

1: OVERVIEW OF THE ECG

The rhythm is regularly irregular with a ventricular rate of 84bpm. There is group beating – 4 beats followed by a pause, repeated. There are 2 different QRS morphologies – one narrow and the other wide.

Group beating occurs with a number of different mechanisms. Common causes include:

- Ventricular or atrial bigeminy or trigeminy
- Mobitz type I (Wenckebach) AV block

MORE DETAILED ANALYSIS OF THE ECG

The wide QRS complexes are premature and follow the narrow ones with 2 different coupling intervals. The QRS before the pause follows a slightly shorter R-R interval (270ms) than the one which appears to be sandwiched between 2 narrow complexes (300ms).

Premature wide QRS complexes may originate in the ventricles (PVCs), the atrium (PACs) or the AV junction, conducted with bundle branch block. To determine their origin, examine the morphology of the QRS in the chest leads and decide whether or not they are compatible with either left, or right, bundle branch block.

In this ECG, the wide complexes resemble left bundle branch block (LBBB) – negative in the right chest leads and positive in V4 - V6. While the QRS in V6 looks like typical LBBB, this can be very misleading. Look at the initial R wave in V1 - V3. It is more than 50ms wide and the timing from the onset to the nadir of the S wave is 100ms. The r wave should be less than 40ms and the onset to nadir less than 70ms (Figure 1). There is therefore no evidence of normal (rapid) right ventricular activation, which is a sine qua non for diagnosis of LBBB, as right ventricular activation must be normal for conduction to occur. LBBB is therefore excluded which means that the wide complexes cannot be conducted from the atrium or the junction, and must arise in the ventricles. In addition, no P waves are visible before the wide complexes. This, however, is unreliable as the P could be hidden in the preceding T wave. The distinction depends on the QRS morphology.

The pattern is one of unifocal ventricular bigeminy, but is unusual in that only every second PVC is followed by a compensatory pause. The others are interpolated, i.e. occur between 2 conducted QRS complexes without interrupting their rhythm.

Further inspection reveals a P wave before each narrow QRS, but the PR interval following the PVC is longer. This is because the PVC conducts retrogradely and penetrates the AV node sufficiently to prolong conduction of the succeeding P wave without blocking it completely (Figure 2). The subsequent

PVC occurs 30ms earlier and penetrates the AV node more deeply, sufficient to completely block the subsequent P wave. This P wave is concealed in the T wave. The result is a fully compensatory pause which is commonly seen with PVCs.

While (a) is correct, the best answer is therefore (b): Sinus rhythm with interpolated and non-interpolated PVCs.

None of the other options is possible, because the wide complexes are not compatible with LBBB.

Mobitz type I (Wenckebach) AV block does cause group beating, but the mechanism of the change in PR interval and the non-conducted P is explained in Figure 2 and is related to the PVCs, not to disordered AV conduction.

2: MANAGEMENT OPTIONS

Had this been Wenckebach AV block, permanent pacing should be considered, but it is not indicated for the rhythm occurring in this patient.

While a Class I or Class III antiarrhythmic drug may suppress the PVCs, there is no indication for their use in this patient who is asymptomatic. Antiarrhythmic drugs are of limited efficacy and carry significant risks, particularly in patients with ischaemia or other structural heart disease. The landmark Cardiac Arrhythmia Suppression Trial (CAST) demonstrated the lethal properties of Class Ic antiarrhythmics (encainide, flecainide and moricizine) in survivors of myocardial infarction, in which the total mortality of those on treatment was 2.5 fold those on placebo, with 3.6 times as many arrhythmic deaths.⁽¹⁾ Similar

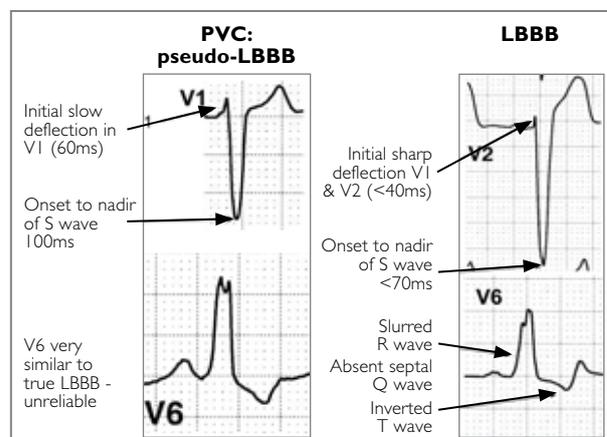


FIGURE 1: Features which distinguish true left bundle branch block from the pseudo-LBBB pattern of the premature ventricular complexes.

adverse effects on survival were shown in the SWORD trial, using the Class III agent d sotalol.⁽²⁾ Numerous other studies attest to the risks associated with the use of antiarrhythmics, including observations on cohorts of patients on these drugs in atrial fibrillation trials.⁽³⁾

The prognosis of PVCs depends on the company they keep. In people with otherwise normal hearts, they appear to have no adverse effect on survival, whereas frequent PVCs are markers for poorer outlook in survivors of myocardial infarction.⁽⁴⁾ In the 1970s and '80s, this led to widespread attempts to suppress them with Class I antiarrhythmic agents, until the CAST demonstrated the harm that this caused.

Before deciding whether to reassure him, you need to look for other evidence of heart disease. The ECG is a good place to start. Besides the arrhythmia, is there evidence of any other abnormality?

The conducted QRS complexes are of normal width (90ms) and axis (+60°). There are no pathological Q waves and the R wave progression in the chest leads is normal. However, there is unusual fractionation of the QRS complexes, best seen in leads III, aVL, aVF and V3. There is also a suggestion of terminal delay in V2. The T wave in V1 is inverted, which may be normal. There is no T wave inversion in V2 - V6 and there is no Epsilon wave. The QT interval is normal.

The morphology of the PVCs is that of pseudo LBBB, with an inferior axis of +80°, suggesting an origin in the right ventricular outflow tract. There is notching of the QRS complexes in the inferior leads which suggest a free wall origin. PVCs from this site are usually benign and not associated with structural heart disease. However, PVCs arising from the right ventricle can be due to Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC), which is due to a genetic abnormality of the desmosomal proteins which results in fibro-fatty degenera-

tion of the right ventricle. While it does not usually cause heart failure in the early stages of the disease, it is often complicated by ventricular tachycardia which may be life threatening. While fractionation of the QRS and the pattern in V2 are not specific and may be normal,⁽⁵⁾ they raise the suspicion of underlying scarring in the ventricles. Structural heart disease, like ARVC, needs to be considered in this patient.

Even in the absence of the QRS abnormality, it would be wise to do further tests before concluding that all is well. An echocardiogram is a first step. In view of the suspicion of ARVC, pay particular attention to the right ventricle, although subtle changes in RV function and morphology can be missed in the early stages of the disease. Cardiac MRI may be needed to accurately look for areas of akinesia and dyskinesia and to measure RV function and RV volumes. Left ventricular function is likely to be normal, but very frequent PVCs may, over time, cause reversible left ventricular dysfunction ("premature ventricular contraction-induced cardiomyopathy").

Consider a signal averaged ECG to look for late potentials, although the test result may be inaccurate in the setting of frequent PVCs. This test uses computer signal averaging to eliminate random noise and allows for much greater amplification. This may reveal low amplitude electrical activity after the main QRS complex which correlates with slow conduction and the potential for re-entrant ventricular tachycardia (Figure 3).

A 24 hour Holter monitor should also be done to see if any episodes of ventricular tachycardia occur and to quantify the burden of PVCs. While the absolute PVC burden required to cause LV dysfunction is unclear, a cardiomyopathy is unlikely to develop in the setting of <15% PVC burden/24 hours. In patients with higher PVC burdens, serial measurements of LV function may be required to monitor for the development of LV dysfunction.



FIGURE 2: Ladder diagram depicting the retrograde penetration of the AV node from the PVCs. The PVC before the pause occurs slightly earlier and penetrates more deeply into the node, resulting in failure of that P wave to conduct. The result is a pause in which the interval between the 2 sinus beats approximates 2 R-R intervals, i.e. fully compensatory. It is actually slightly shorter, because of the change in PR intervals. The later PVCs cause delay, but not complete blockade of conduction from the atrium, resulting in the sandwiching (interpolation) of the PVC between 2 sinus beats.

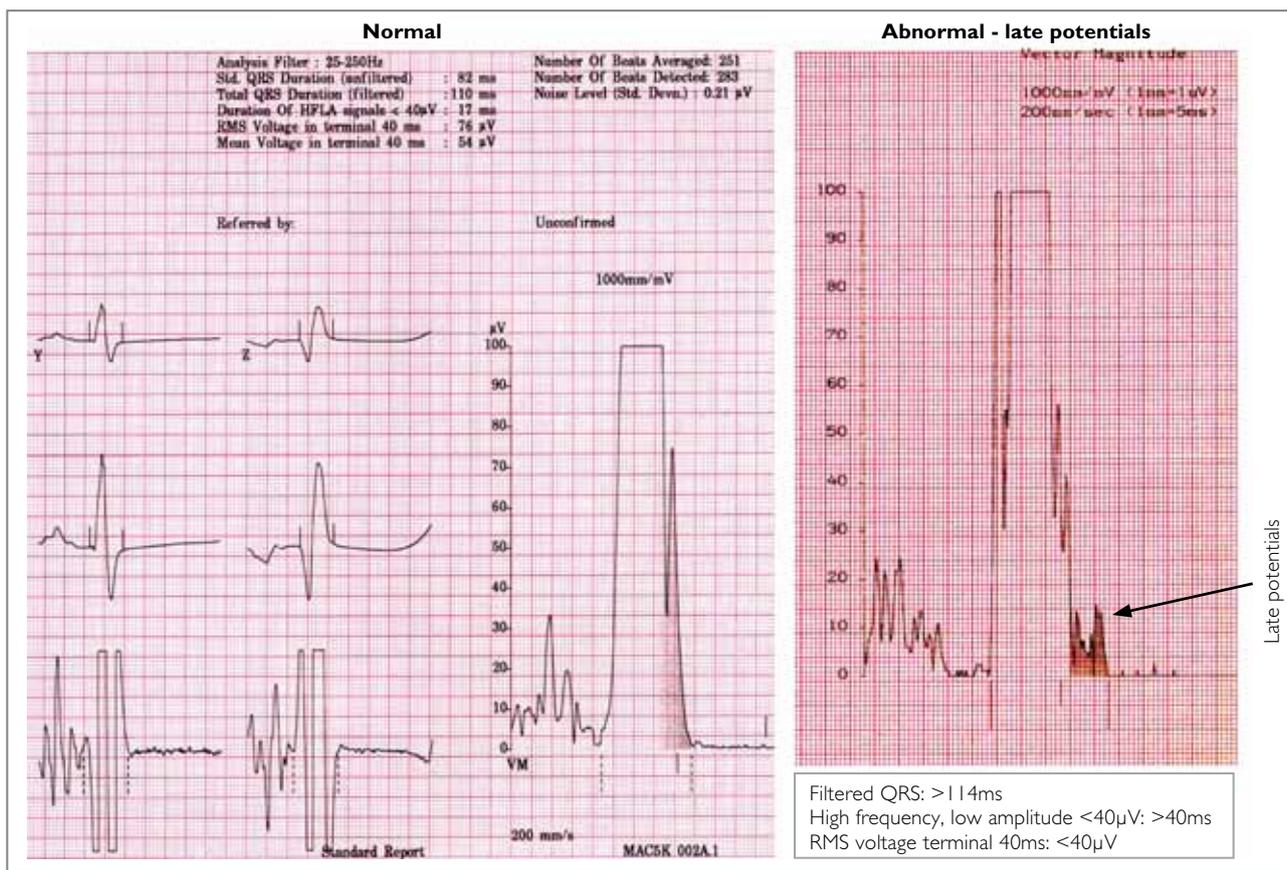


FIGURE 3: Examples of a normal signal averaged ECG (left) and one showing abnormal late potentials from a patient with ARVC (right). Late potentials are low amplitude signals persisting after the main QRS deflections are over. They indicate late activation of ventricular muscle and are a substrate for re-entry ventricular arrhythmias.

An exercise stress test may be done to exclude ischaemia, although this is an unlikely cause of right ventricular PVCs. The PVCs will probably be suppressed by sinus tachycardia, but exercise may provoke sustained VT in both idiopathic right ventricular tachycardia (RVOT) and ARVC.

If the tests are normal, he can be reassured. Treatment is not necessary in the absence of symptoms.

The answer to (2) is (b): Do further tests to establish whether his heart is structurally normal.

LESSONS AND CONCLUSIONS

- The significance of PVCs depends on their context – whether or not there is structural heart disease.
- Many people are unaware of PVCs, whereas in others they cause severe symptoms.
- Drug treatment of PVCs is potentially harmful.
- Right ventricular PVCs may be idiopathic and benign, if they arise from the outflow tract.
- PVCs arising from anywhere in the right ventricle may be a sign of ARVC, a more sinister condition.

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