

Country cardiograms case 59: Answer

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There is a very slow ventricular rate of 26 per minute. Left bundle branch block is present, with QRS duration of 0.17 seconds. Each QRS complex is preceded by a P wave, with a long (but constant) PR interval of 0.28 seconds. P waves are also noted toward the end of the T waves that follow the QRS complexes. These are not conducted to the ventricles and are followed by a pause. They appear to have similar morphology as the conducted P waves, but it is hard to be certain as they are superimposed on the preceding T waves.

There is therefore a pattern of alternating short and long PP intervals. The short PP intervals measure 0.75 seconds; the long PP intervals measure 1.52 seconds.

There are 2 possible explanations for these observations. The first is sinus bradycardia with atrial bigeminy with blocked premature atrial complexes (PACs). The second involves a combination of sinoatrial (SA) exit block and second-degree atrioventricular (AV) block.

The fact that the long PP intervals are almost exactly double the length of the short PP intervals suggests that SA exit block may be present. In this condition, the sinus impulse is blocked at the SA junction. If this is the case in Figure 1 (page 68), every third P wave is absent, indicating 3:2 SA exit block. (Wenckebach SA exit block should be considered, but typically in this condition the longer PP interval would be less than twice the short PP interval.)

Of the P waves that are evident, every second one is not conducted. If P wave morphology differed from

that of the conducted P waves, blocked PACs could be diagnosed with confidence, but in this case one cannot be sure. Second-degree AV block of the 2:1 variety must therefore be considered.

As the left bundle branch block is old, it is not possible to know whether such block would be at the level of the AV node or at the level of the right bundle branch. (If QRS complexes were narrow, the block would be at the level of the AV node, and if the left bundle branch block were new, this might make a block at the bundle branch level more likely.)

Regardless of the precise interpretation details, this patient presented with symptomatic bradycardia, and the absence of a junctional or ventricular escape rhythm is worrisome. Discontinuation of medications that slow conduction in the AV node (in this case, diltiazem) is always a safe first step. However, if blocked PACs are thought to be the cause, suppression of these ectopic beats would be considered, but some of the treatments available might adversely affect AV conduction and therefore precipitate complete heart block. If the SA exit block and second-degree AV block scenario is considered more likely, pacing (or an attempt with atropine) would need to be considered.

Asking the patient whether symptoms increase with exertion might be useful. Cautiously having the patient walk while recording the rhythm on the monitor may help differentiate the level of block or may alter the frequency of PACs, if indeed they are present. Monitoring of symptoms and vital signs will

be essential in deciding whether this patient could be managed locally, allowing time for the effect to occur of stopping diltiazem treatment (with transcutaneous pacing available if urgently required), or whether early referral for transvenous pacing is indicated. Regardless of which decision is made, symptomatic bradycardia should be regarded as a serious and potentially ominous presentation.

One of these explanations involves a single abnormality: blocked PACs. The other involves abnormalities at 2 separate levels: the SA node and the AV node. Isn't the simplest, least complex interpretation (blocked PACs) more likely? Perhaps, based on just 1 tracing, but simply sitting at

the bedside, watching the monitor screen and examining further tracings, can provide the answer. In this case, a high-grade second-degree AV block ensued, followed by third-degree AV block, and this patient received a dual-chamber pacemaker on an urgent basis.

SA exit block, although relatively rare, may be a benign vagotonic phenomenon in healthy people. It can also be a marker of more generalized nodal disease, as may have occurred in this case.

For the question, see page 68.

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Country Cardiograms

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