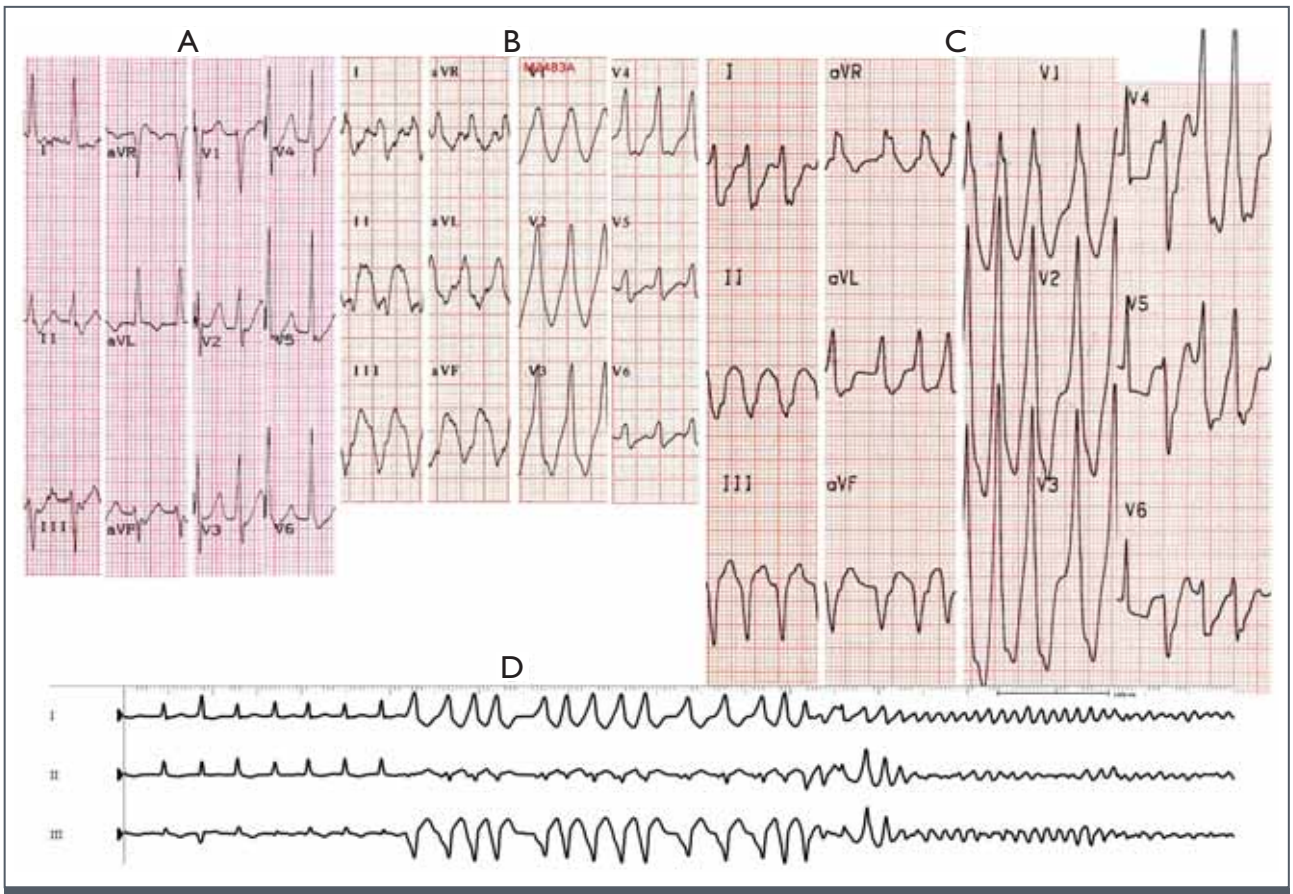

FIGURE 2: Arrhythmias in WPW syndrome (ECGs from different patients).

A. Orthodromic atrioventricular re-entry tachycardia (AVRT), in which the AP forms the retrograde limb of the circuit and the AV node the antegrade limb. A regular, narrow QRS tachycardia. Note the retrograde P waves distorting the ST segments, best seen in SII, about 90ms after the QRS. This is the most common arrhythmia in WPW.

B. Antidromic AVRT, in which the AP forms the antegrade limb, with retrograde activation via the AV node. A regular wide QRS tachycardia with QRS morphology that is not compatible with bundle branch block. Ventricular activation is slow and resembles ventricular tachycardia.

C. Pre-excited atrial fibrillation, in which the ventricles are activated rapidly through an AP with a short refractory period. Note the irregular wide QRS with bizarre morphology. Occasional capture via the AV node results in narrower complexes.

D. Orthodromic AVRT triggers AF with a rapid ventricular response which degenerates after a few seconds into ventricular fibrillation. This is the mechanism of sudden arrhythmic death in WPW syndrome.

sufficient impulses from the fibrillating atrium to induce VF, its refractory period must be short – less than 240ms. The fact that his AP conducts only intermittently at slow heart rates indicates a long refractory period and therefore no risk of inducing ventricular fibrillation.

The answer to (2) is: (a). Tell him that the ECG is benign and no further action is necessary, unless he develops symptoms.

If his ECG had shown consistent pre-excitation and the patient is asymptomatic, there are 2 possible management strategies:[3]

- Observation, without further evaluation or treatment, is reasonable in asymptomatic patients, provided the patient is informed of the small risk of life-threatening arrhythmias.
- Further risk stratification. An exercise stress test is useful if the delta wave disappears at higher heart rates. This, however, is not always easy to determine if there is artefact in the exercise tracing. The ECG should be evaluated carefully to ensure that the delta wave is truly absent as left sided accessory pathways may have subtle and varying degrees of pre-excitation.

Both strategies are reasonable and management will depend on the patient's wishes after balancing a very small lifetime risk of sudden death and a very small immediate ablation risk, the availability of EPS studies and the age of the patient (further risk stratification is preferred in the younger patient).

Should an exercise test be inconclusive or the delta wave persists to maximum heart rate, an invasive electrophysiological study (EPS) should be considered to measure the refractory period of the AP. This should be done with the clear understanding by the patient that it will progress to radiofrequency catheter ablation of the AP, should its refractory period be less than 240ms.

There is now no place for the use of toxic antiarrhythmic drugs in WPW, except in rare instances when catheter ablation fails or is contra-indicated.

LESSONS AND CONCLUSIONS

- The WPW pattern on the ECG is due to fusion of impulses activating the ventricles prematurely via the accessory pathway (delta wave) with that via the AV node.
- Delta waves may be consistent, intermittent or absent (concealed pre-excitation).
- Intermittent delta waves indicate a long refractory period of the AP and therefore very low risk of atrial fibrillation triggering VF.
- In the absence of symptoms, intermittent pre-excitation does not need treatment.

Conflict of interest: none declared.

REFERENCES

1. Millar RS, Chin A. ECG Quiz 42 answer. SA Heart J 2016;13:122-3.
2. Munger TM, et al. A population study of the natural history of Wolff-Parkinson-White syndrome in Olmsted County, Minnesota, 1953-1989. Circulation. 1993;87:866-873.
3. 2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia, JACC 2016;67:e27-115.

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