

Systematic analysis of the ECG shows:

1. Rate and regularity: regular with a ventricular rate of 72 bpm.
2. Atrial activity: Discrete P-waves are present and occurring regularly although some are obscured by the QRS complexes. The probable atrial rate is $14 \times 6 = 84$ bpm (Figure 1). Clearly, there is dissociation between the atrial and ventricular complexes and although some P-waves appear just before a QRS, there is no consistent P-QRS relationship and no alteration in the regularity of the ventricular rhythm, nor change in QRS morphology. Thus, there is no evidence of atrio-ventricular conduction giving a diagnosis of complete AV heart block. Analysis of the P-waves (best seen just before the 2nd and 6th QRS complexes) shows that they are likely to be of sinus node origin (being positive in Leads I, II and aVF and with an axis of 30°).
3. The QRS complexes are abnormal:
 - a. QRS width: 180ms ($4\frac{1}{2}$ blocks \times 40ms per block). Sharp spikes are visible at the beginning of most QRS complexes. These are pacemaker spikes. (Note that there are spike-like recording artifacts when a lead changes e.g. V3 to V6 and seen just left of V6).
 - b. QRS axis: this is very important. The QRS in aVF is almost isoelectric; the lead perpendicular to this is Lead I which

shows deeply inverted QRS. As the QRS is slightly positive in aVF, the axis is just short of 180° , viz. 165° .

- c. QRS morphology: In the chest leads, do these abnormal QRSs look more like Right Bundle Branch Block (RBBB) or Left (LBBB)? The QRSs are not typical of either; however, being predominantly positive in V1, they are reminiscent of RBBB.
4. ST segments and T-waves: Due to the abnormal ventricular depolarisation, repolarisation must be abnormal too. The T-waves and ST segments are expected to be opposite to the polarity of the QRS: thus, in Lead I, the T-waves are positive and ST upsloping; in V1, with the dominantly positive QRS, the ST segments slope down to inverted T-waves. Upright T-waves here would be distinctly inappropriate, suggesting changes not just secondary to abnormal depolarisation but the presence of other pathophysiology such as ischaemia.
5. QT interval: In V1 this is easily measured: 640ms (3.2 big blocks \times 200ms) which is markedly prolonged. This must be evaluated together with the wide QRSs, which are almost double normal. Unless the QRS duration is normal, the permitted QT interval is unknown.



FIGURE 1: AV dissociation in Lead VI

Complete Heart Block is present as evidenced by AV dissociation and the observation of P-waves in positions that would have been expected to conduct but did not. The rhythms on both sides of the block can be described:

1. The atrial rhythm is regular (solid red arrows) at 84 bpm, and of sinus origin as determined from the limb leads. The dotted red arrows indicate probable atrial activity masked by QRS complexes.
2. The ventricular rhythm is regular at 72 bpm with wide complexes. The R is dominant. Although this is a paced rhythm, it is not abnormal to see almost no pacing or only intermittent pacing spikes (blue arrows).

In summary, there is complete AV heart block. Whenever this diagnosis is made, 2 rhythms, i.e. above and below the block must consciously be described. Here we have:

- Normal sinus rhythm, above and
- A ventricular rhythm below.

The ventricular rhythm can be due to either intrinsic depolarisation or paced. They may appear identical. Obvious clues to this being a paced rhythm are the pacing spikes and the rate of 72 bpm. If the pacemaker spikes had not been visible and the rate was slower e.g. 30-40 bpm, it would have been impossible to account for the origin of the QRS; an intrinsic ventricular escape rhythm would have been diagnosed. Please note that a 12 lead ECG does not always show pacemaker spikes, as in VI and the variable size in Lead III on this ECG. Absence of pacing spikes throughout, or intermittently, and variability do not at all reflect pacemaker dysfunction but merely ECG recording or resolution "difficulty" of these sharp short signals. (Remarkably, the duration of most pacing spikes is 0.5ms; i.e. one small block, 40ms, would fit 80 of these spikes if placed together!).

With this ventricular rhythm (whether paced, as it is in this patient, or intrinsic, as it could have been, even at this rate due to accelerated idioventricular rhythm, which is not uncommon in the acute infarct setting or during reperfusion), most features of myocardial ischaemia or infarction are masked. It is only when ST and T-waves are not appropriately abnormal, that mechanisms other than abnormal depolarisation can be inferred.

The scenario in this patient is quite common: he presented with complete heart block probably due to degenerative disease, with no evidence of myocardial infarction, normal renal function and potassium level. (Hyperkalaemia itself would not have been a primary cause of complete heart block but should be excluded in

anyone with symptomatic bradycardia with wide QRS complexes especially when no atrial activity can be detected.) He was taken to the cardiac cath lab and a simple ventricular permanent pacemaker (VVI) was implanted. So, the question is whether everything is now OK?

It is important that a 12 lead ECG be done after every permanent pacemaker implantation. A single channel ECG strip from the lab or afterwards from the bedside monitor is totally inadequate. Yes, it may show pacemaker spikes and the appropriate paced heart rate. However, it may miss the QRS axis and morphology.

The 12 lead ECG after a standard simple ventricular pacemaker implantation when the RV lead is typically placed in the RV apex will show: (Figure 2 and Figure 3)

- A QRS axis that is directed superiorly: minus 45° to -90°, because of the apical situation of the lead; and
- QRS morphology that is most reminiscent of LBBB with QRS negative in VI and most of the anterior chest leads. V6 will usually be positive except in big hearts in which case the lateral lead may need to be placed further round the side of the chest. However, leads I and aVL can be regarded as high left lateral "extensions" of the series of 6 chest leads, and should be positive in a pattern of LBBB-like morphology. Why LBBB-like? Because, with the pacing lead in the right ventricle, this ventricle, just like in LBBB with its functioning right bundle branch, activates first and is followed by slow and late depolarisation of the left ventricle.

This patient's post pacemaker implantation ECG shows neither of these 2 ECG findings. The axis is markedly to the right and the QRS is more like RBBB. The pacemaker lead is clearly not in the right ventricular apex and is pacing the left ventricle. A chest Xray

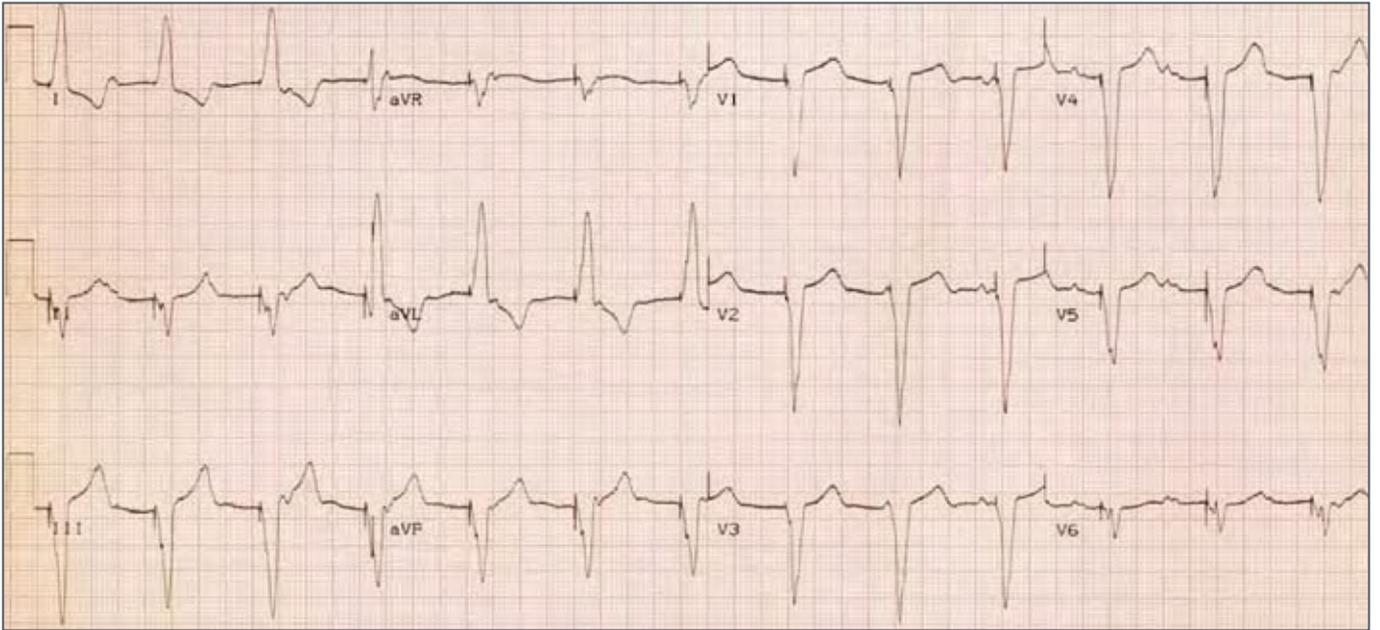


FIGURE 2: Right ventricular apical pacing

This 12 lead ECG shows complete heart block with AV dissociation, normal sinus rhythm and a paced ventricular rhythm. The latter is typical of RV apical pacing with an axis of minus 45 and “LBBB-like” morphology with negative QRS in V1 and, although unimpressive QRS in V6, typical “LBBB-like” QRS in Leads I and aVL.

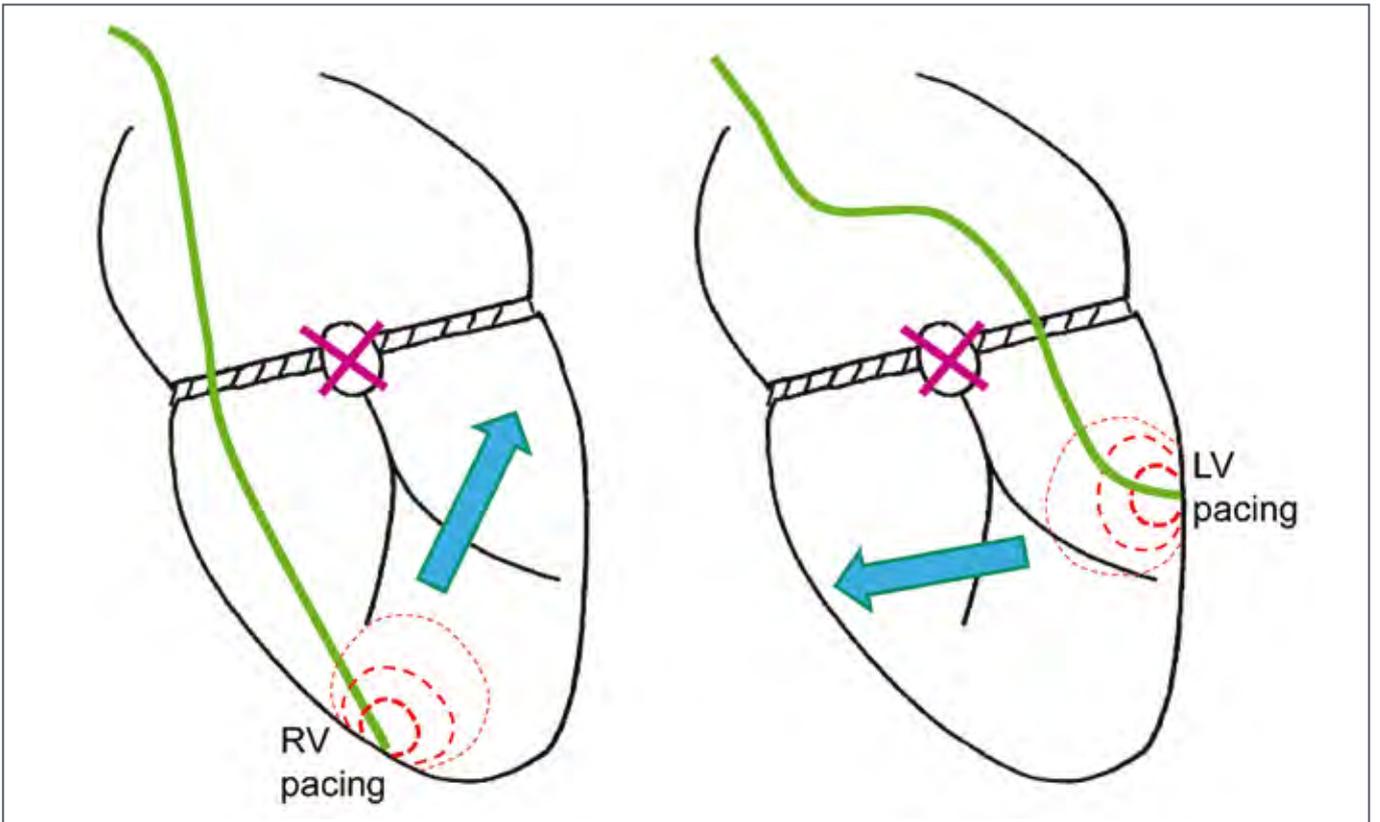


FIGURE 3: Right ventricular vs Left ventricular pacing

RV pacing results in a left or superior QRS axis with LBBB-like morphology: rapid and early right ventricular depolarization and late activation of the left ventricle. The converse applies in LV pacing with QRS axis to the right or in the North-West quadrant and RBBB-like QRS morphology.

may help to confirm this but cannot be relied on alone. So what is the problem pacing the left ventricle?

One needs to consider how, when the intention had been to pace the right ventricle, it is that the left ventricle can be paced: In order of decreasing frequency:

- Correct RV apical pacing but problem with the ECG recording: misplaced limb ECG electrodes or chest leads not far enough to the left. Misplaced leads giving a bizarre QRS axis would also affect the P-wave axis: in this case, the P-waves are normal confirming that incorrect lead placement is not the "cause".
- Perforation of the RV inferior wall or apex, with the RV lead in the pericardial space and pacing the left ventricle from its epicardial aspect.
- Inadvertent crossing of the interatrial septum via an atrial septal defect (ASD) or patent fossa ovalis (PFO), left atrium, mitral valve, left ventricular cavity to the LV apex.
- Passage of the pacing lead through the coronary sinus to a cardiac vein: the posterior or middle cardiac vein placement may look very similar to RV apex; or left lateral cardiac vein which is used in biventricular cardiac resynchronisation pacing.
- Perforation of the RV septum with the pacing lead, and pacing the LV directly; rare, because the septum is thick, unless diseased or infarcted.
- Crossing of a ventricular septal defect (VSD).

The relevance is that RV perforation may result in cardiac tamponade. LV endocardial pacing (via ASD, VSD or septum) may be associated with thromboembolism related to the lead and stroke, and if discovered late, may require lifelong anticoagulation. Cardiac vein pacing may be associated with pacing threshold problems. Thus, as soon as recognised, but only when still in the acute peri-implant situation, the ventricular pacing lead needs to

be repositioned correctly in the right ventricle. This patient was taken back to the lab and the lead that had crossed a PFO was repositioned. The subsequent ECG confirmed correct placement in RV (see Figure 2.) Thus, the correct answer is: (e).

This incorrect LV pacing can be avoided by taking care during the implantation procedure, swinging the Xray C arm to lateral to confirm the RV apical position which is very anterior and just behind the sternum, and checking afterwards and before discharge of both chest Xray and the 12 lead ECG.

CONCLUSIONS / LESSONS

- Whenever Complete AV Heart Block is diagnosed, both rhythms on either side of the block must be analysed and described.
- A 12 lead ECG of the paced rhythm is compulsory after implantation of a pacemaker or arrhythmia device. If the pacing is intermittent, the pacemaker should be programmed or magnet applied so as to record the paced 12 lead ECG.
- Knowledge of the QRS axis and morphology of expected paced rhythms is obligatory if pacing procedures are being done.
- RV apical pacing typically gives a left or superior axis and a LBBB-like QRS morphology.