ECG Quiz 60

CASSA
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ANSWER

OVERVIEW OF THE ECG

This ECG shows a regular, wide complex rhythm (QRS 200ms) with a ventricular rate of 84bpm. Ventricular pacing spikes are seen before each QRS compatible with a paced ventricular rhythm. P waves are visible in the ST segments best seen in II, III and aVF with a very long PR interval (almost 600ms).

MORE DETAILED ANALYSIS OF THE ECG

The paced QRS morphology and axis are the keys to determine the site of ventricular pacing.

This ECG shows a paced morphology with a dominant R wave in V1 to V3 (RSR pattern) and a QS pattern in I and V6. Depolarisation is therefore moving away from I and V6 (left sided leads) and towards V1, V2 and V3 (right sided leads). The paced QRS axis is -120 degrees. While the morphology of P waves is difficult to assess, they are most likely retrograde P waves (caused by the preceding paced QRS complexes with a fixed RP interval). This is VVI pacing with a very long PR interval (almost 600ms) and not DDD pacing. In DDD pacing, the P waves will be tracked by QRS complexes, usually with a programmed PR interval (between 120 and 200ms).

Conventional right ventricular (RV) apical pacing usually causes a QS or RS pattern in V1 and V2 (atypical LBBB-like morphology) with a QS pattern in V6 because depolarisation is moving away from V1, V2 and V6 (RV apical region) and superiorly to the left (with left axis deviation). Dominant R waves in V1 - V4 with an axis of -120 degrees is incompatible with RV apical pacing. In 8% - 15% of patients, a dominant R in V1 and V2 (atypical RBBB-like morphology) can occur with RV apical pacing, but the QRS complexes are always negative by V3.[1,2] Figure 1 shows an example of RV apical pacing with a dominant R in V1 and V2 with negative complexes in V3 - V6.

His bundle pacing involves pacing the His bundle only (selective His capture) or His bundle and local myocardium (non-selective His capture). Selective His capture usually produces a narrow QRS (similar to the conducted QRS) in the absence of bundle branch block with a normal QRS axis. Non-selective His capture produces a pseudo delta wave (due to local myocardial capture) with an initial widening of the QRS with a normal axis.

Left bundle branch (LBB) pacing is a relatively new form of pacing which involves pacing of the left main bundle and left septal myocardium. The pacing lead is positioned deep inside the interventricular septum 1cm - 1.5cm below the His bundle. Paced morphology is usually an atypical incomplete RBBB pattern (qR or Qr pattern) with a normal axis.

Conventional RV apical pacing, His bundle pacing and LBB pacing can therefore be excluded based on the QRS morphology and axis.

Cardiac resynchronisation therapy (CRT) is simultaneous RV and left ventricular (LV) pacing following a sensed or paced P wave (with a usually short PR interval). CRT-paced morphology can be highly variable between patients because of different positions of the RV and LV leads. A qR or Qr complex in lead I is usually indicative of CRT pacing with a QRS duration between 120ms and 200ms with a north west axis. While there are some features on this ECG compatible with a CRT pacing morphology, the very wide QRS and lack of DDD pacing are features against CRT pacing.

This ECG has all the features of LV pacing (VVI) only. The site of pacing appears to be from the inferior/lateral aspect of the left ventricle with the axis of -120 degrees. The very wide QRS suggests epicardial LV pacing rather than endocardial LV pacing.

The correct answer is e) Left ventricular pacing (VVI).

Isolated LV pacing is usually not a preferred site for standard pacing for atrio-ventricular block. This ECG was performed after a temporary pacing lead was inserted via the left subclavian vein. The temporary pacing lead was inadvertently malpositioned in the coronary sinus and was pacing the epicardial surface of the LV. Fluoroscopic images confirmed the lead tip to be positioned in the coronary sinus in the PA and lateral projections (Figures 2A and B). The anatomical chambers of the heart are superimposed on a standard PA and lateral X-ray for reference (Figure 3).

The site was confirmed with a routine echocardiogram which confirmed the lead tracking into the coronary sinus. This temporary lead was left in-situ as it had stable, adequate pacing thresholds until a permanent pacemaker was inserted the following day.

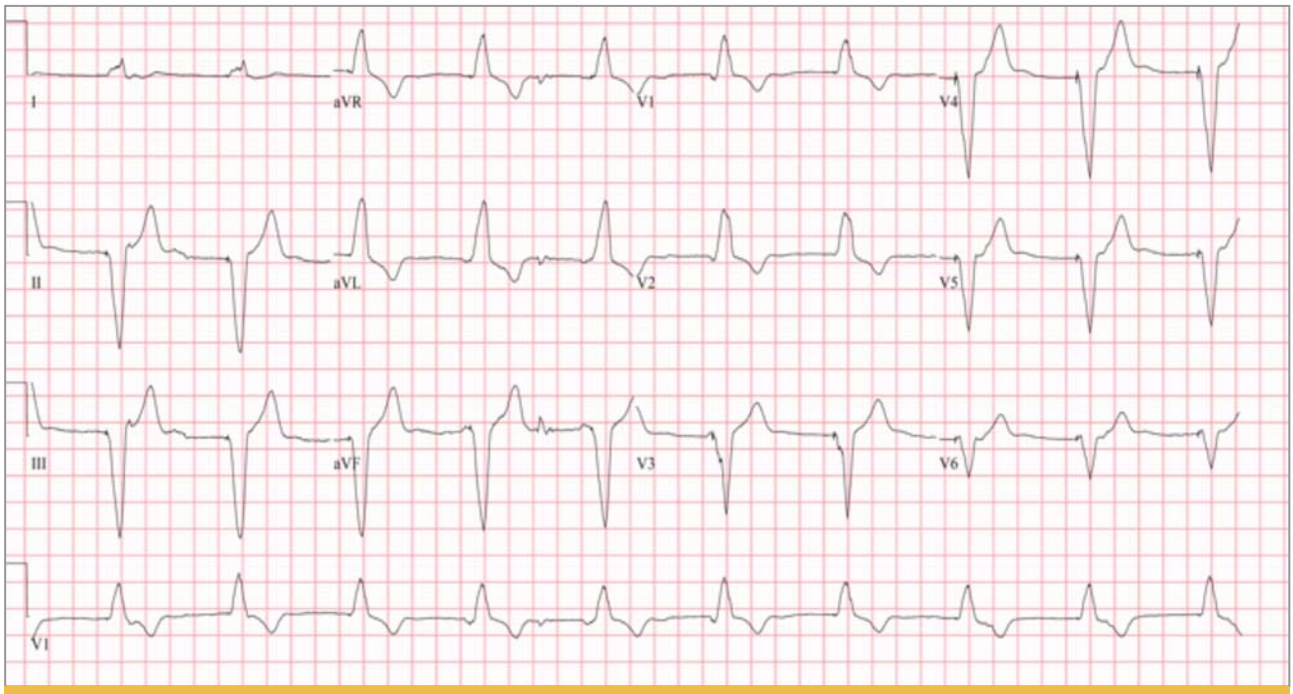

FIGURE 1: ECG shows an example of right ventricular apical pacing with a dominant R wave in V1 and V2, but negative complexes in V3 - V6. A dominant R in V1 can occur with RV apical pacing in 8% -15% of patients (see text for details).[1,2]

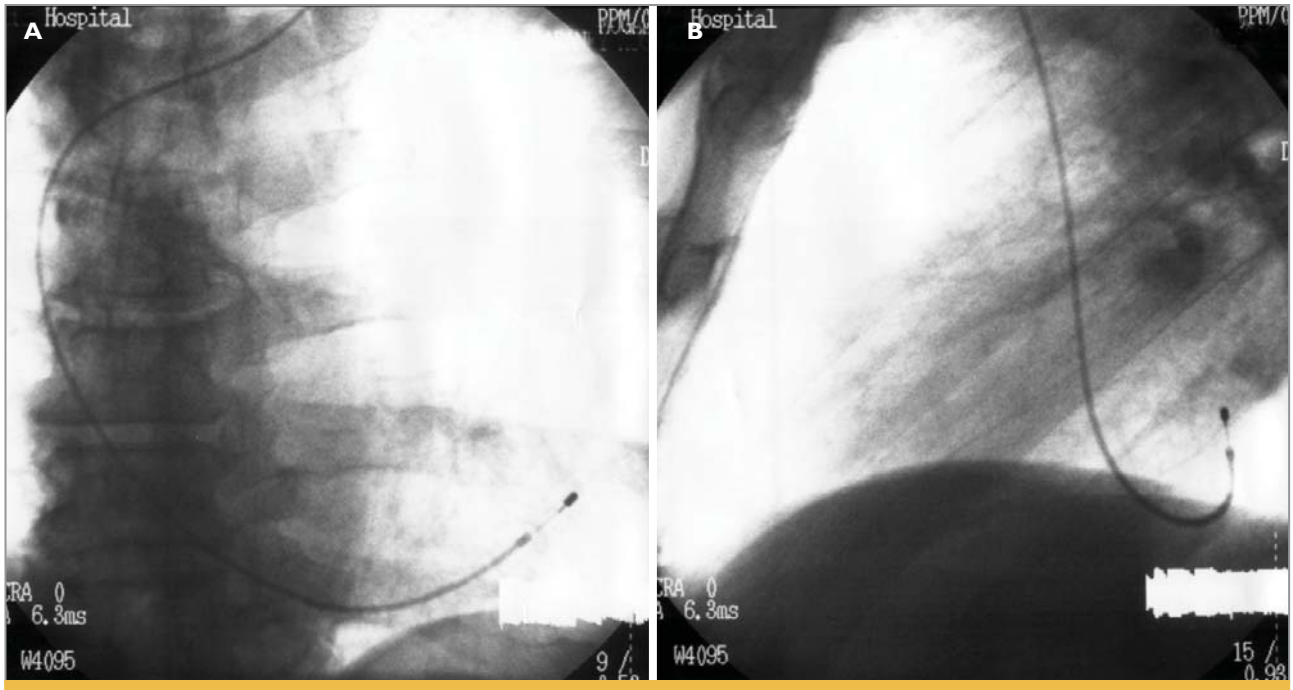

FIGURE 2: Fluoroscopic images showing the temporary pacing lead is in the coronary sinus. Note the lead tip is positioned at the left heart border shadow in the PA projection (2A) and the lead is tracking posteriorly on the lateral projection (2B).

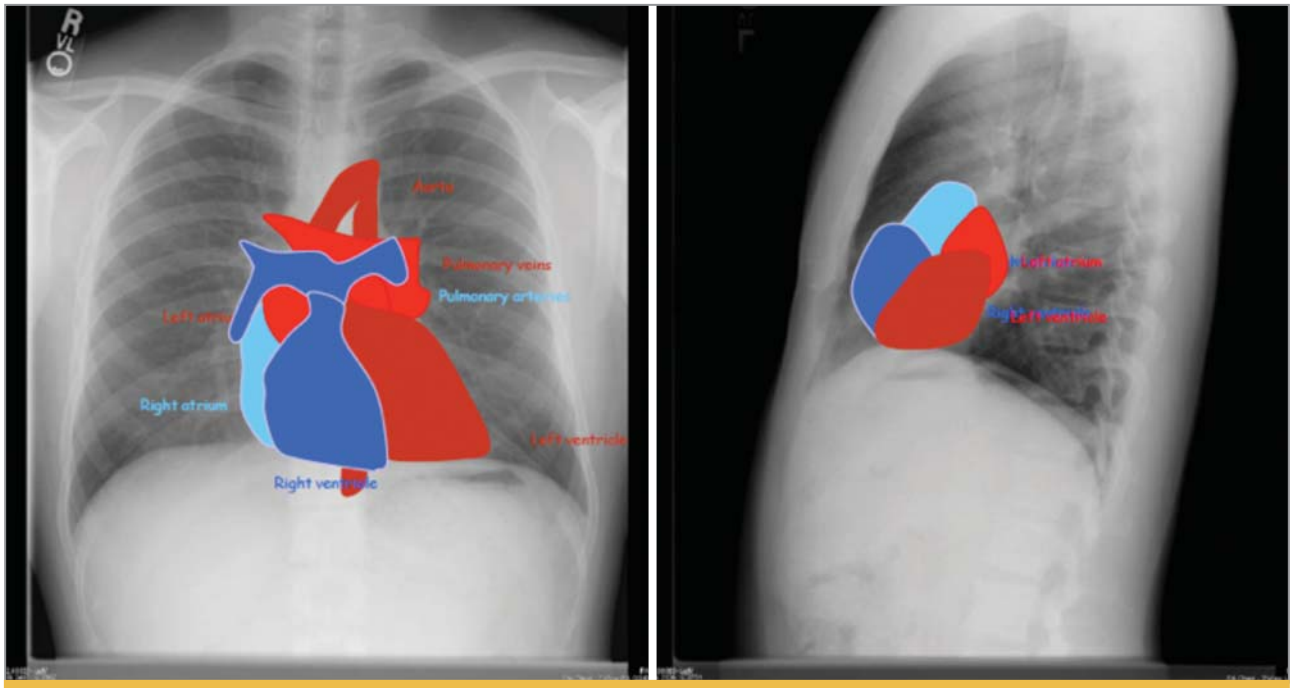

FIGURE 3: Anatomical chambers and great chambers of the heart superimposed on a standard PA and lateral chest X-ray.

DISCUSSION

Although malpositioning a temporary pacing lead in the coronary sinus with subsequent LV pacing is usually not associated with any major sequelae, the clinician should be able to prevent and recognise this problem. This is especially important when implanting the permanent pacing lead.

Permanent LV or coronary sinus pacing is rarely a chosen site for bradycardia pacing (in the absence of CRT), usually because of its added complexity, higher pacing thresholds and phrenic nerve stimulation. On rare occasions, LV pacing via the coronary sinus can be a chosen site for selected patients e.g. a patient with a prosthetic tricuspid valve when positioning a lead across the tricuspid valve is not possible e.g. mechanical tricuspid valve or when RV lead placement is not possible because of high pacing thresholds.

Clinicians should be aware that transvenous and permanent pacing leads intended for the RV can easily be malpositioned in the coronary sinus as in this case. Care should be taken to ensure that the pacing lead has crossed the tricuspid valve during lead positioning. This can be achieved by first positioning the lead in the pulmonary artery/RV outflow tract, then withdrawing and allowing the lead to fall into the apical or septal region, watching for ventricular ectopics during the process. Absence of ventricular ectopics may suggest the lead is in the coronary sinus. A left or right lateral fluoroscopic image can be helpful to ensure the lead is tracking anteriorly as the RV is an anterior structure. If a 12 lead ECG is connected at the time of implant (this is not often the case in most cath labs), V1 pacing morphology should be carefully inspected for an atypical LBBB morphology of RV pacing. However, in 8% - 15% of patients, RV apical pacing may produce a dominant R wave in V1 and V2.[1,2] This can be due to clockwise rotation of the heart relative to the V1 and V2 electrodes and if V1 and V2 are incorrectly placed in second-intercostal space.

Other LV lead malpositions have been reported. A pacing lead can perforate the interventricular septum or track across a PFO/atrial septal defect and pace the endocardial surface of the LV. Perforation of the free wall of the RV with the lead tip positioned over the epicardial surface of the LV in the pericardial space can occur. A pacing lead can also enter the subclavian artery and pace the endocardial surface of the LV with the lead across the aortic valve. Prompt recognition of a malpositioned lead is essential, preferably at the time of implant, or in the immediate post-operative period, so that urgent lead repositioning can be performed.

A routine post pacemaker ECG is essential and the pacing morphology and axis can be the first clue of a malpositioned lead. A standard chest X-ray with or without an echocardiogram or CT chest can also be performed to confirm the site of pacing.

SUMMARY

A routine ECG must be performed after every pacemaker to confirm the pacing lead position.

Malpositioning of the pacing lead in the coronary sinus is not uncommon.

Isolated LV pacing usually causes a very wide atypical RBBB paced morphology with a dominant R in V1, V2 and V3 which can extend to V4 - V6 with an apical position.

In 8% - 15% of patients, pacing from the RV apex can produce an atypical RBBB paced morphology with a dominant R in V1 and V2 with negative complexes in V3 - V6.

Conflict of interest: none declared.

REFERENCES

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