

OVERVIEW OF THE ECG

The rhythm is regular at 60bpm. The QRS complexes are narrow and are preceded by P waves.

MORE DETAILED ANALYSIS OF THE ECG

The rhythm is sinus with a P wave axis of about $+60^\circ$, with normal P wave morphology. The normal P axis excludes mirror image dextrocardia. In this condition, the sinus node is in the usual position around the entrance of the superior vena cava, but this is now on the left side. Activation of the atria therefore proceeds from left to right and downwards. The P wave axis changes from about $+60^\circ$ to $+120^\circ$. The P waves remain upright in II and III, but are inverted in I and equiphasic, rather than negative, in aVR (Figure 1). Similarly, the most common lead misplacement (switching left and right arm electrodes) is excluded. That results in negative P waves in I and positive or equiphasic in aVR.

The normal PR interval (140ms) and QRS duration (90ms) exclude Wolff-Parkinson-White pattern.

There is right axis deviation, almost $+150^\circ$ and the R wave in V1 is dominant. There is late transition in the precordial leads. This pattern is usually due to right ventricular hypertrophy (RVH). However, Lead I and aVL consist of deep Q waves which are not usual for RVH. Figure 1 shows an example of RVH with a similar QRS axis, but Leads I and aVL consist of rS waves. The other feature against RVH is the positive T waves in V1 to V3, with T wave inversion in V5 and 6.

Left posterior fascicular block (LPFB) cannot be distinguished from RVH with certainty on a single ECG. Absence of a dominant R in V1 does not confidently exclude RVH. If there is no clinical or echocardiographic evidence of RVH, LPFB can be diagnosed if no other cause of right axis deviation is present. LPFB is usually associated with an rS pattern in I and AVL and a qR pattern in III and aVF. Isolated LPFB is rare, as it is usually a component of bifascicular block – right bundle branch block (RBBB) with otherwise unexplained right axis deviation. In the absence of RBBB, LPFB can be diagnosed if it is transient or develops immediately after an event, such as cardiac surgery or myocardial infarction, without RVH. In the example of isolated LPFB shown in Figure 1, an ECG done 3 days later showed left axis deviation due to left anterior fascicular block.

The deep Qs in I and aVL are compatible with infarction of the lateral left ventricular wall. The loss of lateral forces results in right axis deviation. While one might expect a pathological Q wave in V6, this is not always the case. The R waves get smaller from V4 to V6, consistent with loss of lateral LV forces.

The tall initial R wave in V1, together with an upright T wave, is characteristic of posterior myocardial infarction, although this enduring terminology has been challenged⁽¹⁾ by anatomic studies derived from cardiac magnetic resonance imaging, which demonstrate that this pattern is produced solely by lateral wall infarction, caused by occlusion of a nondominant left circumflex artery or of its marginal branch.

Figure 2 gives examples of the main mechanisms⁽²⁾ of a dominant R in V1 (R:S ratio >1). Most are easily excluded in this ECG. RBBB does not alter the early part of the QRS, which reflects normal left ventricular depolarisation; the delay is confined to the latter part of the QRS. The rhythm is clearly sinus, not ventricular, and the QRS is not pre-excited. Note the difference between the pattern of posterior infarction and that of severe RVH, in which the T wave is invariably inverted, opposite to the main QRS deflection, whereas the T wave in posterior infarction is upright, concordant with the QRS. This pattern is an inverted image of a lead placed posteriorly over the site of infarction, which would show a Q wave and inverted T.

The correct answer is therefore (b): posterolateral myocardial infarction.

LESSONS AND CONCLUSIONS

- Right axis deviation can be due to a variety of different mechanisms. It is important to analyse the whole ECG with these possibilities in mind.
- Posterior myocardial infarction commonly accompanies inferior infarction, which may result in left axis deviation. In this case, however, the infarct involved the posterior and lateral walls, causing right axis.
- A dominant R in V1 can also be due to a number of mechanisms. Right ventricular hypertrophy and healed posterior infarction produce similar QRS patterns, but can be distinguished by their opposite effects on T wave polarity.

Conflict of interest: none declared.

REFERENCES

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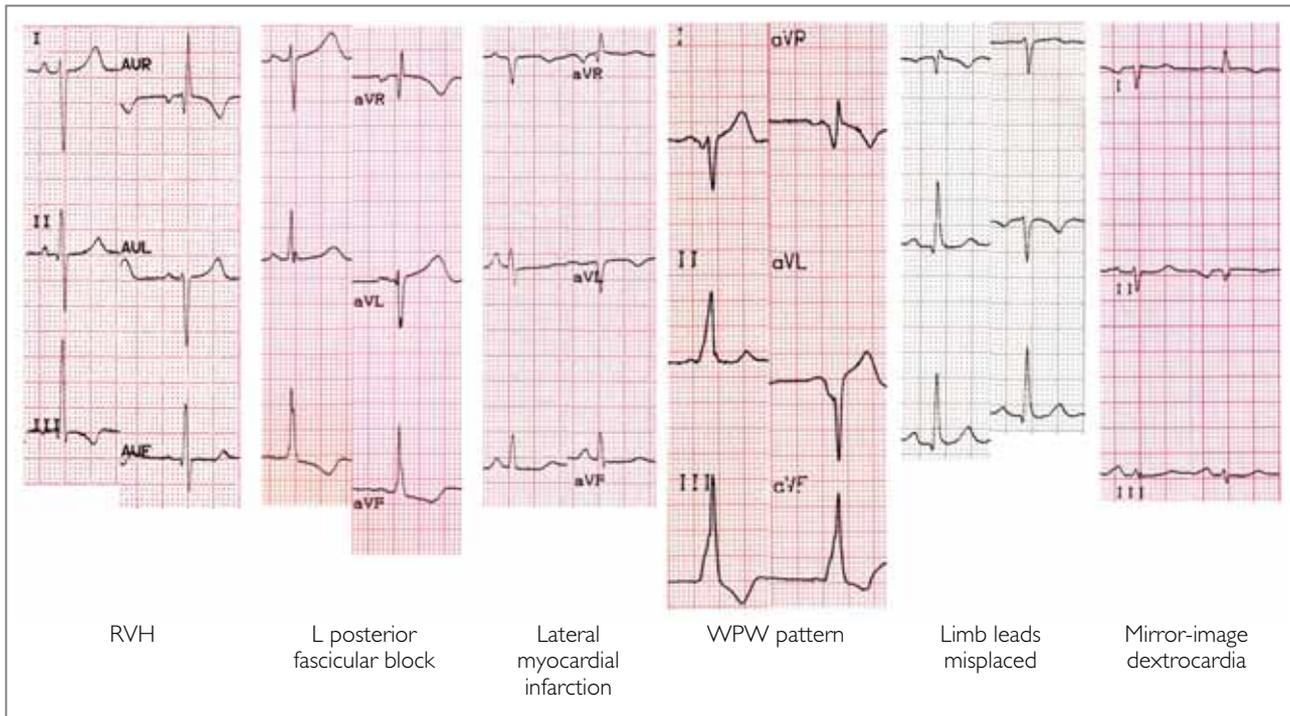


FIGURE 1: Mechanisms of right axis deviation.

RVH: right ventricular hypertrophy, WPW: Wolff-Parkinson-White.

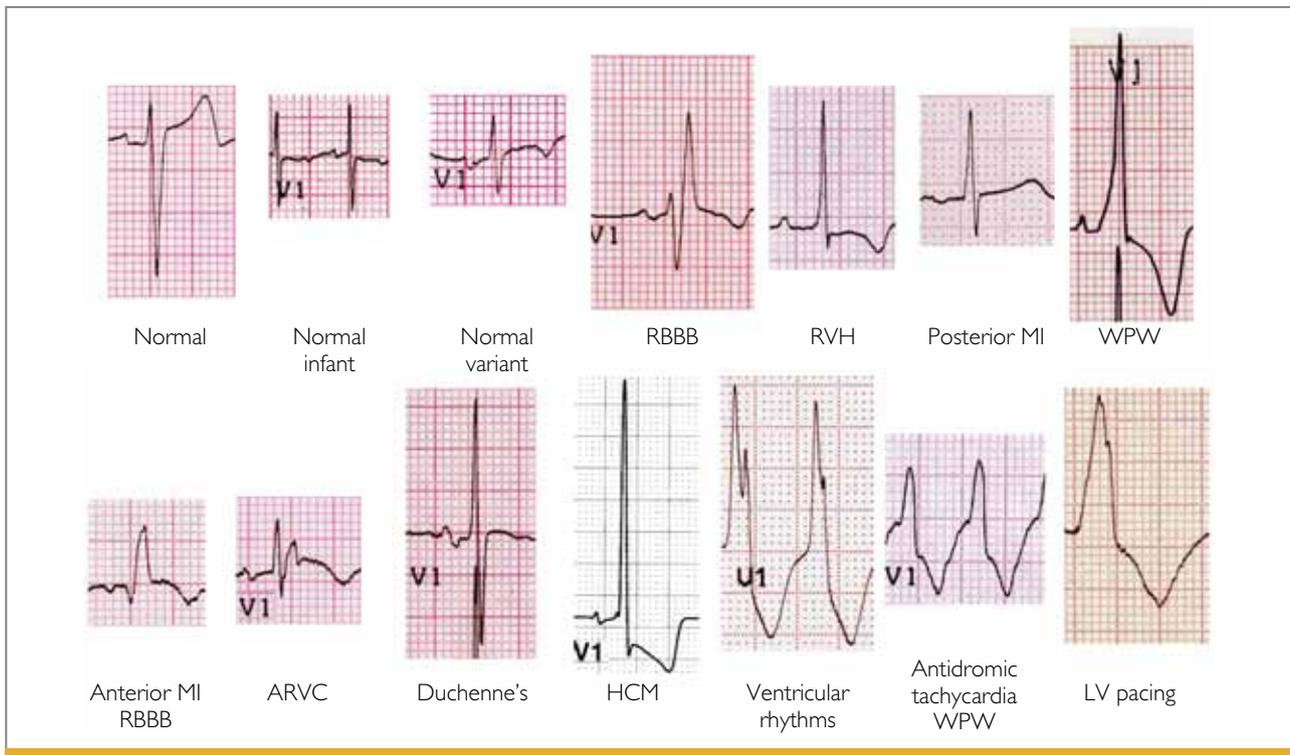


FIGURE 2: Mechanisms of a dominant R in V1 (R:S>1).

RBBB: right bundle branch block, RVH: right ventricular hypertrophy, MI: myocardial infarction, WPW: Wolff-Parkinson-White, ARVC: arrhythmogenic right ventricular cardiomyopathy, HCM: hypertrophic cardiomyopathy, LV: left ventricle.

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