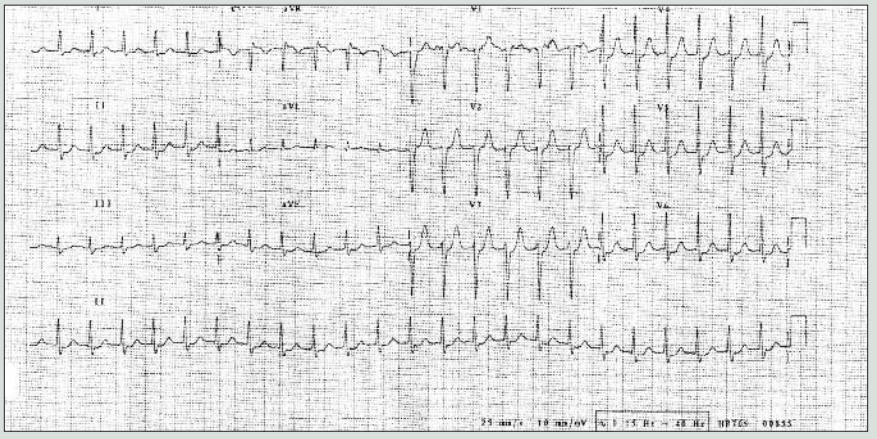
Is this a junctional tachycardia?

VIVIENNE MILLER
MB BS, FRACGP, DRACOG, DCH, MACPM, MWAME

ECG Education articles use real cases to illustrate the importance of knowledge about ECGs in relation to clinical situations in general practice. Management is not discussed.

A 69-year-old man unknown to your practice walks into your waiting room and tells the receptionist that he is having heart palpitations and needs an urgent ECG. He tells her that he has had fast heart rates several times in the past year but they returned to normal before he could obtain an ECG.

He is currently taking no medications and is usually well. He seems stable but anxious and the receptionist informs you immediately of his request. You perform an urgent ECG, which showed the following arrhythmia.



Q1. What are the differential diagnoses in the interpretation of this ECG?

The ECG shows a narrow-QRS or supraventricular tachycardia. High on the list of differential diagnoses is a junctional tachycardia, also known as an atrioventricular junctional (or nodal) re-entry tachycardia (AVJRT or AVNRT). Other important possibilities are an accessory pathway-mediated tachycardia (associated with Wolff–Parkinson–White [WPW] syndrome) and a focal atrial tachycardia.

Atrial flutter with a 2:1 block cannot be excluded but is not obvious. The patient's heart rate is 140 bpm. There is some ST depression in leads II, aVF and V3 to V6. This does not necessarily indicate myocardial ischaemia, but you quickly check the patient for symptoms or signs of possible ischaemia or heart failure (i.e. pain in the chest, jaw or arm, dyspnoea, tachypnoea, sweating, hypotension, pulmonary crackles or elevated jugular venous pressure).

Q2. What are junctional tachycardias?

Junctional tachycardias are arrhythmias presenting with a rapid heart rate (usually between 140 and 250 bpm) due to an abnormal re-entrant circuit in the atrioventricular (AV) junction (near the AV node and the bundle of His). Typically, the abnormal impulses are conducted retrogradely to the atria and anterogradely to the ventricles.

AVJRT and accessory pathway-mediated tachycardia can both typically be terminated using adenosine and often respond to carotid massage or other vagal stimulation. Atrial tachycardias and flutters, by contrast, are not usually terminated by adenosine or vagal manoeuvres, and continue with AV block and dissociation (especially after adenosine).

Q3. How do junctional tachycardias appear on the ECG?

Junctional tachycardias always have a narrow QRS complex unless there is a pre-existing or rate-related bundle branch block. They usually cause a heart rate of between 140 and 250 bpm. P waves are typically close to (less than 0.12 seconds) or coincide with the QRS complex. If they follow the QRS complex then they may distort the ST segment. They are negative in the inferior leads but may be difficult to discern.

Q4. What can cause a junctional tachycardia?

The common type of junctional tachycardia, AVJRT, is simply a quirk of normal junctional physiology. However, automatic junctional tachycardias may occur in the following situations:

- as complications of myocardial infarctions
- after surgical repair of the heart (especially of congenital conditions and manipulation of the heart, typically during valve replacement)
- due to metabolic changes associated with bypass surgery
- due to digitalis toxicity
- during successful resuscitation after cardiac arrest.

CARDIOLOGY TODAY 2012; 2(1): 39-40

Dr Miller is a GP in Sydney, NSW. She is also an editor, author and medical journalist and is the Medical Editor of *Cardiology Today*.

Q5. How do junctional tachycardias present clinically?

There is a lot of variation in age and presentation of patients with junctional tachycardias. The arrhythmias have been documented in utero and do occur in neonates and children, especially if there is congenital heart disease such as tetralogy of Fallot. They also occur in younger adults with structurally normal hearts. The common forms of tachycardia usually start and stop suddenly. They may go on for seconds, hours or days and rarely may be continuous.

Significant symptoms of junctional tachycardias are more likely in people with underlying heart conditions and include a fast, thumping heart rate that can be felt in the neck, fatigue, faintness, chest discomfort, shortness of breath and increased effort of breathing. Heart failure may occur. Sudden death is usually due to an associated cardiac condition or the underlying condition causing the junctional tachycardia, rather than the tachycardia itself.

Q6. Could this patient have Wolff–Parkinson–White syndrome?

Yes. The classical short PR interval (less than 120 msec) and delta wave (broadening of the QRS complex and slurring of its onset) is not present during the tachycardia. It is important to perform another ECG in sinus rhythm after termination of the tachycardia. The ECG signs of this syndrome may be intermittent and electrophysiological studies are indicated if it is found or suspected.

Q7. What is Wolff–Parkinson–White syndrome?

WPW syndrome is the constellation of a short PR interval and delta waves on the sinus rhythm ECG, and documented paroxysms of symptomatic supraventricular tachycardia. It is the classic example of an AV re-entrant tachycardia syndrome. An accessory pathway, previously called a bundle of Kent, that links the atria to the ventricles is abnormal in patients with this condition. In 10% of cases there is more than one abnormal pathway.

The tachycardia results from a premature atrial impulse travelling down the AV node to the ventricles and then retrogradely through the accessory pathway back to the atria. This abnormal electrical conduction keeps repeating rapidly, causing a narrow complex ('orthodromic') tachycardia. Less commonly the re-entry occurs with retrograde conduction through the AV node and anterograde conduction from the atria to the ventricles via the accessory pathway, causing a wide complex ('antidromic') tachycardia.

If the delta wave is positive in the V1 lead (WPW type A), the accessory pathway is likely to insert into the left ventricle. Conversely, if the delta wave is negative in the V1 lead (WPW type B) then the accessory pathway is likely to insert into the right ventricle.

Q8. What is the danger of Wolff–Parkinson–White syndrome?

Sudden death may occur from ventricular fibrillation if the patient with WPW syndrome gets atrial flutter or atrial fibrillation and the accessory pathway is capable of dangerously frequent conduction (short refractory period). The refractory period of the accessory pathway can generally only be measured by electrophysiology study, but can be estimated as the shortest RR interval from ECG monitoring during an episode of atrial fibrillation.

Q9. Which medications should be avoided in Wolff–Parkinson–White syndrome?

Some drugs that block the AV node (e.g. digoxin and verapamil) may cause increased conduction through the abnormal pathway, worsening the tachycardia and resulting in ventricular fibrillation.

Outcome

The cardiologist was pleased that the ECG and echocardiography showed the heart was structurally normal. The patient underwent electrophysiological studies to diagnose and treat the tachycardia. It was an AVJRT, not WPW syndrome. The patient went on to have successful catheter ablation of the re-entry pathway and has remained well and asymptomatic. **CT**

Key points

- **Junctional tachycardias present with a rapid heart rate (usually between 140 and 250 bpm) and are due to an abnormal re-entrant circuit near the AV node and the bundle of His. Typically, the abnormal impulses are conducted retrogradely to the atria and anterogradely to the ventricles.**
- **Both AVJRT and accessory pathway-mediated tachycardias (such as Wolff–Parkinson–White syndrome) can typically be terminated using adenosine, and often respond to carotid massage or other forms of vagal stimulation.**
- **Atrial tachycardias and flutters are not usually terminated by adenosine or vagal manoeuvres, and continue with AV block and dissociation (especially after use of adenosine).**