



## THE PRACTITIONER LE PRATICIEN

### Country cardiograms case 38: Answer

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**F**igure 1 (on page 164) displays a regular narrow-complex tachycardia with a rate of 178 beats/min. P waves cannot be reliably identified. Diffuse ST-T changes are present, with prominent T wave inversion in the inferior leads. The differential electrocardiogram (ECG) diagnosis includes sinus tachycardia (which does not fit the clinical picture of abrupt onset and termination), atrial flutter and supraventricular tachycardia.

Atrial flutter is a notorious mimic. The ventricular rate of 178 beats/min is a little faster than the typical rate of about 150 beats/min seen in atrial flutter, but does not exclude the diagnosis. Likewise, the typical saw-tooth pattern of atrial flutter is not seen in this tracing, but it is conceivable that 2:1 atrioventricular conduction is present, and that every alternate flutter wave is masked in the QRS complexes. Atrial flutter therefore cannot be fully excluded.

Supraventricular tachycardia is the likeliest possibility. In this setting, such ST-T changes are not infrequently seen and have been termed “rate-related.” Alternately, they may reflect an ischemic process resulting from an imbalance between myocardial oxygen supply and demand. In supraventricular tachycardias, retrograde P waves are often present, and can occur a considerable time after the QRS complex. It is possible that retrograde P waves are superimposed on the T waves in this tracing, giving the erroneous impression of deep T wave inversion in the inferior leads.

It is therefore not feasible to identify the rhythm with total precision in this case. Had it persisted, vagal manoeuvres or an intravenous bolus of adenosine would have been a reasonable

diagnostic and therapeutic choice, as they may have been effective in terminating supraventricular tachycardia. Although they would have been less likely to have an effect on atrial flutter, vagal manoeuvres or intravenous bolus adenosine may be of diagnostic value in unmasking flutter waves.

Regardless, once sinus rhythm has been re-established, it is essential to obtain a follow-up 12-lead ECG. In a case such as this, one would want to look at the ST segments and T waves in particular to establish whether the changes initially seen were still present. Indeed, normal sinus rhythm is present, at a rate of 85 beats/min, and almost all the ST-T changes have resolved (Fig. 2, on page 165).

However, something else unusual is evident in Figure 2. Left axis deviation ( $-35^\circ$ ) has developed, and the QRS complexes are notably different from those in Figure 1: there are deep, abnormal Q waves in leads III and aVF, along with a q wave in lead II. This is what has given rise to the computer interpretation of inferior infarct.

A disciplined, systematic approach to ECG interpretation now reaps dividends. After noting rate and rhythm, we move on to PR interval, QRS duration and QT interval, axis, and then morphology. It thus becomes clear that the PR interval is near the lower limit of normal, at 0.125 seconds, and that the QRS duration is increased at 0.115 seconds. This combination should always prompt a consideration of pre-excitation, and, indeed, in this case delta waves are visible in most leads, including I, aVL, and V2 through V6. In fact, the negatively inscribed delta waves in the inferior leads give rise to the “pseudo-myocardial

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infarction pattern” and the erroneous interpretation of inferior myocardial infarction.

Knowing that pre-excitation is present, it is possible to conclude that the tachyarrhythmia was almost certainly a reciprocating supraventricular tachycardia. This is the most common tachycardia associated with pre-excitation, and usually responds to standard treatment. In contrast, atrial flutter is rare in this scenario.

However, the second most common tachyarrhythmia associated with pre-excitation is atrial fibrillation. Although reciprocating supraventricular tachycardia is usually benign, atrial fibrillation in this setting is associated with significant complications and mortality. These are related to the potentially extremely rapid ventricular rate if the impulse travels down the accessory pathway rather than through the atrioventricular node. Degeneration

into ventricular fibrillation frequently occurs. Some of the medications usually used to treat atrial fibrillation, such as calcium channel blockers and digoxin, increase the incidence of this. They are therefore contraindicated when atrial fibrillation occurs in the presence of known pre-excitation, and when atrial fibrillation occurs with an unexpectedly rapid ventricular response of over 200 beats/min. In such cases, cardioversion is the treatment of choice; if cardioversion is unavailable, amiodarone or procainamide can be administered.

In this case, with a firm diagnosis established, discussion with the patient should ensue regarding the possibility of referral to an electrophysiologist and consideration of radiofrequency ablation of the accessory pathway.

**For the question, see page 164.**

### Cardiogrammes ruraux

Avez-vous eu à décrypter un ECG particulièrement difficile récemment?

Dans la plupart des numéros du *JCMR*, nous présentons un ECG assorti de questions.

Les réponses et une discussion du cas sont affichées sur une autre page.

Veillez présenter les cas, accompagnés d'une copy de l'ECG, à Suzanne Kingsmill, rédactrice administrative, *JCMR*, 45, boul. Overlea, C. P. 22015, Toronto (Ontario) M4H 1N9 ; [cjrm@cjrm.net](mailto:cjrm@cjrm.net)