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

# ANSWER TO ECG Quiz 6

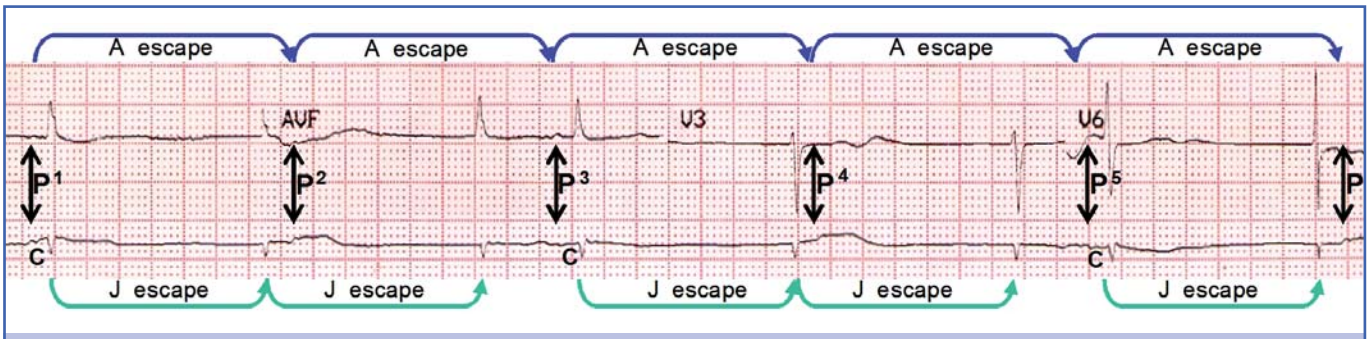


**FIGURE 1:** The lower 2 traces of ECG #6 showing sinus arrest and the 2 escape rhythms:
1. Atrial escape rhythm with atrial activity marked out P1 to P6, and
2. Junctional escape rhythm that is reset by the conducted QRS, C.

One's first impression of the ECG is that there is a bradycardia. But what if the ECG had been recorded at double normal speed (i.e. at 50mm/s)? The QRS complexes at normal heart rate would then be further apart and just give the false appearance of a bradycardia. Therefore, it is important before analyzing the ECG any further to quickly check the recording speed. Although no speed is given on this ECG, there is a calibration mark just before lead III. The width is one 'big block' confirming a normal recording at 25mm/s and 2 blocks high indicating amplitudes are 10mm/millivolt. Furthermore, if the recording speed were fast, all the QRS complexes would also be double in width – instead, they are surprisingly narrow. Thus, the patient does have a bradycardia and answer (e) is wrong.

A number of important observations can be made:

- Overall average heart rate is 54 bpm (=9 QRS complexes in this 10 second recording x 6).
- The rhythm is irregular. Could this signify atrial fibrillation (A Fib) ?
- The irregularity is not totally irregular as can be confirmed with a pair of calipers or scrap paper marking the QRS to QRS distance (R-R intervals). In fact, there are 2 recurring R-R intervals, one around 6½ big blocks wide and the second 3 big blocks. This regular irregularity is not in keeping with conducted A Fib. This excludes answer (c).

To establish the mechanism of any bradycardia, it is most important to observe what is going on in the atria. This is so that the 2 questions can be considered: is it a problem of atrial impulse generation (e.g. sinus problem) or is it a problem of conduction of atrial impulses to the ventricles? If one examines this ECG very closely, one can observe that:

- There is some discrete atrial activity but it is not normal. Clearly this is not fine A Fib.
- Using your calipers or scrap paper marking one small atrial impulse to the next (see P1 to P2…etc. in Fig. 1), one can "march" this interval to the end of the ECG confirming an almost regular atrial rhythm of 8 big blocks, around 35 bpm. In addition, analysis of the axis and morphology of these P waves indicates that they are not from the sinus node. (See "ECG: PQRST morphology – clues and tips" published in the January 2006 issue of SA Heart.) Thus, there is a problem of impulse generation with sinus arrest and this atrial rhythm is an atrial escape rhythm – a slow subsidiary rhythm that is a back-up if the sinus node pacemaker fails.
- A-V dissociation that is now obvious (see Fig. 1), poses the question: is there perhaps also an impulse conduction problem? Is there AV block, complete or high degree? The diagnosis of AV block can only be made if one finds an atrial impulse, P wave, which, despite being in a position that one would have expected to have conducted to the ventricles, did not. In this ECG, P1 conducted, as did P3 and P5. P2, P4 and P6 did not, but they would not be expected to conduct. These Ps occur at a time when the ventricles are refractory and when conduction to them is impossible. Thus, despite only intermittent AV conduction, nowhere can we find even a single P wave that should have conducted but did not. This excludes complete heart block and answer (b).

Why did P2, P4 and P6 not conduct? Because the ventricles had already depolarized. Where from? These QRS complexes not preceded by P waves are narrow and almost identical to the conducted QRSs and are due to a junctional escape rhythm that is slightly faster (6½ blocks = 45bpm) than the atrial escape rhythm noted above. Thus, after P1 conducts to the ventricle, the junctional escape focus is reset and fires before P2 occurs. P2 cannot conduct because the ventricles are refractory. The junction escapes again and probably gets ready to fire again, but this time P3 got in first and conducted to the ventricles and also reset the junctional escape focus. Both of these escape rhythms would have been suppressed and we would not have been aware of their existence if the sinus node had been functioning. The correct answer is (c).

What is wrong with this patient? Consideration of all the extrinsic causes of impulse generation problems is needed before diagnosing the intrinsic sick sinus syndrome (Table 1). The ECG is helpful: it is unlikely to be hyperkalaemia as the QRS complexes are so narrow; there is no evidence of hypothermia. A TSH level of 34 showed that the diagnosis is profound hypothyroidism. The treatment is not a pacemaker but slow and careful thyroid hormone replacement.

**Table 1.** Causes of failure or suppression of impulse generation

| Intrinsic | Extrinsic | |
|---|---|---|
| Sick sinus syndrome (tachy-brady syndrome; sinus pauses / arrest; chronotropic incompetence) | Hyperkalaemia | The "h"s |
| Idiopathic degenerative disease (fibrosis) | Hypothermia | |
| Coronary artery disease | Hypothyroidism | |
| Infiltration | Hypoxia | |
| | Hypercarbia | |
| | Head injury | |
| | Hyperautonomia | |
| | Drugs | |