ECG Quiz 53

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

ANSWER

(1) OVERVIEW OF THE ECG

This is a wide QRS tachycardia at138bpm (23x6). It is a complex ECG, because there are at least 3 different QRS patterns. The first 6½ seconds of the tracing consists of a regular wide QRS tachycardia at about 145 bpm. This is terminated by a ventricular complex (Figure 2 - arrow). It is followed by an irregular wide QRS tachycardia at 114bpm.

Figure 1 shows the differential diagnosis of tachycardias, based on QRS width and regularity. The default diagnosis for a regular wide QRS tachycardia is ventricular tachycardia (VT), whereas an irregular wide QRS tachycardia is more likely to be atrial fibrillation (AF) with bundle branch block or pre-excitation. This rhythm is regular to start with and then irregular for the last few seconds.

MORE DETAILED ANALYSIS OF THE ECG

The key to differentiating the mechanisms of wide QRS rhythms is the detailed morphology of the QRS 1, 2 using the leads V1 and V6. With a complex ECG such as this, each different QRS pattern needs to be analysed separately.

The first 16 complexes are regular, about 145bpm. They are at least 160ms wide. V1 and V2 have dominant R waves, suggesting possible right bundle branch block (RBBB). However, unlike RBBB, it is the initial R waves that are broad and slurred. This slow initial activation is not compatible with RBBB, but suggests a ventricular origin or pre-excitation.

The next complex also has an initial, slurred dominant R in V1, but a different pattern. This is most likely a ventricular complex, the effect of which is to terminate the regular wide QRS tachycardia. This complex either arises from a different site or follows a different pathway. It renders part of the tachycardia circuit refractory – thus terminating the tachycardia. This can happen with either VT or antidromic atrioventricular re-entry tachycardia (AVRT).

Following termination of the regular wide QRS rhythm, the QRS changes to a pattern that is compatible with RBBB, albeit with an initial Q wave in V1. The delay is now in the terminal part of the QRS. Although the S wave in V6 is bigger than the R wave, the upstroke of the R is relatively brisk, suggesting rapid left ventricular depolarisation – as one would expect for RBBB. This, together with the slower rate and random irregularity, suggests

atrial fibrillation with RBBB. The longer R-R interval after termination of the regular rhythm shows rapid, irregular atrial activity compatible with AF.

While the QRS morphology of the regular rhythm does not exclude antidromic AVRT, the transition from a positive complex in V2 to a negative with initial Q in V3 suggests VT. If AV dissociation were present, antidromic AVRT would be excluded and VT confirmed. There are irregularities of the baseline between the regular QRS complexes (best seen in V1) that suggest AF, confirmed during the pause following the tachycardia termination. Antidromic AVRT cannot occur while the atria are fibrillating; the lack of irregularity despite AF implies AV dissociation, so confirming VT. The slight variation in QRS morphology seen in V1 during the regular tachycardia can be explained by subtle degrees of fusion of the conducted beats and VT – another feature of AV dissociation.

The clinical context, suggesting myocardial infarction (MI), strongly supports a diagnosis of monomorphic ventricular tachycardia. After the VT stops, AF with RBBB is revealed. In addition, the small initial q in V1 and the deep, wide Q in V4 indicate infarction. The raised ST segments in V4 and V5 suggest that it is recent. Note also the raised ST in V3 during VT. The Q waves during VT cannot be used to diagnose MI, but ST segment elevation tends to persist in the affected leads, even during ventricular tachycardia or paced rhythms.

The correct answer is therefore (c): Monomorphic VT + atrial fibrillation; recent anterior myocardial infarct

Sustained monomorphic VT is unusual so early in the course of acute myocardial infarction, as the substrate is the subendocardial scar of a healed infarct. The mechanism is re-entry, related to functionally abnormal muscle trapped within the scar. It is likely that this patient had a pre-existing scar.

(2) CONFIRMING THE DIAGNOSIS

Carotid sinus massage may temporarily slow the ventricular response to the AF, but would not contribute to the diagnosis of the regular wide QRS tachycardia.

Adenosine, likewise, would not help. Adenosine may precipitate VT or VF because of the acute sympathetic activation and is best avoided in patients with wide complex tachycardia.

A signal averaged ECG is a filtered, high magnification ECG recording used to detect late potentials. These may give a clue to the presence of a substrate for VT (scar delaying conduction), but would not be helpful here. The wide QRS of the conducted rhythm precludes the detection of late potentials.

An ECG repeated after termination of VT will confirm the AF (Figure 2) with random irregularity and visible fibrillatory waves,

and help to distinguish it from the regular VT – thereby confirming that diagnosis because the lack of irregularity implies AV dissociation.

In this case, an ECG had been done shortly before the one shown (Figure 1). This showed regular sustained monomorphic VT, with QRS morphology clearly different from the conducted AF (Figure 3). The pattern of the QRS complexes is diagnostic

REGULAR	IRREGULAR
Narrow QRS ■ Sinus tachycardia ■ **Atrial flutter** ■ **AVJRT (AVNRT, AVRT)** ■ Atrial tachycardia ■ Junctional ectopic tachycardia	**Narrow QRS** ■ **Atrial Fibrillation (AF)** ■ **Atrial flutter/tachy + variable AV block** ■ Multifocal atrial tachycardia
Wide QRS ■ **Ventricular tachycardia** ■ SVT with BBB ■ SVT with non-specific intraventricular conduction delay ■ Paced rhythm ■ Antidromic AVRT ■ Pre-excited SVT	**Wide QRS** ■ **AF with bundle branch block** ■ **A flutter/tachy + variable AVB + BBB** ■ Pre-excited AF ■ Polymorphic VT

FIGURE 1: Differential diagnosis of tachycardias, based on regularity and QRS width.
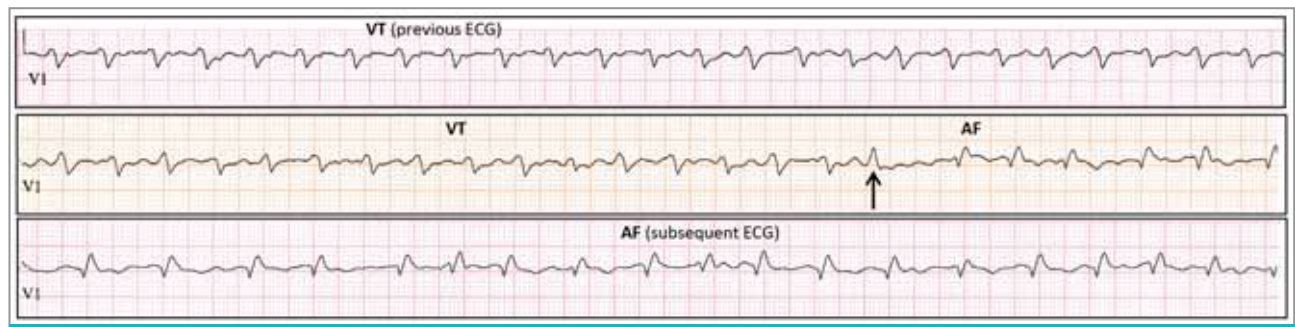
FIGURE 2: V1 rhythm strips from a previous ECG (top), during sustained monomorphic VT; middle: transition from VT to AF, triggered by a change in QRS morphology (arrow); bottom: a subsequent ECG in AF with right bundle branch block.
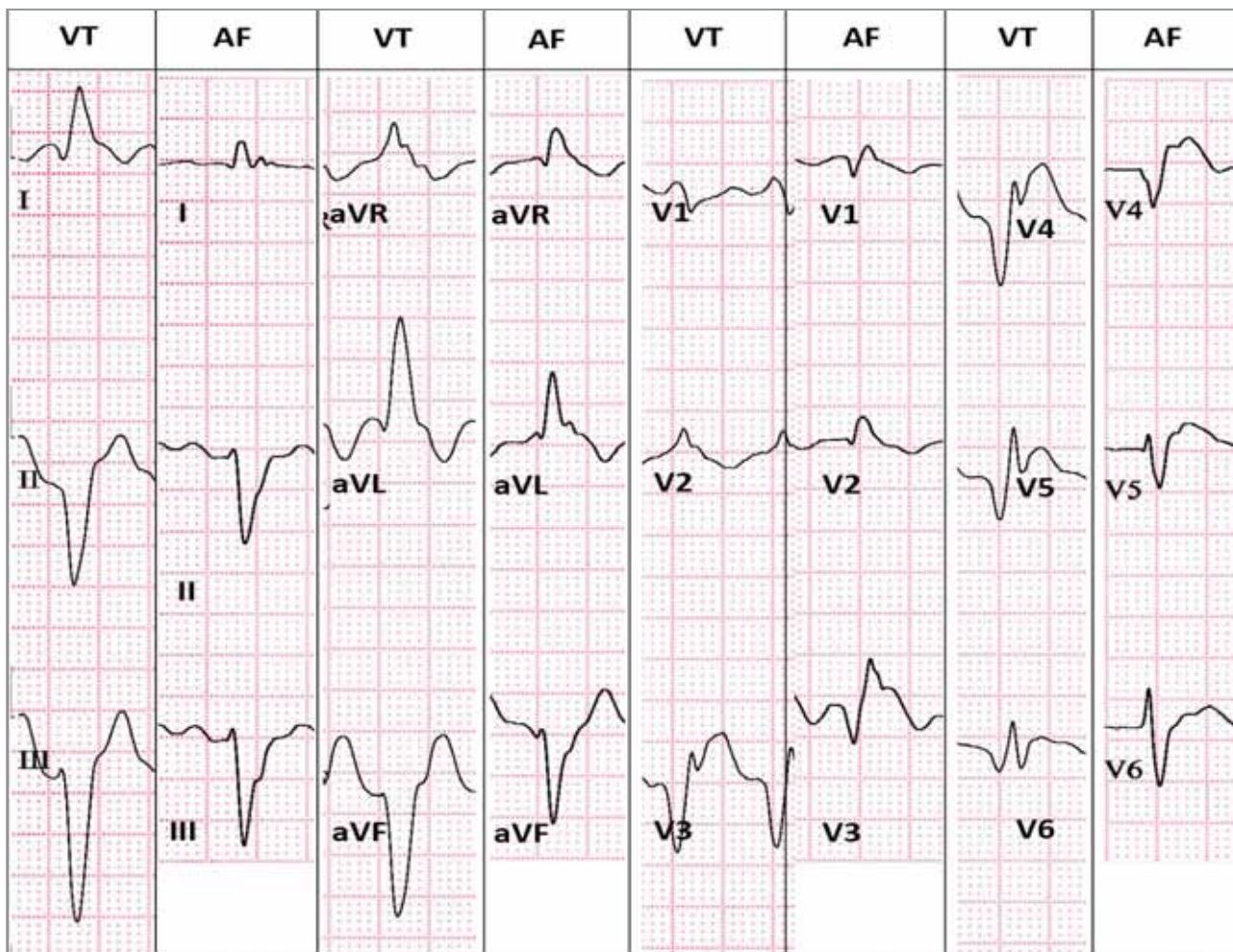
FIGURE 3: Shows the differing QRS patterns in ventricular tachycardia and atrial fibrillation. Note, particularly, V1: initial slow R in VT, initial Q and terminal R in AF, and V6: broad Q in VT vs brisk initial R in AF with RBBB.

of VT. Bundle branch block is excluded by the slow initial depolarisation in all leads, and the deep Qs in V4 - 6 is pathognomonic of VT, indicating depolarisation of the left ventricle from apex to base.

The answer to (B) is c. another ECG.

LESSONS AND CONCLUSIONS

■ Measurement of QRS width and regularity allows tachycardias to be divided into 4 possible categories (Figure 1) – each of which has a differential diagnosis and different probabilities.

■ Occasionally, 2 tachycardia mechanisms can exist simultaneously.

■ Ventricular tachycardia is the default diagnosis for a regular wide QRS tachycardia, whereas atrial fibrillation with bundle branch block or pre-excitation is more likely if the tachycardia is randomly irregular.

■ QRS morphology is the key to the diagnosis of wide QRS tachycardias.

REFERENCES

1. Scott Millar R, Chin A. SA Heart J 2016;13(3):311 ECG Quiz 43, answer.
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Conflict of interest: none declared.