ECG Quiz 22

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

ANSWER

Systematic analysis of the ECGs: Let us look at ECG 2 first.

ECG 2 SHOWS

1. Rate and regularity: regular with a ventricular rate of 48 bpm (6 x 8 beats)
2. Atrial activity: P-waves compatible with sinus origin are seen preceding each QRS. The P-waves are slightly broad in Lead I and peaked in V1.
3. PR interval measures 11 small blocks x 0.04s = 440ms; and is constant.
4. QRS complexes are narrow, with normal axis of around 45° and nil suggestive of myocardial infarction.
5. ST and T-waves are normal.

Thus, in ECG 2 there is sinus bradycardia with first degree AV block. Clearly, the computer analysis is incorrect. In V1, one may at first glance mistake the P-wave as a QRS complex and possibly a bigeminy but this would be wrong. The only part of the computer diagnosis that is correct is "abnormal ECG".

ECG 1 SHOWS

1. Rate and regularity: regular with a ventricular rate of 96 bpm.
2. Atrial activity: Is there any? The ECG computer appears to suggest that there is none and that the QRS complex is now wide with two components as seen in V1. Again, the computer is wrong, as careful analysis shows that the QRSs in ECG1 are no different to those in ECG 2. Just after the QRS and before the peak of the T-wave, best seen in the V leads, is a notch that is a P-wave. Unfortunately, in the first half of the ECG, in the limb leads, the quality of the recording is poor with artefact, which may be misinterpreted as additional atrial activity. If one looks carefully to find something equivalent to the notches seen in the chest leads (V1-6) that have been attributed to P-waves, one can see that they are also present in the limb leads: as in aVR, and also well seen after the second and fourth QRS complex in leads II and III. Interestingly, the rate in ECG 2 is almost half of that in ECG 1. Before suggesting that this halving of rate represents possible sinus node 2:1 exit block in ECG 2 accounting for the bradycardia, it is important to establish whether the P-waves in ECG 1 are of the same origin as in ECG 2, viz. the sinus node. Looking at leads II, III and aVF and ignoring the artefact, the P-waves in ECG 1 are negative and thus cannot be arising from the sinus node. Thus, this rhythm is not sinus and the doubling of heart rate is coincidental. What this rhythm is will become apparent. The negative P-waves in the inferior leads indicate that activation of the atria is from inferior to superior direction suggesting 2 possibilities: a low atrial ectopic rhythm or retrograde conduction from the AV node to the atria.
3. PR interval: There is a 1:1 relationship between P and QRS complexes. The question is which comes first: whether the P-wave causes the QRS or, vice versa, the QRS causes the P-wave? Although a PR interval can be measured as 500ms (12.5 x 40ms), this does not imply that the causative sequence is P causing QRS. Furthermore, an RP interval can be calculated: 2 blocks x 40ms = 80ms or a short RP relationship. Mechanistically from the cardiac function point of view, a P-wave occurring during or immediately after a QRS, whatever the mechanism, will cause the atria to contract while the ventricles are contracting, i.e. the atria contract against closed AV valves resulting in "cannon A-waves", which may also be seen in "pacemaker syndrome". This was, in fact, observed in this patient during ECG 1, with prominent rapid pulsations in the JVP and accounted for his presenting complaint.
4. QRS complexes are narrow and as mentioned above, the same as in ECG 2.

In summary

The ECGs have clearly recorded: two completely different rhythms; neither are tachycardias because the rates are both <100bpm; and long PR intervals. The rhythm in ECG 1, also

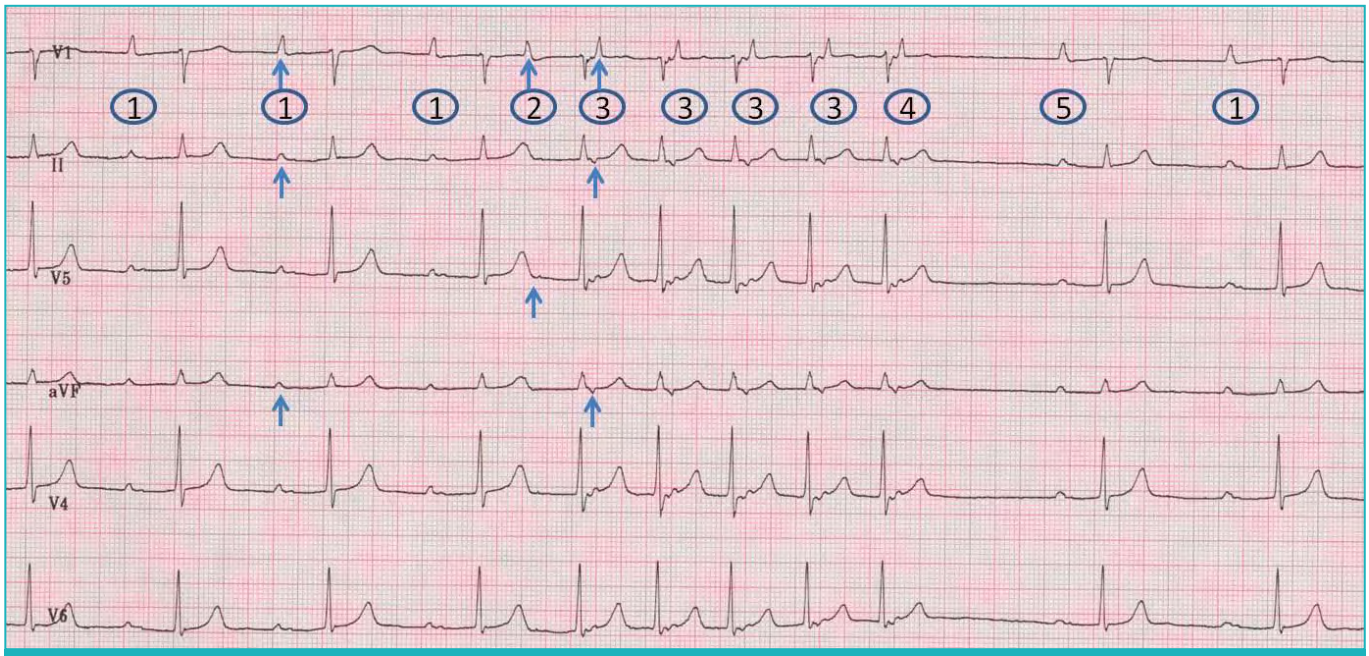

FIGURE 1: ECG of "Change of Rhythm"
This multichannel ECG rhythm strip shows a spontaneous onset and termination of a change of rhythm.

1. This marks the sinus beats of the sinus bradycardia at the start and at the very end of this recording strip. The arrows point to the positive P-waves in Leads II and aVF of sinus rhythm.
2. This atrial ectopic/premature beat triggers off a five beat run of "rhythm change". Note that the morphology of the P-wave is not the same as sinus beats; arrowed clearly in Leads V1 and V5.
3. These P-waves occurring immediately after the QRS with an RP interval of 80ms are "skinnier" in all the leads including V1 and more importantly negative in the two inferior leads shown: II and aVF. These features are typical of atrial activation originating from or close to the AV node as in retrograde AV node conduction (in which case this is fast pathway conduction) or an ectopic focus, respectively.
4. The last retrograde P-wave of this run of five similar beats. This is the final event of this run and is not followed by a QRS. This combination of two phenomena: termination of the arrhythmia together with failure of AV conduction is in keeping with antegrade block in the slow pathway of an AV node with dual pathways and almost certainly excludes a focal atrial tachycardia that just stopped "firing".
5. After a pause due to resetting of the sinus node, a sinus beat arises and conduction via a slow AV node pathway resumes.

mentioned above, is either an atrial ectopic rhythm or a rhythm arising from the AV junction or AV node with retrograde P-waves.

Further discussion

Can we get closer to the diagnosis? Long multichannel ECG rhythm strips which were obtained after recording ECG 2 revealed an interesting recurring mechanism.

Figure 1 shows the bradycardia as in ECG 2. Next, there is an atrial ectopic/premature beat. This is clearly not a normal sinus beat because the P-wave is different; seen best in Lead V1 where the usual odd looking P-wave changes slightly. This P-wave is followed by an even longer PR interval. Immediately after the next and subsequent four QRS complexes are P-waves which are more easily seen than in ECG 1 because this is a technically better recording. These P-waves are clearly different to the usual P-waves and different to the ectopic P-wave. Of great importance is the observation that this run of arrhythmia terminates with the last "event" being a P-wave. This combination of a P-wave not being followed by a QRS and, at the very same time as this failure of conduction, the termination of the arrhythmia, most strongly suggests that this is not an atrial driven rhythm. Why should the

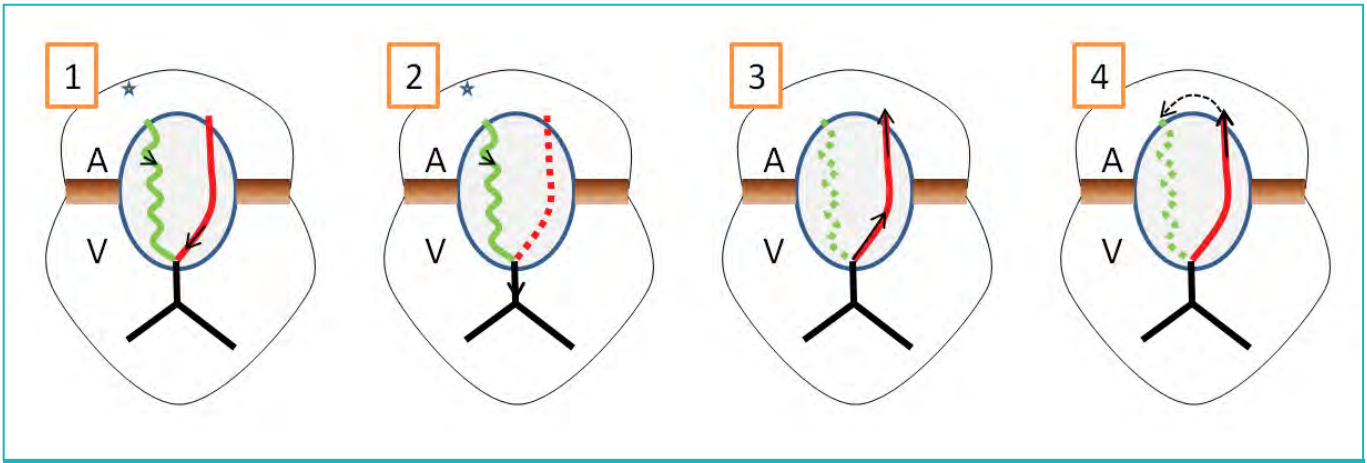

FIGURE 2: AV node dual pathways
The diagrams depict the atria "A" and ventricles "V" separated by an "insulation" and a magnified view of the AV node.

1. In the commonly found AV node with dual pathway physiology (NB not a pathology), a sinus beat will enter both the slowly conducting pathway (green) and the fast pathway (red). The fast pathway "wins" this race and delivers an impulse to the V with a normal PR interval.
2. In this patient, in sinus rhythm, the fast pathway has malfunctioned and conduction is via the slow pathway with a long PR interval and first degree AV block.
3. After conduction of an ectopic atrial beat with further slowing of an already slowly conducting pathway, a critical alteration in timing allows entry of the impulse into the "bottom" end of the fast pathway and retrograde AV node conduction to the atria (with a short RP time). This impulse originates in the atrium and 'echoes' back to the atrium.
4. The echo beat carries on echoing, passing through atrium and back down the slow pathway. This results in the AV nodal re-entry rhythm or AV Node Re-entry Tachycardia (AVNRT).

last P of a run of atrial rhythm or tachycardia not be conducted to the QRS? Rather, this is rhythm driven by the AV junction and the P-wave or atrial activation follows the QRS.

In this case, with no evidence of an accessory pathway or Wolff Parkinson White syndrome, the electrophysiological mechanism is what occurs in typical "slow-fast" AV nodal re-entry tachycardia (except that this is not quite in the tachycardia range). This patient is most likely to have dual AV node physiology, a common variant of normality with a fast and a slow AV nodal pathway. (See Figure 2.) These pathways are often capable of conducting in both directions i.e. antegradely and retrogradely. In this patient, antegrade conduction from atria to ventricles (A to V) is slow during normal sinus rhythm/bradycardia (ECG 2) accounting for the long PR interval and indicates that the fast pathway is mal-functioning. With the atrial ectopic, the AV conduction down the slow pathway is even slower, again a normal physiological response of the AV node to ectopics. However, this then sets up an interesting echo from the "bottom" of the AV node with the impulse travelling up to the atria quickly with a short RP time via a fast retrogradely conducting AV node pathway. This re-entry phenomenon recurs until the antegrade conduction in the slow pathway fails and after a pause, a normal sinus beat appears.

Correct answer: **(e)**

CONCLUSIONS/LESSONS

- Palpitations can occur during rhythms that are neither fast, slow nor irregular.
- Documentation (preferably by multichannel ECG recording) of the moment of change of rhythm whether it occurs spontaneously or is the result of a manoeuvre (e.g. vagal or after a drug) is often crucial in making the diagnosis.
- Analysis of the P-waves is important to determine whether their origin is from the sinus node, ectopic site or retrograde.
- ECG computer analysis is frequently incorrect and should never be relied on.

ECG and **QUESTION** on page 58