



THE PRACTITIONER

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Country cardiograms case 30: Answer

INTERPRETATION AND DISCUSSION

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The ECG illustrated on page 119, taken at the time of the patient's presentation to the emergency department, displays a wide complex tachycardia, rate 164 beats/min. As often happens with this kind of tachycardia, the QRS complex merges with the downsloping ST segment and deeply inverted T waves, making exact measurement of QRS duration difficult, but QRS duration is at least 0.15 seconds. QRS morphology is unusual, and neither typically a right bundle branch block nor a left bundle branch block pattern. There is pairing of the QRS complexes, in a very regular pattern: R-R intervals are alternately 0.29 seconds and 0.44 seconds.

When confronted with any wide complex tachycardia, the differential diagnosis includes:

- ventricular tachycardia
- supraventricular tachycardia with pre-existing bundle branch block
- supraventricular tachycardia with aberrancy.

It is therefore important to try to obtain a copy of a previous ECG for comparison, and background history on the patient's cardiac status, if available. In this case the previous ECG, taken when the patient was asymptomatic (Fig. 1, illustrated on this page), does show QRS widening, but with a different morphology, especially in the precordial leads. It shows Q waves in leads V4, V5, I and aVL consistent with old anterolateral myocardial infarction. Her records show that she has a ventricular aneurysm, which may account for the coved ST segment elevation in leads V3-V5. Her left ventricular ejection fraction has been reported as 15%.

Based on this previous tracing,

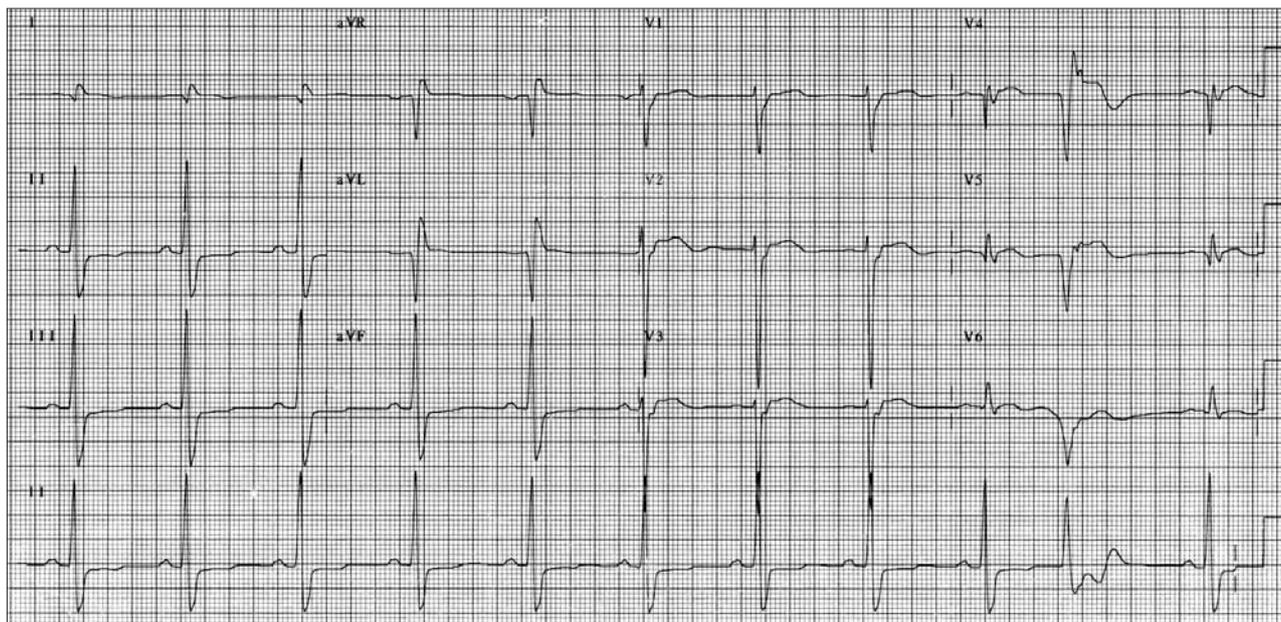


Fig. 1. Copy of a previous ECG, taken when the patient was asymptomatic, obtained for comparison.

supraventricular tachycardia with pre-existing bundle branch block can be ruled out.

The most unusual feature of the figure on page 119 (Question section) is the pairing of the wide QRS complexes, with the longer R–R interval less than twice the shorter R–R interval. This ratio is typical of Wenckebach's phenomenon, and in this scenario there are 2 possible causes to be considered. One is ventricular tachycardia with a 3:2 Wenckebach exit block from the ectopic ventricular focus. The other is supraventricular: atrial flutter with 3:2 Wenckebach conduction to the ventricles, along with aberrant conduction related to the rapid rate.

In general, ventricular tachycardia can often be "ruled in" by looking for certain clues, such as dissociated P waves, fusion beats and capture beats, but the absence of these phenomena does not rule it out. A close look for any atrial activity is therefore crucial in analyzing a wide complex tachycardia.

Scrutiny of this tracing shows some interesting features in the rhythm strip. First, there is a small negative deflection before every second QRS complex. Second, the QRS complexes alternate in height, as does the depth of the subsequent T wave inversion. Could atrial activity be causing these subtle repetitive changes?

The answer may lie in lead V1, always a good lead to use to look for atrial activity. A probable saw-tooth pattern can be seen "marching through"

the paired QRS complexes in this lead at a rate of 246 beats/min (Fig. 1, previous page).

The most likely ECG diagnosis thus becomes atrial flutter with aberrancy, and 3:2 A–V conduction.

Vagal manoeuvres may be useful in confirming this. Symptoms, vital signs and degree of cardiac compromise will dictate the urgency and type of treatment — drugs, or synchronized cardioversion. In this case the low ejection fraction alone dictated the need for rapid treatment — she was given a loading dose of amiodarone without effect, and then cardioverted, followed by the return of sinus rhythm.

It often remains impossible to determine whether a wide complex tachycardia is due to a ventricular or supraventricular cause. In the past, physicians were faced with potential catastrophic consequences if digoxin or verapamil were inadvertently used on a ventricular tachycardia. When in doubt, amiodarone can now be used effectively in either scenario, and adenosine is unlikely to exacerbate a ventricular tachycardia. Nonetheless, the old maxim of assuming a ventricular origin for the tachycardia, unless there is good evidence to the contrary, still holds true.

For the Question, see page 119.

Acknowledgement: I am indebted to Dr. Anton Venter for clinical details on this case.

Country Cardiograms

Have you encountered a challenging ECG lately?

In most issues of *CJRM* an ECG is presented and questions are asked.

On another page, the case is discussed and the answer is provided.

Please submit cases, including a copy of the ECG, to Suzanne Kingsmill, Managing Editor, *CJRM*, Box 1086, Shawville QC J0X 2Y0; cjrm@lino.com

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.

Veuillez présenter les cas, accompagnés d'une copie de l'ECG, à Suzanne Kingsmill, rédactrice administrative, *JCMR*, CP 1086, Shawville (Québec) J0X 2Y0; cjrm@lino.com