ECG Quiz 70

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

ANSWER

OVERVIEW OF THE ECG

This is a narrow QRS tachycardia, with an average rate of 150 per minute and regular, apart from 2 gaps. A P wave is visible before the QRS that ends the gap. Aside from this, P waves are less easy to distinguish.

More detailed analysis of the ECG

The T waves in leads II and aVF are peaked, suggesting that there are P waves superimposed on the T waves. In V1, there is a sharp negative deflection, which is too high a frequency to be part of the T wave. This is borne out by the 2 P waves after the gaps, which are negative, and the negative deflections in V1 correspond with the peak of the T waves in II and aVF. The negative deflection after the T wave is absent in the complex before the pause. Therefore, there is a 1:1 atrioventricular/ventriculoatrial (AV/VA) relationship. It is a long RP tachycardia (Figure 1).

The R-R interval of the tachycardia is 345 ms, equivalent to a rate of 174 per minute. The P-P interval in the 2 gaps is 660 ms, slightly shorter than 2 tachycardia cycle lengths (Figure 2). The PR intervals before and after the gaps are the same, about 140 ms. The P wave axis is about +50° (using the single visible P wave in the augmented limb leads). The QRS duration is normal (80 ms). The QT interval cannot be measured accurately because of the superimposed P wave. The differential diagnosis of a long RP tachycardia is listed in Table I.

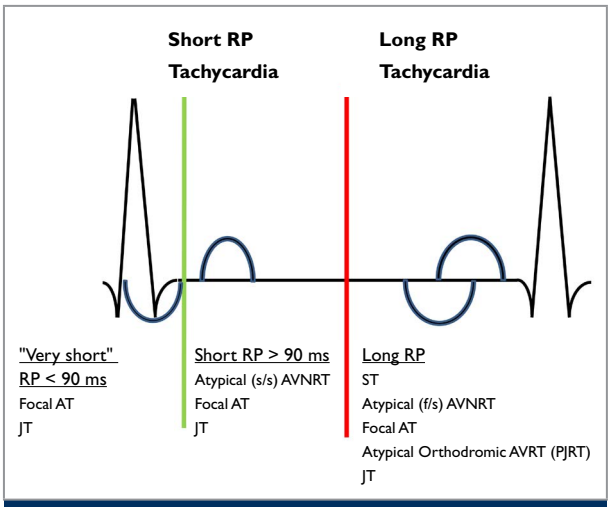

FIGURE 1: Regular narrow QRS tachycardias can be divided into short or long RP, depending on the relationship of the P wave to the QRS complex. A short RP describes P waves in the first half of the R-R interval and can be further divided into short and very short. In the latter case, the P wave may be hidden completely or partially within the QRS. Depending on this relationship, the differential diagnosis varies. However, note that focal atrial tachycardias and junctional tachycardia can have P waves in any of these positions. ***AT: atrial tachycardia, AVNRT: atrioventricular nodal re-entry tachycardia, AVRT: atrioventricular re-entrant tachycardia, f/s: fast-slow, s/f: slow-fast, s/s: slow-slow, JT: junctional tachycardia, PJRT: permanent junctional reciprocating tachycardia***

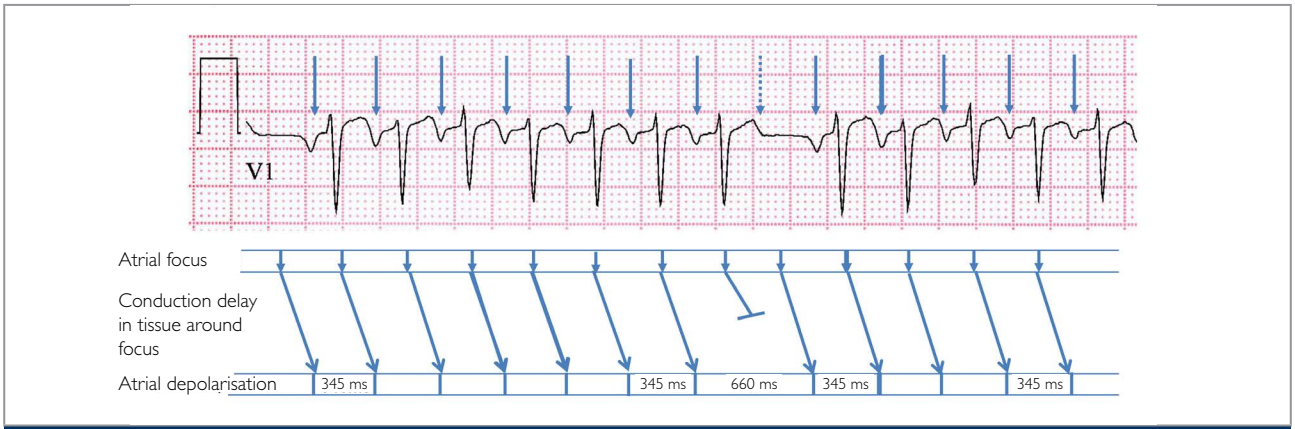

FIGURE 2: The ladder diagram shows the relationship between the origin of the tachycardia and the resultant P waves. The degree of conduction delay through the tissue surrounding the focus is unknown; however, the resulting P-P interval reflects the discharge rate of the focus. The dotted arrow shows the presumed discharge, which is blocked in the tissue around the focus. The resulting pause is 30ms shorter than 2 P-P intervals. A possible explanation is the slightly faster conduction through the surrounding tissue after the pause, analogous to Wenckebach atrioventricular block.

TABLE I

- Sinus tachycardia
- AV junctional re-entry tachycardia:
  - Atypical AV nodal re-entry tachycardia
  - AV re-entry tachycardia with a slowly conducting accessory pathway
- AT
- Junctional ectopic tachycardia with retrograde P waves

AT: atrial tachycardia, AV: atrioventricular

DISCUSSION

This is highly unlikely to be sinus tachycardia. The electrocardiogram (ECG) was done at rest. The predicted maximum heart rate for a 49-year-old man is about 170 per minute (220-age). This will only occur during maximum exercise or other extreme physiological stress. Sinus rates above 130 per minute at rest are uncommon, even with conditions such as thyrotoxicosis or shock. If the rate had been much slower, a diagnosis of sinus rhythm with intermittent exit block could be considered, as the P wave axis is normal.

Atrioventricular (AV) junctional re-entry tachycardia is not usually interrupted for brief periods, roughly equal to 2 R-R intervals. Termination and restarting are usually random. AV nodal re-entry tachycardia (AVNRT) is triggered by an atrial premature complex, which blocks in the fast AV nodal pathway (Figure 3) and conducts via the slow pathway, resulting in a prolonged PR. This may then return retrogradely via the fast

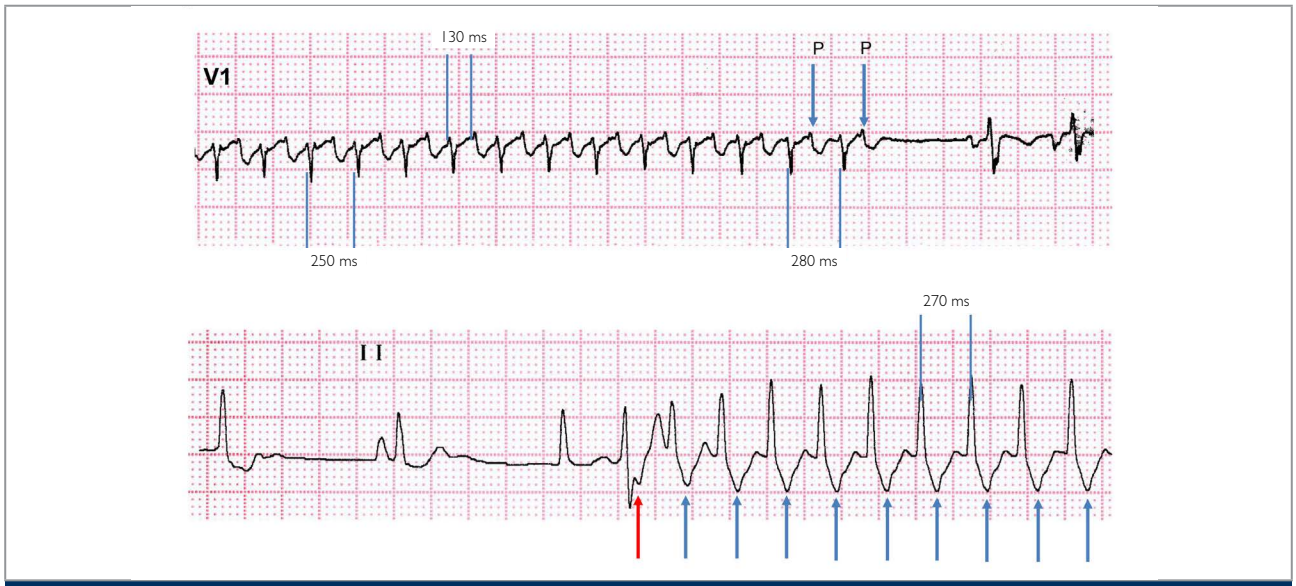

FIGURE 3: An example of a borderline long RP tachycardia due to atypical (fast-slow or slow-slow) atrioventricular nodal re-entry tachycardia (AVNRT).
Upper strip (V1): The tachycardia stops spontaneously and ends with a P wave not followed by a QRS due to block in the antegrade fast pathway. This pattern excludes an atrial tachycardia as it is doubtful that the tachycardia would stop simultaneously with atrioventricular block.
Lower strip (lead II): The tachycardia restarts spontaneously, following a junctional escape beat and a premature complex of undetermined origin, probably junctional. This complex is followed by a retrograde P (red arrow) with a short RP interval, which re-enters the circuit to restart the tachycardia. In this strip, the RP has shortened slightly to 120 ms, so it is now a short RP (cycle length 270 ms). While this is an unusual form of AVNRT, it illustrates the difference between an atrioventricular junctional re-entry tachycardia and a focal atrial tachycardia. In atrioventricular junctional re-entry tachycardia, the RP interval is usually fixed (RP linking), which is not the case with an atrial tachycardia.

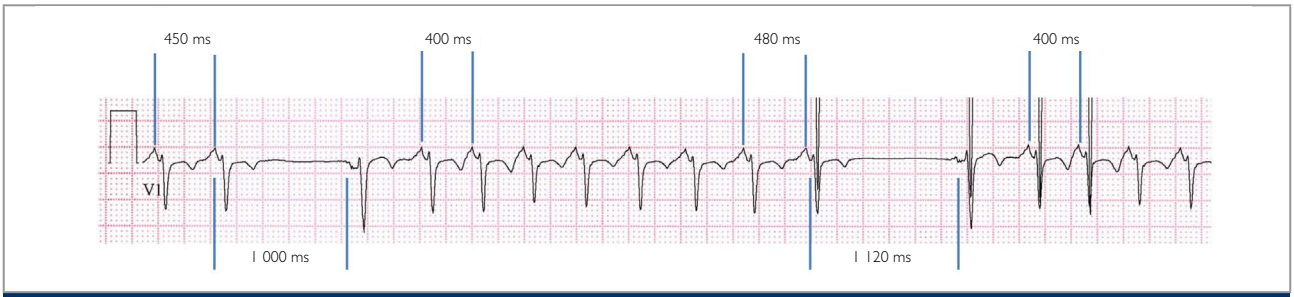

FIGURE 4: A repetitive atrial tachycardia with brief pauses. Note that the pauses are unequal and bear no relation to the tachycardia cycle length. The triggering P wave has a different morphology. Each cycle terminates with a QRS, not a P wave, consistent with an atrial tachycardia, which, in this case, arose from the left atrium. Also, note that the atrial tachycardia slows before it stops.

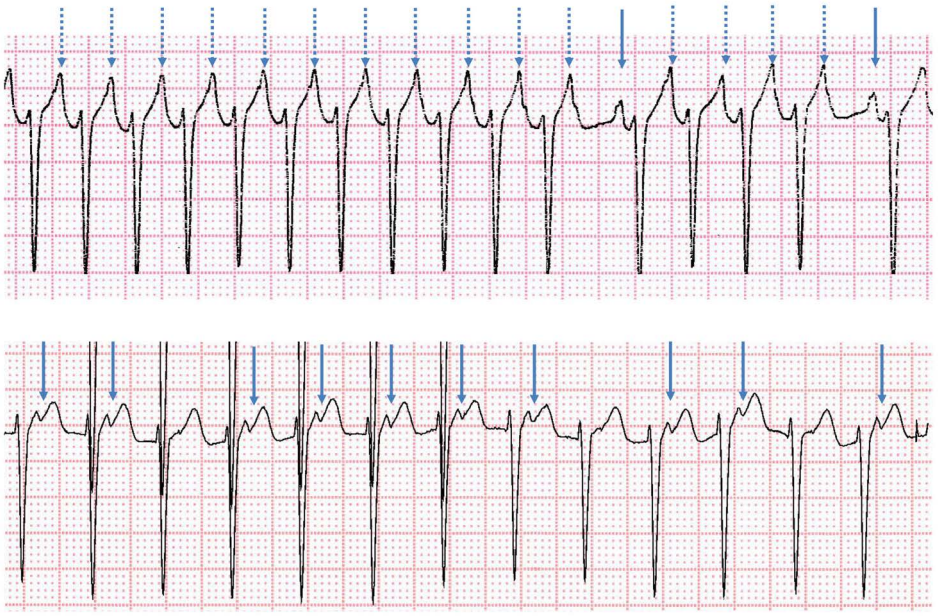

FIGURE 5
Top panel: The P waves in this atrial tachycardia interrupt the peak of the T waves (dotted arrows). Occasional P waves fail to conduct (2:1 atrioventricular block), but there are no missing Ps. This observation excludes junctional re-entry tachycardias, which require a 1:1 atrioventricular relationship.
Bottom panel: This is a short RP tachycardia due to an ectopic focus in the atrioventricular junction (JET). There are retrograde P waves (solid arrows) with intermittent retrograde atrioventricular block. This excludes both atrioventricular junctional re-entry tachycardias (1:1 atrioventricular relationship) and atrial tachycardias, which require 1 or more P waves per QRS.

pathway, if it is no longer refractory. The PR intervals after the pauses are only 140 ms. In orthodromic AV re-entry tachycardia (AVRT) (Wolff-Parkinson-White Syndrome), the initiating premature atrial complex (PAC) blocks in the refractory accessory pathway. In repetitive atrial tachycardia (AT), the gaps are not necessarily a multiple of the tachycardia cycle length (Figure 4). AT with variable AV block is characterised by intermittently blocked P waves (Figure 5).

AT with intermittent exit block is characterised by the intermittent absence of a P wave with no or minimal disturbance of the rhythm, such that the pause is a multiple of the tachycardia cycle length. In this case, the P-P interval in the pauses is 660 ms, slightly shorter than 2 tachycardia cycle lengths (345 ms x 2 = 690 ms). The likely cause is a shortening of the conduction time from the discharging atrial focus to the surrounding atrial tissue after the brief rest engendered by the block, analogous to Wenckebach (Type I) AV block. Importantly, the R-R intervals of the 2 pauses are identical. The exit block is convincing evidence that this is a focal AT or possibly micro-re-entry, surrounded by a zone of atrial tissue with variable conduction properties. It is doubtful that a repetitive AT would restart with exactly the same P-P and R-R intervals. While the P waves resemble sinus Ps, they are sharply negative in V1, which is not the case during sinus rhythm in the same patient (Figure 6).

Therefore, the correct answer is (c): Atrial tachycardia with an intermittent 2:1 exit block.

Focal ATs spread centrifugally from their site of origin, which can be anywhere in either atrium. Those that occur close to the sinus node or in the crista terminalis may be mistaken for sinus tachycardia. While the P wave morphology may mimic sinus rhythm, the key difference is that the rate is physiologically inappropriate. There are several possible underlying mechanisms for focal AT. Triggered activity due to afterdepolarisations is related to calcium shifts across the cell membrane. Micro-re-entry is confined to a small area and is therefore focal. Automatic discharge of cells in a small area acts as a pacemaker, if the rate exceeds that of the sinus node. Focal ATs are commonly incessant and can result in tachycardia-induced cardiomyopathy.

Adenosine is useful for terminating a suspected AV junctional re-entry tachycardia if vagal manoeuvres fail to terminate it. However, while the termination with a vagal manoeuvre is highly specific for tachycardias involving the AV node as part of the circuit (AVNRT and AVRT), adenosine is much less so. About 50–60% of focal ATs can terminate with adenosine.[1] Termination with adenosine appears to be specific for focal ATs that are due to triggered activity. Focal automatic ATs do not terminate but tend to slow or be transiently suppressed.[1]

Intravenous verapamil should not be used, even for narrow QRS tachycardias. If it does not restore sinus rhythm, which it will not with AT, it can cause haemodynamic collapse.[2] This is particularly likely if the tachycardia is incessant and has resulted in tachycardia-induced cardiomyopathy.

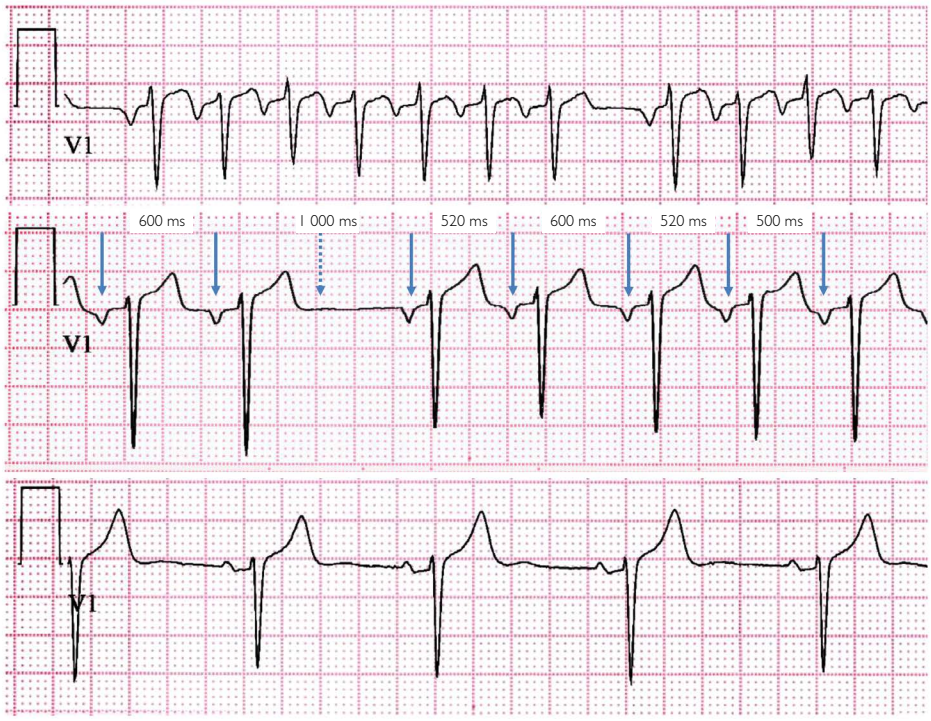

FIGURE 6
Top panel: Shows the presenting ECG with intermittent 2:1 exit block.
Middle panel: Shows the same P wave morphology, but much slower after treatment with propranolol. The atrial rate is now fluctuating between 100 and 120 beats per minute. This fluctuation makes it difficult to prove that the gap is due to the exit block, but it still seems likely.
Bottom panel: Shows a later ECG in sinus rhythm. The P waves are now clearly different, upright as opposed to inverted in V1. This sequence of ECGs and the response to propranolol suggest that the underlying mechanism is an automatic focus, rather than micro-re-entry.

This patient was successfully treated with oral propranolol. The tachycardia initially slowed without a change in the P wave morphology (Figure 6) and, subsequently, sinus rhythm was restored. While adenosine was not used, the slowing on propranolol implies a focal, automatic tachycardia. An intermittent exit block persisted at a slower rate, with pauses equal to, or close to, 2 P-P intervals.

LESSONS AND CONCLUSIONS

- The distinction between exit block and random pauses depends on the consistent relationship between the length of the pause and the cycle length of the basic rhythm.
- Exit block can occur with any focus acting as a pacemaker:
  - Sinoatrial node (most common) – a form of sinus node dysfunction.
  - Atrial focus.
  - Ventricular focus, e.g. accelerated idioventricular rhythm.[3]
- The differential diagnosis of long RP tachycardia includes all in Table I.
- AT can also be short RP, as the unphysiological fast atrial rate will prolong AV nodal conduction in the absence of catecholamine stimulation.
- ATs are commonly incessant and can lead to tachycardia-induced cardiomyopathy.

REFERENCES

1. Liu CF, Cheung JW, Ip JE, et al. Unifying algorithm for mechanistic diagnosis of atrial tachycardia. Circ Arrhythm Electrophysiol. 2016;9(8):e004028. https://doi.org/10.1161/CIRCEP.116.004028.
2. Lawrenson JB, Okreglicki AM, Millar RN. Cardiovascular collapse due to intravenous verapamil in two patients with persistent atrial tachycardia. S Afr Med J. 1995;85(11 Suppl):1236-8.
3. Okreglicki A. ECG Quiz no. 28. SA Heart J. 2012;9(3).