# ECG Quiz 63

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
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ANSWER

## OVERVIEW OF THE ECG

This is a regular, wide QRS tachycardia at 108bpm with a left bundle branch-like pattern. Although not very fast, the patient was aware of and bothered by the tachycardia.

## MORE DETAILED ANALYSIS OF THE ECG

The QRS width is 160ms; the complexes are negative in the mid-chest leads and positive in the lateral leads. The R wave in V1 is less than 40ms and the time from the onset of the QRS to the nadir of the S wave is 50ms. This is compatible with typical left bundle branch block. There is normal R wave progression, but tiny Q waves in V5 - V6. The QRS axis is -60°.

There appear to be P waves at the end of the T waves, best seen in V1 as positive deflections. The P wave axis is +60°. The PR interval is 240ms. The RP interval (beginning of QRS to beginning of P) is 310ms. This is therefore a long RP tachycardia i.e. the P wave is more than half-way between the preceding and succeeding QRS. The ST segments and T waves are compatible with the changes secondary to the wide QRS. The QT interval is difficult to measure because the P waves distort the end of the T waves.

## INTERPRETATION

Despite the little Q waves in V5 - V6, the QRS morphology is compatible with LBBB (Figure 1). The rapid initial depolarisation in the right chest leads suggests normal right ventricular activation and renders ventricular tachycardia unlikely. An approach to a regular, wide complex tachycardia is shown in Figure 2.

The normal P wave axis and the relatively slow rate make sinus tachycardia a possibility. However, the patient's symptoms are against this, as is the apparent prolonged PR interval. Sinus tachycardia should only be entertained in the context of an appropriate clinical reason. In addition, sinus tachycardia usually results in a short PR interval because of the effects of catecholamines on AV nodal conduction.

The long RP interval and positive P wave axis excludes typical AV nodal re-entry tachycardia, in which the RP is typically <70 - 90ms with negative P waves in the inferior leads (Figure 3). Atriofascicular (Mahaim) tachycardia involves a decrementally conducting accessory pathway which usually inserts into the distal part of the right bundle branch. It only functions antegradely during reciprocating tachycardia which results in a pattern of left axis deviation (LAD) and LBBB (Figure 4). While there is LAD and LBBB in this ECG, the visible P waves are upright in lead II, which excludes retrograde atrial activation.

We are therefore left with atrial flutter with LBBB. This diagnosis would be difficult to entertain if the ECG is viewed in isolation. An atrial tachycardia would be a strong possibility but was not included in the options given. However, in the clinical context of a man who has had a mitral valve replacement, atrial flutter becomes much more likely. The wide QRS may be concealing part of the visible flutter wave and completely con-

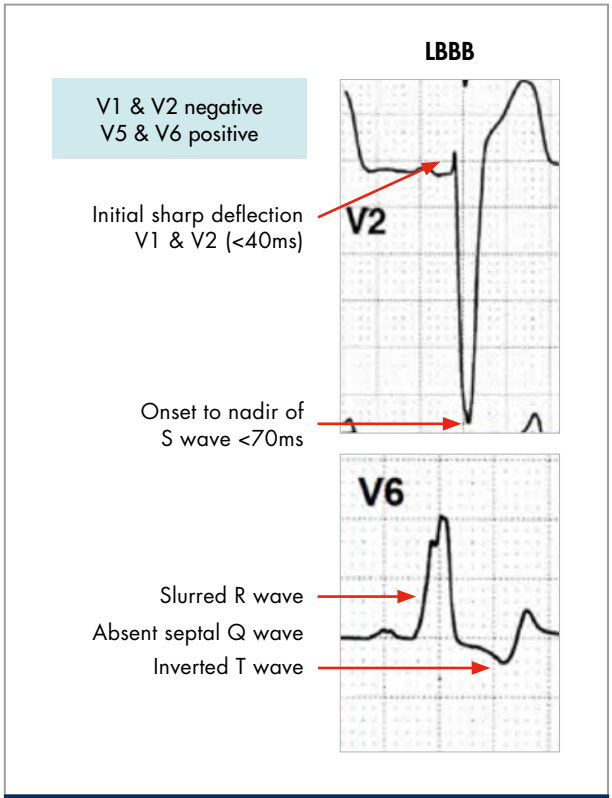


**FIGURE 1: Typical left bundle branch block.** V1 - V2 are the critical leads for diagnosis of LBBB which must conform to the measurements shown. V5 - V6 are much more variable and can be mimicked by other conditions. Even in true LBBB, they may not resemble the typical "M" complex.

ceal the blocked one. Assuming 2:1 AV conduction, the flutter rate is only 216/minute which is slower than usual for flutter (240 - 360). In the presence of long-standing mitral valve disease, the right atrium is likely to be large because of tricuspid regurgitation which will result in a longer flutter circuit and slower rate.

**The correct answer to question 1 is therefore (a): Atrial flutter with left bundle branch block.**

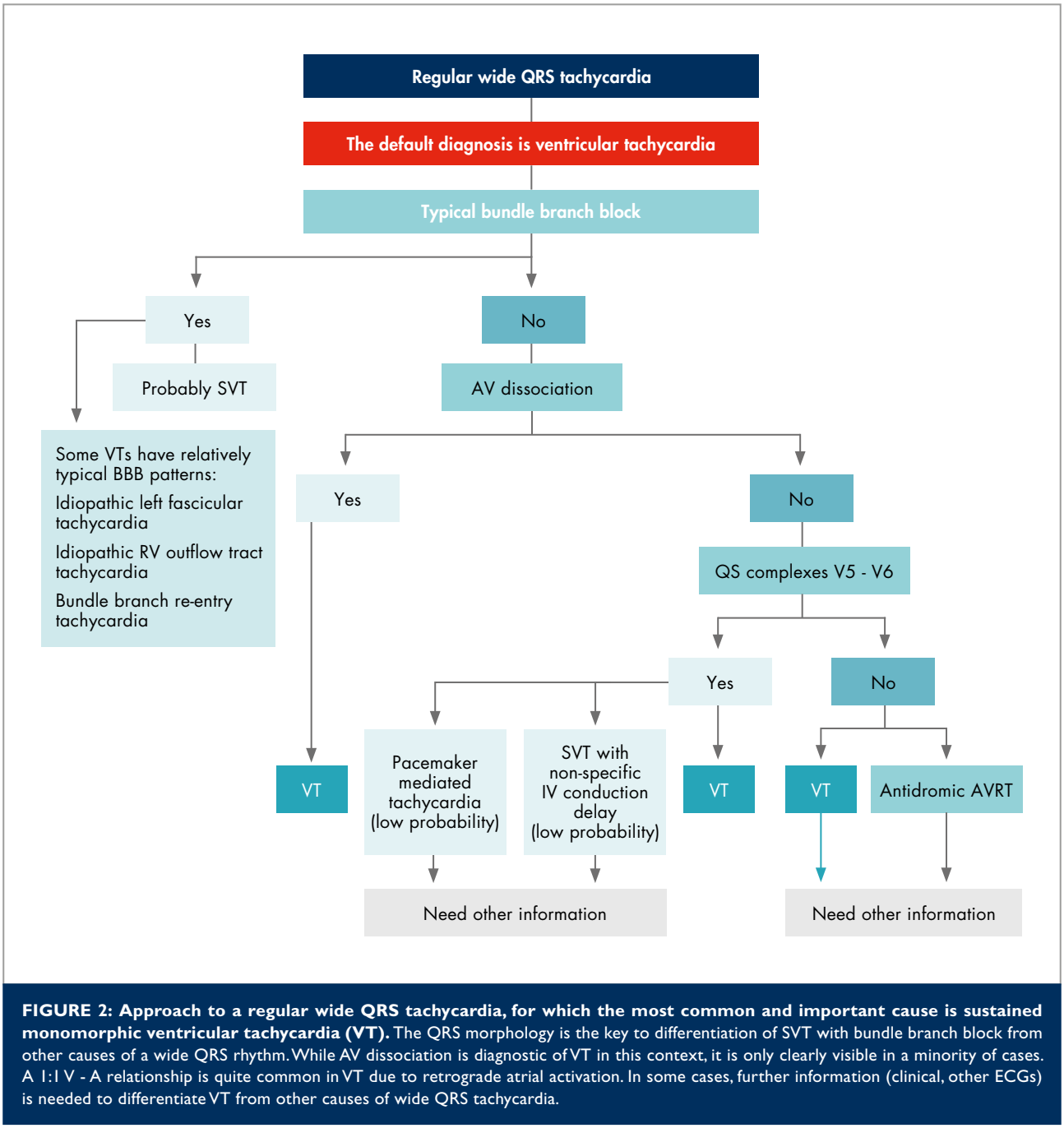


**FIGURE 2: Approach to a regular wide QRS tachycardia, for which the most common and important cause is sustained monomorphic ventricular tachycardia (VT).** The QRS morphology is the key to differentiation of SVT with bundle branch block from other causes of a wide QRS rhythm. While AV dissociation is diagnostic of VT in this context, it is only clearly visible in a minority of cases. A 1:1 V - A relationship is quite common in VT due to retrograde atrial activation. In some cases, further information (clinical, other ECGs) is needed to differentiate VT from other causes of wide QRS tachycardia.

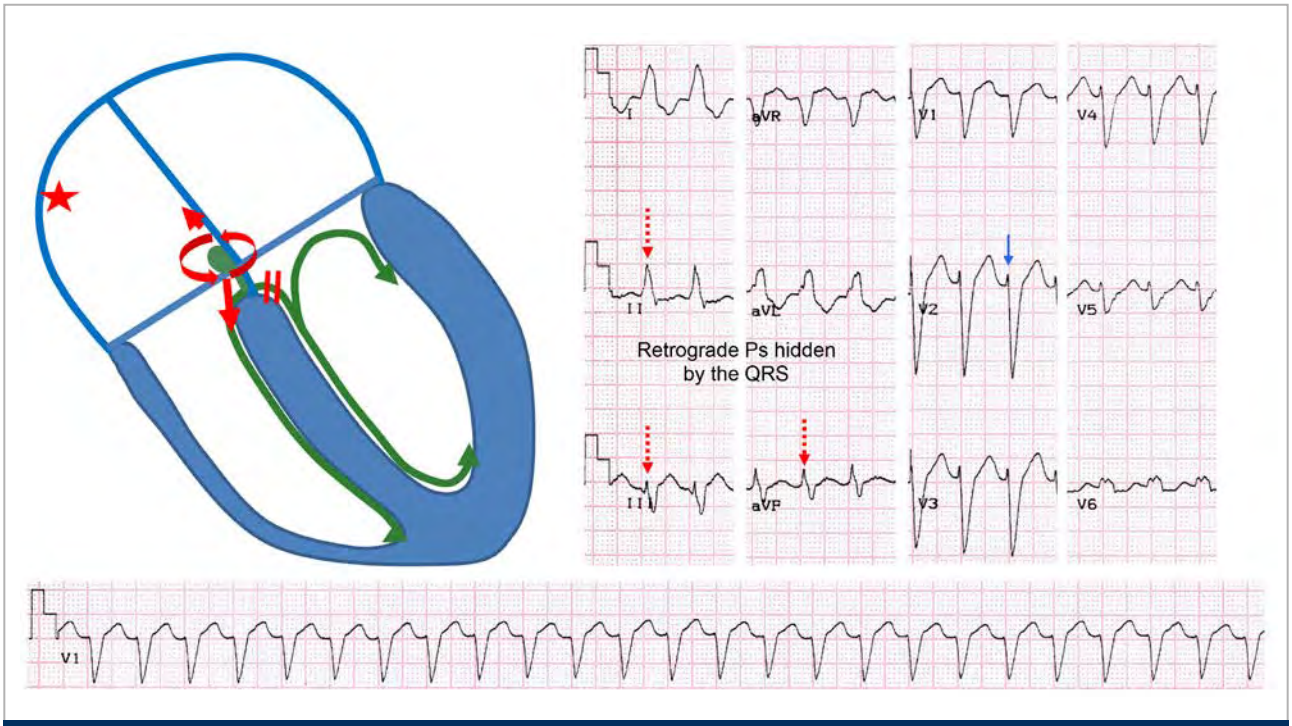


**FIGURE 3: Typical AV nodal re-entry tachycardia with LBBB, in which the slow pathway forms the antegrade and the fast pathway the retrograde limb.** The central position of the re-entry circuit results in almost simultaneous activation of the atria and ventricles. When the QRS is narrow, the retrograde P may be seen distorting the end of the QRS, whereas it is hidden within the QRS if there is bundle branch block, as in this case (diagnosis proven at EPS). The red arrows indicate the probable position of the retrograde P waves.

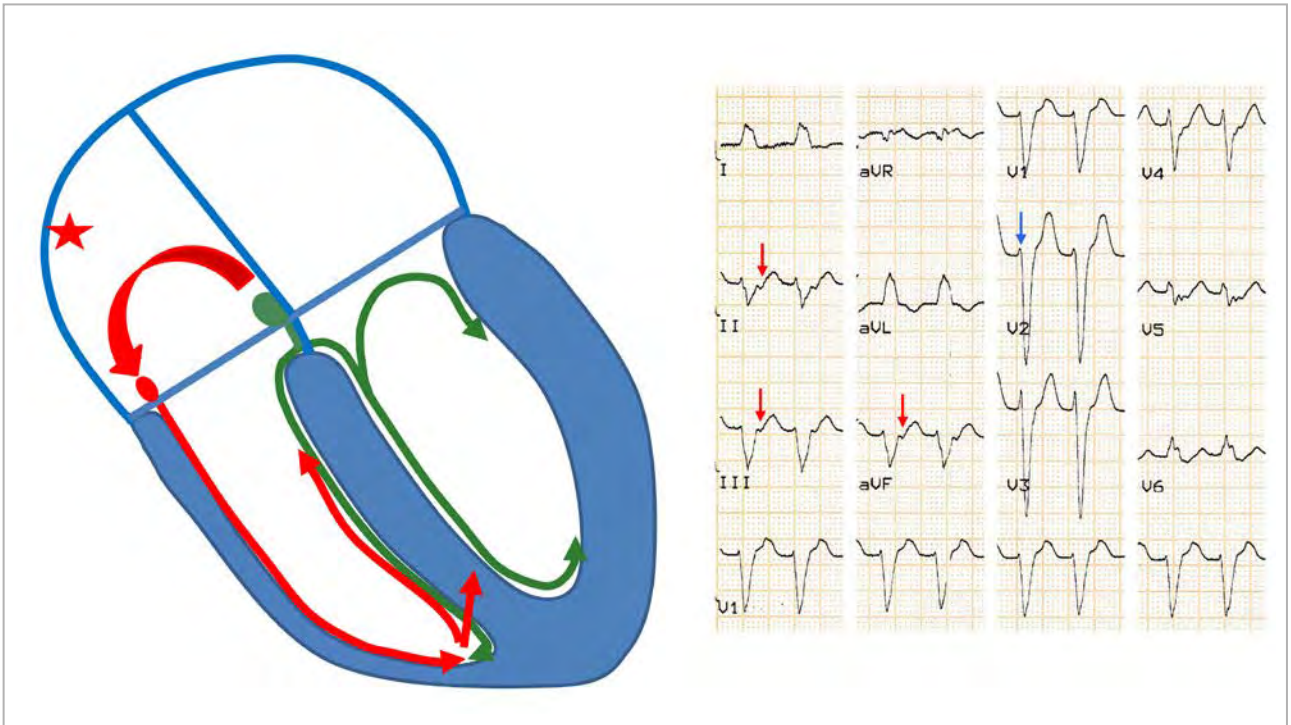


**FIGURE 4: Atriofascicular (Mahaim) tachycardia. The tachycardia circuit is shown in red.** The ECG is of a typical atriofascicular tachycardia. The rate is about 150bpm with a LBBB, left axis deviation pattern. However, the initial r in V2 is >40ms (blue arrow) suggesting this is not true LBBB. Negative (retrograde) P waves are visible in the inferior leads (red arrows).

## TREATMENT

While electrical cardioversion is the treatment of choice and could be safely carried out as long he has been therapeutically anticoagulated with Warfarin and maintained INR values between 2 and 3 for a minimum of 3 weeks, it does not contribute much to the diagnosis. Intravenous amiodarone does not have a high efficacy for termination and has the potential for significant side effects. I.V. adenosine should not be given when atrial flutter is suspected. While it temporarily blocks the AV node, it will not terminate flutter. It results in peripheral vasodilatation and a significant surge in catecholamines which can cause 1:1 conduction of the flutter and an acceleration in ventricular rate, particularly if the rate if the flutter itself is slower than usual as in this case. Figure 5, from another patient, illustrates this effect.

**The answer to question 2 is (b): Perform carotid sinus massage (CSM).**

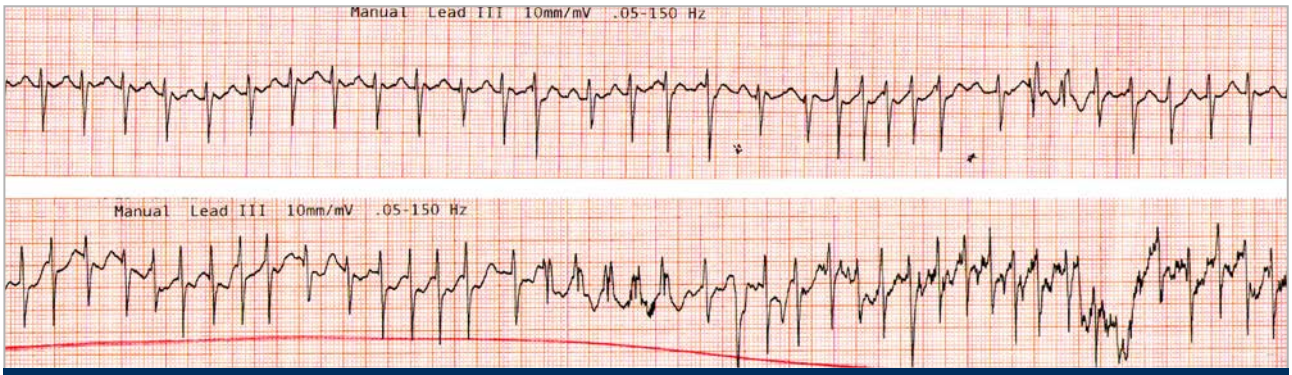


**FIGURE 5: Adenosine causes 1:1 conduction of atrial flutter. 2:1 flutter at 160bpm briefly slows and then accelerates to 250bpm as the catecholamine release shortens AV nodal refractoriness and 1:1 conduction supervenes.** Some R-R intervals are as short as 200ms (300bpm) which could induce ventricular fibrillation.

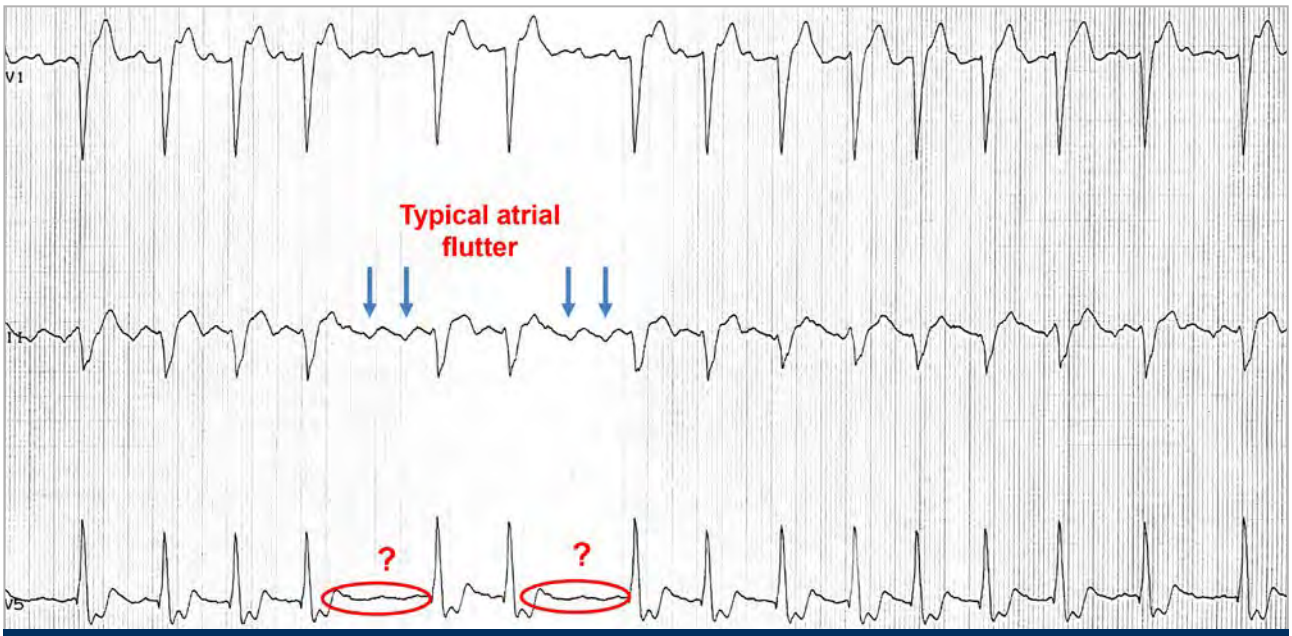


**FIGURE 6: Carotid sinus massage.** CSM briefly slowed AV nodal conduction, long enough to reveal the flutter waves (blue arrows), best seen in V1 and SII. The typical saw tooth pattern, continuous atrial activity with predominant negativity in SII and positivity in VI confirm typical counter-clockwise atrial flutter which is dependant on the isthmus between the inferior vena cava and the tricuspid valve and is therefore amenable to cure by catheter ablation.
This rhythm strip illustrates the importance of recording more than one channel during vagal manoeuvres or administration of adenosine. If only V5 had been recorded the diagnosis of atrial flutter could not have been made with certainty.

This temporarily slowed AV conduction and allowed clear demonstration of the flutter waves (Figure 6) thus confirming the diagnosis. Note that a 3 channel ECG should be run during CSM to be sure to reveal the P waves, which may not be discernible on a single channel. Thereafter the patient was cardioverted to sinus rhythm, which confirmed LBBB.

Since CSM revealed typical counter-clockwise flutter which is dependent on the isthmus between the inferior vena cava and the tricuspid valve, he was referred for catheter ablation of his atrial flutter which has a very high success rate for typical flutter. In view of his valvular heart disease, it is likely that he either has or will get atrial fibrillation. Nevertheless, this is preferable to flutter as the rate is much easier to control and it is less likely to cause troubling symptoms.

## LESSONS AND CONCLUSIONS

- The QRS morphology is key to the differentiation of supra-ventricular rhythms with bundle branch block from ventricular tachycardia and other causes of wide QRS, such as pre-excitation.
- Once SVT with typical bundle branch block is identified, search for P waves to identify the likely mechanism.
- P waves may be difficult to see as they may be hidden by the wide QRS.
- Vagal manoeuvres can be useful to reveal the atrial mechanism and confirm the diagnosis.
- Adenosine should be avoided if atrial flutter is suspected.
- The clinical context may be critical in interpreting the ECG.

**Conflict of interest: none declared.**

**ECG** and **QUESTION** on page 94