ECG Quiz 43

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

ANSWER

(1) OVERVIEW OF THE ECG

This is a regular wide QRS tachycardia at 144 bpm (6 x 24). The QRS in V1 is positive, resembling RBBB and there are sharp deflections at the end of the T wave, suggesting atrial activity.

The default diagnosis of a regular wide QRS tachycardia is (a) ventricular tachycardia (VT) (Figure 1), but the differential diagnosis includes the other choices (b) through (e).

Regular	Irregular
Narrow QRS ■ Sinus tachycardia ■ **Atrial flutter** ■ **AV junctional re-entry tachycardia (AVNRT, AVRT)** ■ Atrial tachycardia ■ Junctional ectopic tachycardia	**Narrow QRS** ■ **Atrial Fibrillation** ■ **Atrial flutter/tachy + variable AV block** ■ Multifocal atrial tachycardia
Regular	**Irregular**
Wide QRS ■ **Ventricular tachycardia** ■ SVT with bundle branch block ■ Antidromic AVRT ■ Pre-excited SVT ■ SVT with non-specific intraventricular conduction delay ■ Pacemaker tachycardia	**Wide QRS** ■ **AF with bundle branch block** ■ **A flutter, variable AV block + bundle branch block** ■ Pre-excited AF ■ Polymorphic VT

FIGURE 1: Tachycardia groups.

AVNRT: AV nodal re-entry tachycardia, AVRT: atrioventricular re-entry tachycardia, AV: atrioventricular, SVT: supraventricular tachycardia, AF: atrial fibrillation.

MORE DETAILED ANALYSIS OF THE ECG

The sharp deflections visible at the end of the T waves in V1 are probably atrial depolarisations – they are too high frequency to be part of the T wave. The rhythm strip shows that these P waves are present with each QRS complex i.e. there is no AV dissociation which, if present, would have confirmed the diagnosis of VT. You may be tempted to assume that a 1:1 A to V relationship proves that this is a supraventricular tachycardia, but that is not correct – 1:1 ventriculo-atrial conduction can occur in up to 20% of VTs.[1] The P wave axis will be negative in the inferior limb leads because the atria are depolarised in caudal to cranial direction. The key to diagnosis of regular wide QRS tachycardias lies in the QRS morphology. Figure 2 illustrates the possible mechanisms of wide QRS complexes in tachycardias.

For supraventricular tachycardia (SVT) with bundle branch block to be considered, the QRS morphology must conform either to left or right bundle branch block (Figure 3).

V1 has a dominantly positive QRS, suggesting right bundle branch block (RBBB). Closer inspection reveals a small q wave followed by a broad, slurred R wave – not typical RBBB. More importantly, V6 shows a broad, slowly descending, notched QS wave. There is no evidence of normal left ventricular depolarisation, which is a sine qua non for diagnosis of RBBB. If the right bundle is blocked, the left ventricle must depolarise normally through the intact left bundle branch, otherwise complete heart block would be present. The pattern in V6 indicates slow cell to cell conduction without the benefit of rapid conduction via the His-Purkinje system. This pattern will be seen when the QRS originates in the ventricles (VT) or when ventricular activation occurs via an accessory pathway (Wolff-Parkinson-White or other pre-excitation). If AV dissociation (V>A) had been present, atrioventricular re-entry tachycardia (AVRT) or a pre-excited SVT (WPW) would have been excluded. AVRT requires a 1:1 ventriculo-atrial relationship, as both are part of the circuit and AV conduction, via an accessory pathway, necessitates at least one atrial for every ventricular depolarisation.

In this case, however, the QRS morphology excludes pre-excitation. The QS complexes in V5 and V6 indicate ventricular depolarisation proceeding from the apex towards the base. In WPW syndrome, all accessory pathways insert into the base of one of the ventricles in the vicinity of the AV groove and cannot activate the ventricles from the apex. The QRS morphology is not compatible with right ventricular pacing and pacing spikes are not visible which excludes pacemaker tachycardia (Figure 2).

The correct answer is therefore (a): ventricular tachycardia.

There remains, however, a small possibility that this QRS pattern could result from severely disturbed intraventricular conduction, as may occur with severe diffuse left ventricular damage.[2] This is called a non-specific intraventricular conduction delay.

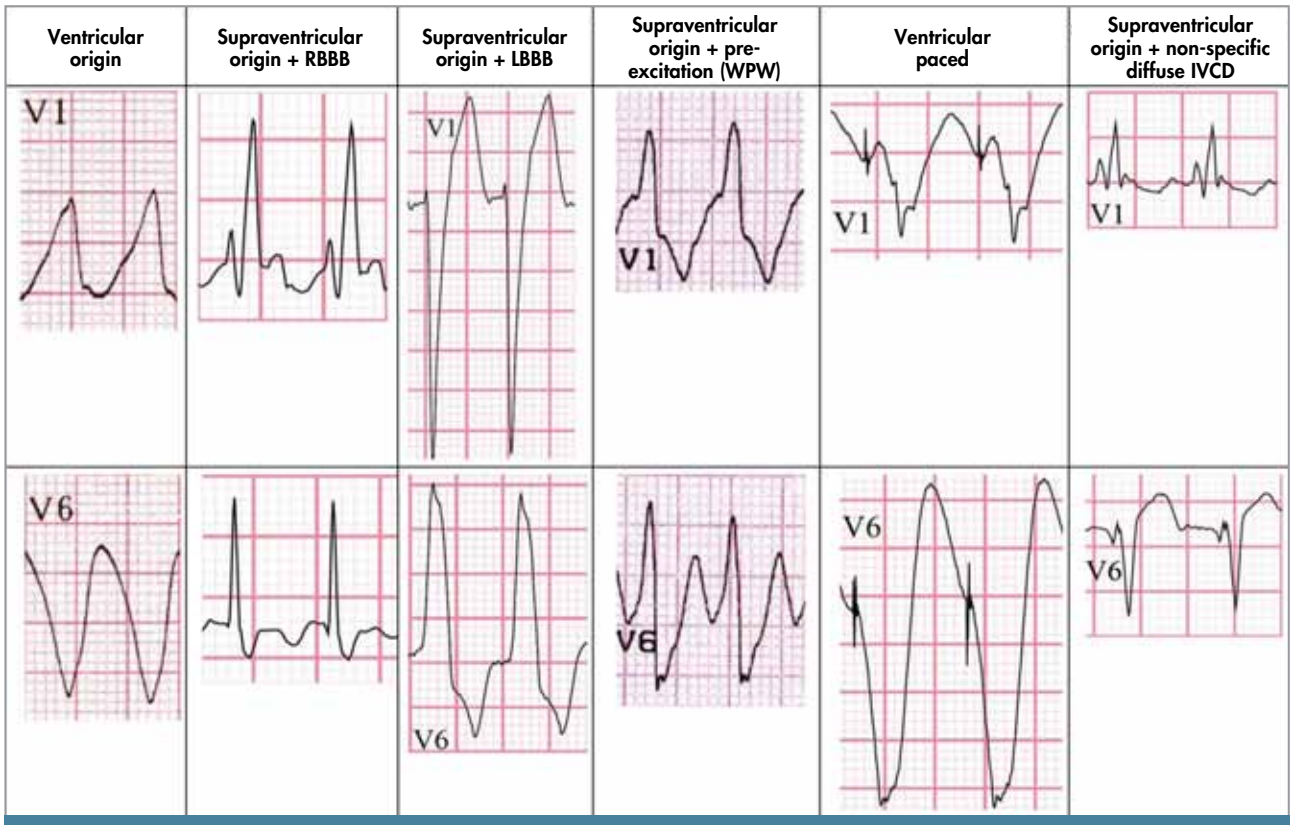

FIGURE 2: Mechanisms of wide QRS in tachycardias.

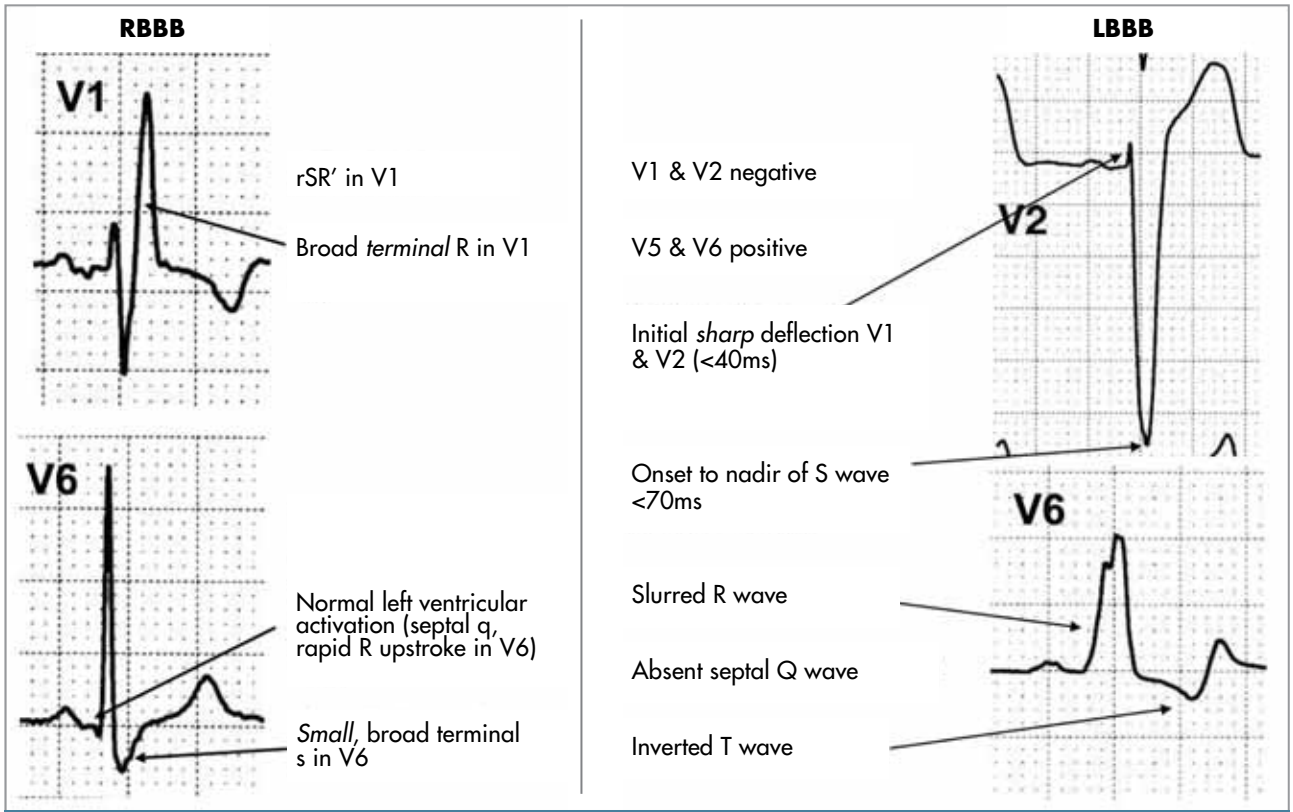

FIGURE 3: Patterns of right and left bundle branch block.

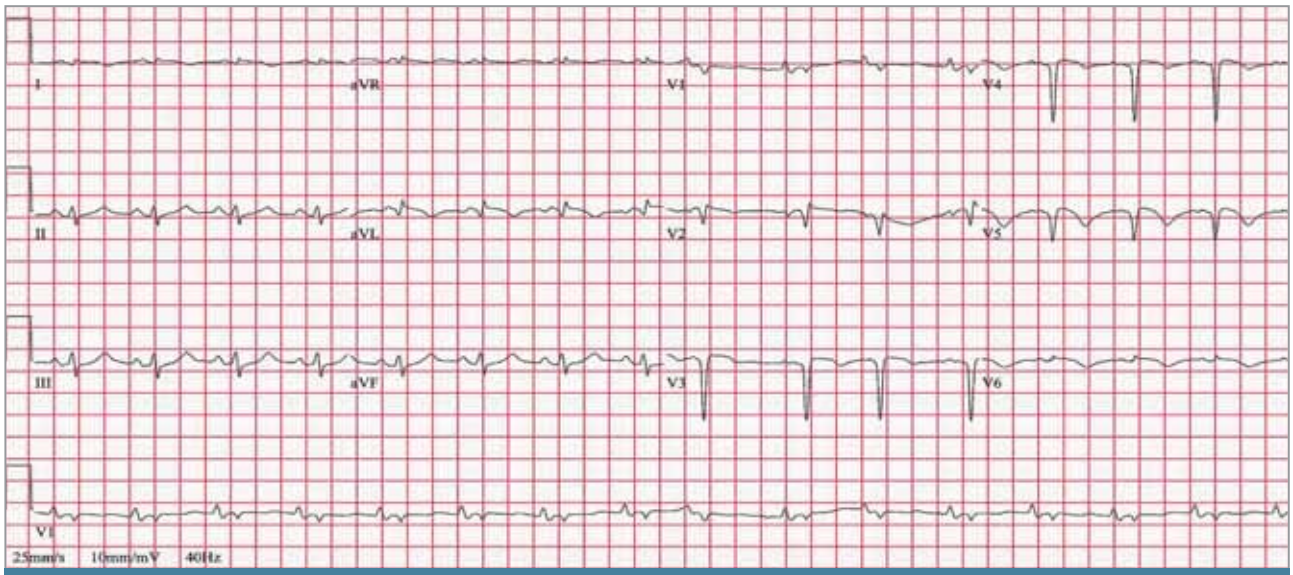

FIGURE 4: ECG after cardioversion.
This shows an antero-lateral myocardial infarction of indeterminate age. The QRS complexes are narrow.

(2) WHAT WOULD ASSIST WITH THE DIAGNOSIS?

Adenosine is best avoided in wide QRS tachycardias unless there is compelling evidence for a supraventricular origin. It will not terminate scar re-entry VT. Following the initial transient AV nodal blockade, there tends to be a surge of sympathetic activity in response to the peripheral vasodilatation and the discomfort accompanying adenosine's effect. This could accelerate the VT or precipitate ventricular fibrillation.

Vagal stimulation can be tried if SVT is strongly suspected, but would be ineffective in this case, although it might induce transient retrograde AV block or AV dissociation and hence confirm VT.

While IV amiodarone is a popular choice for emergency treatment of wide QRS tachycardias, its efficacy is poor (less than 30% of VTs will terminate with amiodarone)[3] and it may cause significant haemodynamic deterioration. Synchronised DC cardioversion is safe and close to 100% effective in terminating VT and most SVTs.

A simple question should be asked: "have you ever had a heart attack?" If the answer is "yes" (as was the case with this patient), it raises the probability of VT from around 85% to more than 95%.[4] The most common substrate for sustained monomorphic VT is endocardial scar from healed myocardial infarction.

An ECG in sinus rhythm after cardioversion will help to confirm the diagnosis of VT, by excluding non-specific intraventricular conduction delay (Figure 4) and confirming old myocardial infarction.

The answer to (2) is (b) a clinical history, and/or (c), an ECG in sinus rhythm.

LESSONS AND CONCLUSIONS

- Ventricular tachycardia is the default diagnosis for a regular wide QRS tachycardia.
- A 1:1 relationship between atrium and ventricle is common in VT and does not prove SVT.
- QRS morphology is the key to diagnosis of wide QRS rhythms.
- AV dissociation (V>A) excludes a pre-excited tachycardia.
- The presence of QS or Qr complexes in V5-6 is diagnostic of VT.

REFERENCES

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4. Tchou P, et al. Useful clinical criteria for the diagnosis of ventricular tachycardia. Am J Med 1988;84:53-6.

Conflict of interest: none declared.

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