ECG Quiz 31

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

ANSWER

(1) OVERVIEW OF THE ECG

There is a regular wide QRS tachycardia at a rate of 180/minute. The QRS complexes in the chest leads resemble left bundle branch block (LBBB). The default diagnosis, even in a teenager, is ventricular tachycardia. The differential diagnosis of regular wide QRS tachycardias is given in the Table below.

TABLE 1: Mechanisms of regular wide QRS tachycardias
Ventricular origin
Ventricular tachycardia
Ventricular paced rhythm
Supraventricular origin with bundle branch block aberration
LBBB
RBBB
Supraventricular origin with pre-excitation
Atrial flutter or tachycardia conducted via accessory pathway
Antidromic atrioventricular tachycardia (WPW)
Atriofascicular tachycardia (Mahaim)
Supraventricular origin with non-specific intraventricular conduction delay
Extensive ventricular damage
Drugs

MORE DETAILED ANALYSIS OF THE ECG

The key to diagnosing the origin of wide QRS tachycardias lies in a detailed analysis of the QRS complexes. P-waves are very often not visible and, even if they are, can be misleading. Measurement of QRS duration is not easy, but it is critical. Choose the widest QRS, checking the onset and offset in simultaneous leads. V2 - V5 are usually the widest. In this case the complexes are 150 - 160ms wide, clearly abnormal.

The LBBB pattern is compatible with a number of the above mechanisms. To distinguish true LBBB from similar-looking complexes, look carefully at the initial depolarisation in V1 and V2 (Figure 1). The initial small r-wave in these leads in LBBB is due to right ventricular depolarisation which is unaffected and should be rapid. This R-wave should be 30ms or less and the onset of the QRS to the nadir of the S-wave should be less than 60ms.[1] In this

case, the R is at least 50ms and the QRS onset to S nadir is at least 80ms. Any form of supraventricular tachycardia with LBBB is therefore excluded. Extensive non-specific ventricular delay associated with widespread left ventricular damage may result in delay throughout the QRS, but is highly unlikely in a 14-year-old boy who is able to play soccer.

This leaves ventricular tachycardia or pre-excited tachycardia as possibilities.

Both idiopathic right ventricular outflow tract tachycardia and VT due to Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) may be induced by exercise, whereas accessory pathway related tachycardias are usually not.

The left axis (-60°) and pseudo LBBB is compatible with antidromic tachycardia (c), or the rare Mahaim tachycardia which involves an accessory pathway with decremental properties connecting the right atrium to the terminal part of the right bundle branch. It excludes idiopathic right ventricular outflow tract tachycardia which
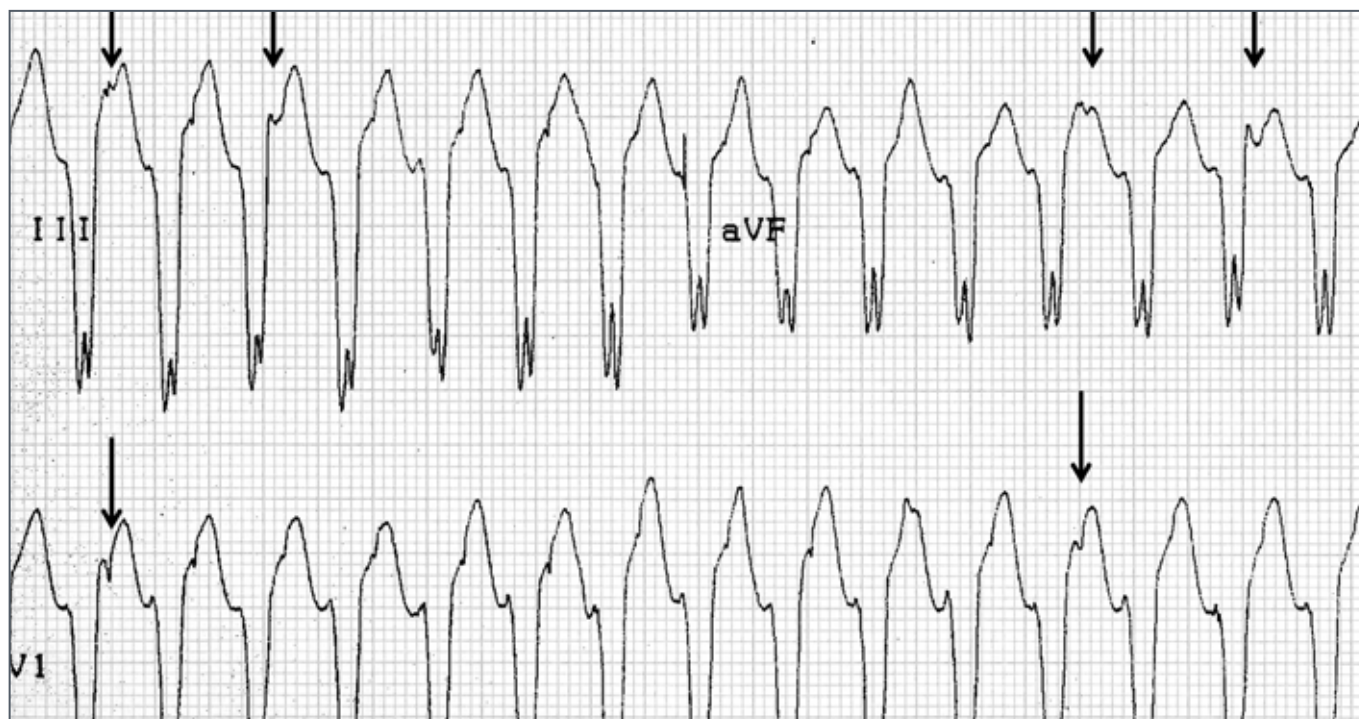

FIGURE 1: Distinguish true LBBB from similar-looking complexes
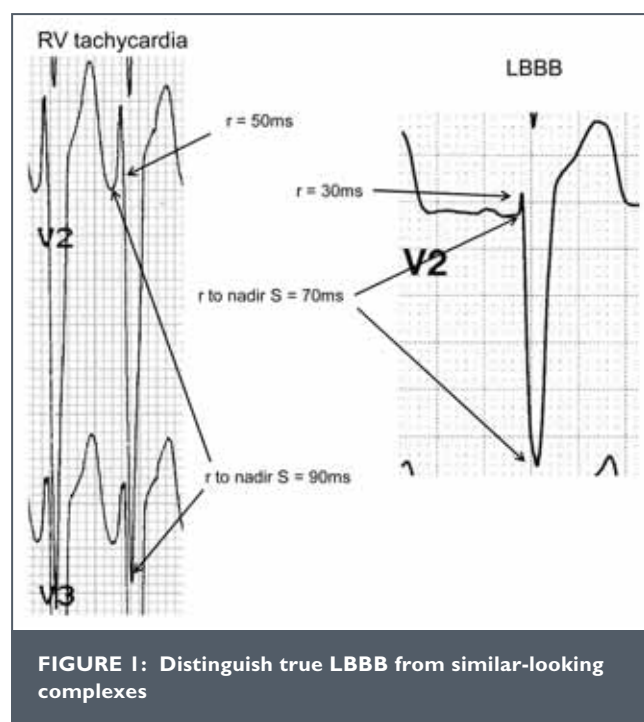
FIGURE 2: P-waves distorting the ST segments in SII

has an axis between +60 and +90°, because of RV depolarisation proceeding downwards.

At this juncture, it is worth searching for P-waves. A 1:1 VA relationship would not be helpful in the differentiation. It is obligatory in antidromic AVRT and classical Mahaim tachycardia, but is common in VT. Fortunately, there is clear evidence of AV dissociation in this tracing. Figure 2 indicates the P-waves distorting the ST segments in SII.

They can also be seen at times in the V1 rhythm strip. The P - P interval is just under 600ms, suggesting sinus tachycardia which would be appropriate. While it could be argued that the AV dissociation could have been the first observation made, thus clinching a diagnosis of VT, this is an unreliable way to approach the problem, given the frequency of 1:1 VA relationships in VT and the difficulty in being certain of the identity of P-waves, particularly when the tachycardia is very fast.

The correct answer to Question 1 is therefore (a): **Sustained monomorphic ventricular tachycardia.**

The VT originates in the right ventricle. Since it is clearly not idiopathic RV outflow tract VT, what are the possible causes? The most common is ARVC, a familial cardiomyopathy characterised by replacement of RV myocytes by fibrous and fatty tissue.[2] Other possibilities include infiltrative and granulomatous conditions, such as sarcoidosis and Wegener's granulomatosis.

(2) HELP WITH DIAGNOSIS

Carotid sinus massage (CSM) will not help, unless the above assessment is incorrect. If there had been a 1:1 relationship between atria and ventricles, then CSM could be useful. If the tachycardia terminated, it would indicate either antidromic AVRT or Mahaim tachycardia. In VT with 1:1 VA conduction, CSM may induce retrograde AV block, either partial or complete (AV dissociation). It is important to run a good quality ECG with 3 or more channels if one hopes to detect this.
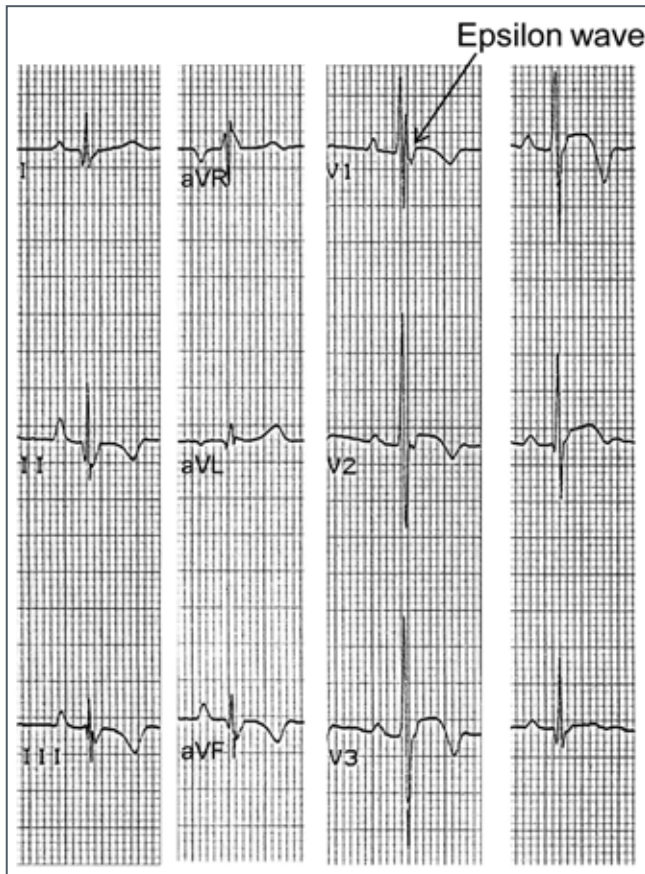
FIGURE 3: An ECG in sinus rhythm

Vector Magnitude
1000mm/mV (1mm=1uV)
200mm/sec (1mm=5ms)
QRS duration = 123ms
Duration <40mV = 51ms
Last 40ms = 15µA

FIGURE 4: The presence of abnormal late potentials strongly suggests ARVC

An ECG in sinus rhythm was diagnostic in this case (Figure 3). It is clearly abnormal, showing a slightly wide QRS (110ms) with an RSRs pattern in V1, and some ST elevation with T-wave inversion in V1 - 4. The delay and splintering of the terminal QRS complex is not typical for RBBB but is an epsilon wave, characteristic of ARVC.[3,4] Not all patients with ARVC have such an abnormal sinus rhythm ECG, however.

Adenosine should be avoided in wide QRS tachycardias, unless there is a strong likelihood of SVT with typical right or left bundle branch block and vagal manoeuvres have failed. The catecholamine surge following adenosine may precipitate VF in a patient with VT and may convert antidromic AVRT to pre-excited atrial fibrillation.

A **signal averaged ECG**, looking for late potentials can be useful. It is particularly helpful in a right ventricular tachycardia with a normal or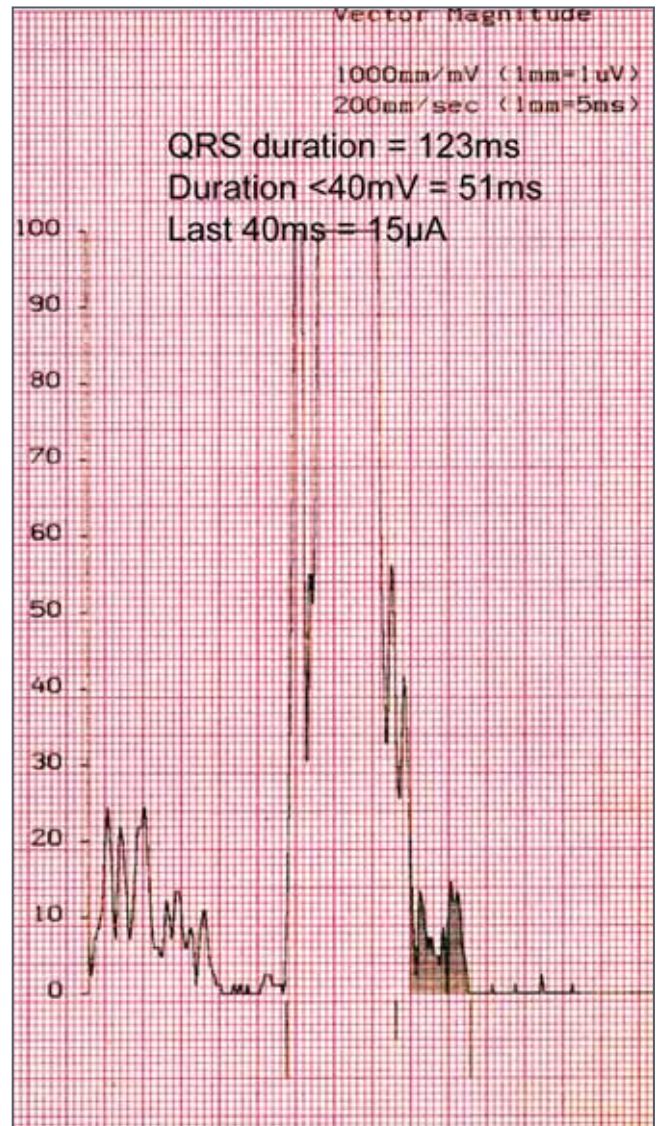 rightward axis, compatible with an origin in the RV outflow tract and a non-diagnostic ECG in sinus rhythm. The presence of abnormal late potentials strongly suggests ARVC (Figure 4).

The answer to Question 2 is **b or d or both**.

Additional investigations may be required to confirm a diagnosis of ARVC, including echocardiogram, MRI, right ventricular angiogram and endocardial biopsy.[2]

LESSONS AND CONCLUSIONS

- Regular, wide QRS tachycardia is usually VT, even in young people.

- QRS morphology is the key to differentiating VT from SVT with bundle branch block.

- P-waves may help to distinguish VT from pre-excited tachy-cardias.

- VT from the right ventricle (pseudo LBBB morphology) requires investigation for ARVC.

SOUTH AFRICAN ARVC REGISTRY

A South African registry of proven cases of ARVC (currently 110), under the auspices of CASSA, is co-ordinated at the University of Cape Town.[5] Please consider sending details of suspected or confirmed cases to: Dr Ashley Chin and/or Prof B Mayosi at arvc@uct.ac.za.

REFERENCES

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Conflict of interest: none declared.