ECG Quiz 15

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

ANSWER

The ECG shows a bradycardia with a regular ventricular rate of 42 bpm (7 QRS complexes in 10sec x 6).

To determine whether the mechanism of a bradycardia is either due to (1) failure of impulse formation e.g. sinus node dysfunction, or due to (2) failure of impulse conduction i.e. atrio-ventricular conduction block, one needs to analyse the ECG for atrial activity. (Table 1). Atrial activity can be observed, thus excluding (1). The observed P waves are clearly not associated with the QRS complexes. AV dissociation does not automatically mean complete heart block/ complete AV block. Only if P waves are seen to be in a position that could have been conducted but did not, can a diagnosis of complete heart block be made. (Figure 1).

TABLE 1: Bradycardia mechanisms: 2 major causes based on analysis of presence or absence of normal atrial activity.

A. Failure or suppression of impulse generation (i.e. sinus node problem)	B. Failure of impulse conduction (i.e. AV node problem) (2° or 3° AV Block)
Intrinsic	Degenerative
Sick sinus syndrome	Ischaemic
Extrinsic	Drugs
Hyperkalaemia	Functional
Hypothermia	Inflammatory
Hypothyroidism	Congenital
Hypoxia	
Hypercarbia	
Head injury	
Hyperautonomia	
Drugs	

In each complete heart block, there are 2 rhythms; one above the block and another below. Often, the ventricular rhythm receives all attention and the atrial rhythm is ignored. One needs to describe both rhythms in detail:

- The rhythm in the ventricles: as mentioned above, it is regular, at 42 bpm, with QRS duration of 100ms (2.5 x 40ms, the duration of a little block) which is the upper limit of normal in an adult. The QRS axis is normal at 75° with normal R wave progression in the chest leads and only a minor QRS abnormality of rSr' in V1. Thus, the escape rhythm in this patient with complete heart block is not ventricular in origin. This is a junctional escape rhythm.

- The rhythm in the atria: also regular; rate 15 x 6 = 90bpm, normal P wave axis of 60° suggesting that this is a normal sinus rhythm. The normal rate also indicates that despite the complete heart block, the patient was probably haemodynamically stable during the recording of this resting ECG.

Is there evidence of pacemaker activity?

Yes; associated with each P wave is a pacing spike, well seen in the bottom V1 rhythm strip. But these spikes follow the P waves. Thus, the atrial rhythm is not due to the pacing. The intrinsic atrial rhythm, which we have already assessed as being sinus in origin, appears to cause the pacing spikes. To "cause" the spike, the pacemaker must have first sensed the atrial activity which then triggered off a pacing spike. This pacing spike clearly does not capture any chamber. So, we have now determined that this cannot be a single chamber system programmed to AAI because sensing would have inhibited the pacing spike. (Figure 2). It must, therefore, be a dual lead or dual chamber system (sensing with one lead, and subsequent pacing with the other) with failure to capture. And the chamber that should have been paced/captured is the ventricle. Luckily, this patient had an escape rhythm; otherwise she would have had ventricular asystole and not returned for her follow-up visit!

What is the problem with the pacemaker?

The most likely problem 6 months after implantation is a raised ventricular pacing or capture threshold, possibly because of lead dislodgement or some fibrosis at its tip following inflammation which the lead evoked despite being steroid-eluting. This necessitates (1) a chest Xray to determine the lead position and to compare it with the position immediately after implantation and (2) checking the pacing threshold with a pacemaker programmer. Could it be any other pacemaker problem? It is not a pacemaker battery problem because normal sensing is occurring

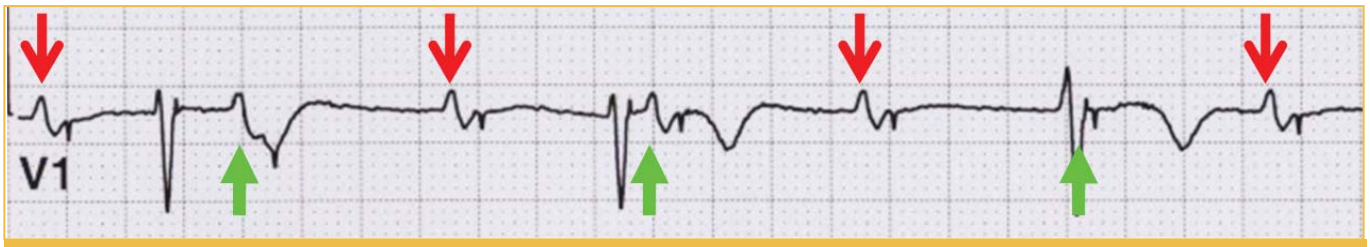

FIGURE 1: Complete heart block
The arrows mark the sinus P waves in the V1 rhythm strip. The green arrowed P waves physiologically cannot be conducted to the ventricles (i.e. a P wave soon after a QRS, or before the completion of a T wave can never normally be expected to be conducted). If these were the only P waves visible, and even though they are dissociated from the ventricular activity, a diagnosis of complete heart block would not be possible. It is only the presence of the red arrowed P waves which happen to be in positions that should have conducted, but did not, that proves there is complete heart block.

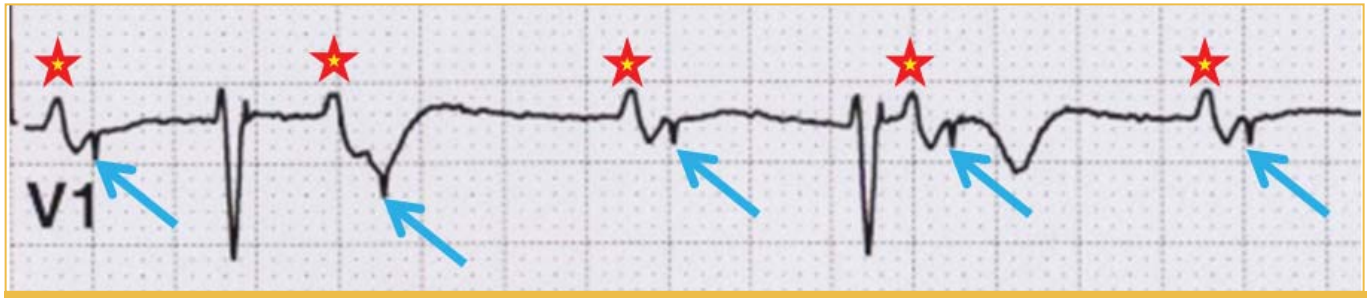

FIGURE 2: Evidence of pacing triggered by atrial sensing
There is evidence of spontaneous sinus rhythm (stars). The pacing spikes (arrows) are closely linked and follow the P waves. The constant relationship between P wave and pacing spike confirms that the pacemaker must be sensing the P waves. The pacing spikes are not followed by any depolarisation indicating failure to capture.

and spikes are being delivered appropriately albeit without capture. Could the lead have fractured? Unlikely, because this would have caused failure of pacing spikes to reach the heart and therefore to the ECG. Could the ventricular lead of a dual chamber pacemaker have dislodged and fallen into the atrium? This is a possibility. Obviously, the atria would then receive a spike via the ventricular lead soon after the sensed P wave and capture would not be physiologically possible. Could it be failure to capture by the V lead, dislodged or not, of a single chamber ventricular VVI pacemaker? No, because the pacing spikes are clearly related to the atrial activity – this needs sensing by a lead that triggers the pacing spike.

ECG and **QUESTION** on page 118

A chest Xray showed no dislodgement of the atrial and ventricular pacing leads as compared to immediately after implantation. The pacemaker programmer showed that the pacing lead impedance was normal, therefore not fractured. The ventricular pacing output had been set to 2.0 V. Testing revealed that the pacing threshold had risen and was now 2.5V. With the pacemaker reprogrammed to an output above this, ventricular capture resumed with a new ECG now showing atrial sensing and ventricular pacing. (Figure3).

Therefore the answer is: (e) a dual chamber pacemaker with sensing in the atrium and failed capture in the ventricle.

Other observations

The ECG with failed ventricular capture (on page 118) shows T wave inversion in V1 to V5, II and aVF. The original indication for pacing had been congenital complete AV block with mild bradycardia related symptoms. There was no history and any symptoms suggestive of ischaemic heart disease that could have caused these T inversions. Interestingly, the T inversion is limited to the leads that when V pacing capture occurs, have negative QRS complexes. Abnormal depolarisation causes abnormal repolarisation. This abnormal repolarisation with T wave inversion remains even after pacing has been stopped / or fails. This is T wave memory and may persist for weeks after chronic pacing.

Conclusions / Lessons

- Complete AV block can only be diagnosed if there is evidence of atrial activity that should have been conducted but was not: hence the pivotal role of identifying P waves or atrial rhythm.
- If pacing spikes are seen to be related to some preceding electrical activity, then there is evidence that sensing must be occurring.
- If failure of pacing capture occurs, one needs to consider and exclude:
  - lead dislodgement,
  - lead fracture (less likely)
  - increased pacing threshold which may be due to
    - fibrotic inflammatory reaction to the lead
    - myocardial infarction which happens to involve that region where the lead is situated,
    - drugs (such as newly initiated antiarrhythmics e.g. amiodarone) and
    - electrolyte disorders (e.g. hyperkalaemia).

If all reversible causes are excluded, increasing pacing output amplitude will solve the problem but at the expense of more rapid battery depletion.

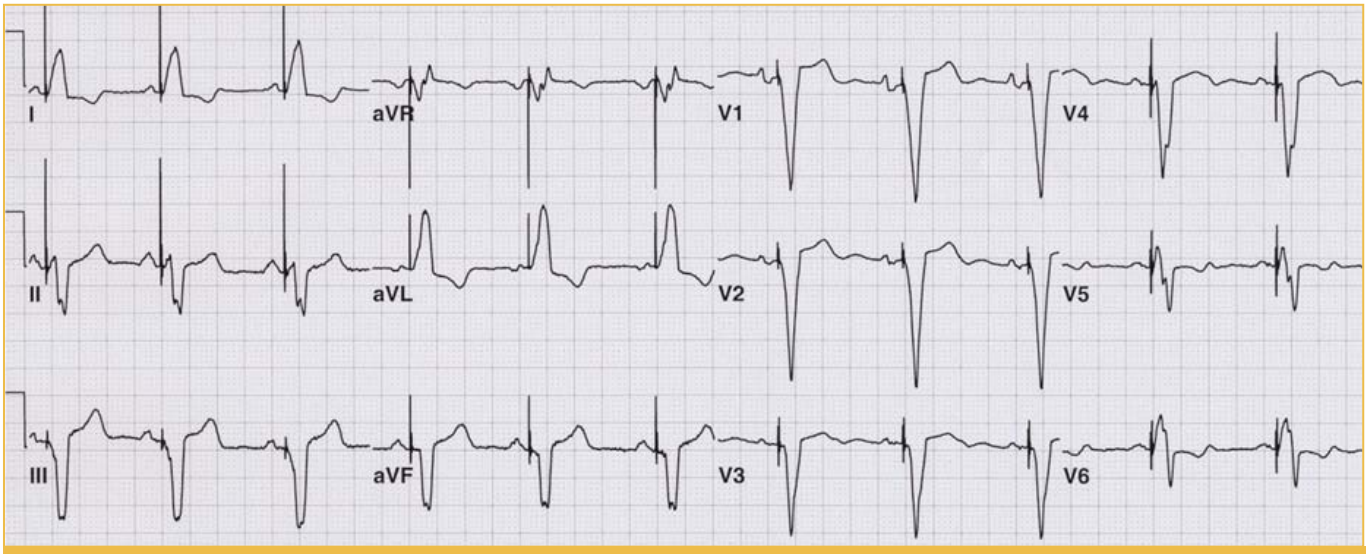

FIGURE 3: Normal dual chamber pacemaker function with A sensing and V pacing
Note the wide complex paced ventricular beats, the axis of minus 45°, and the left bundle branch block-like morphology typical of right ventricular apical pacing. The leads that have negative paced QRS complexes, are the ones in which when pacing does not occur, T waves are inverted. This is T wave memory.