What Is Happening Here?

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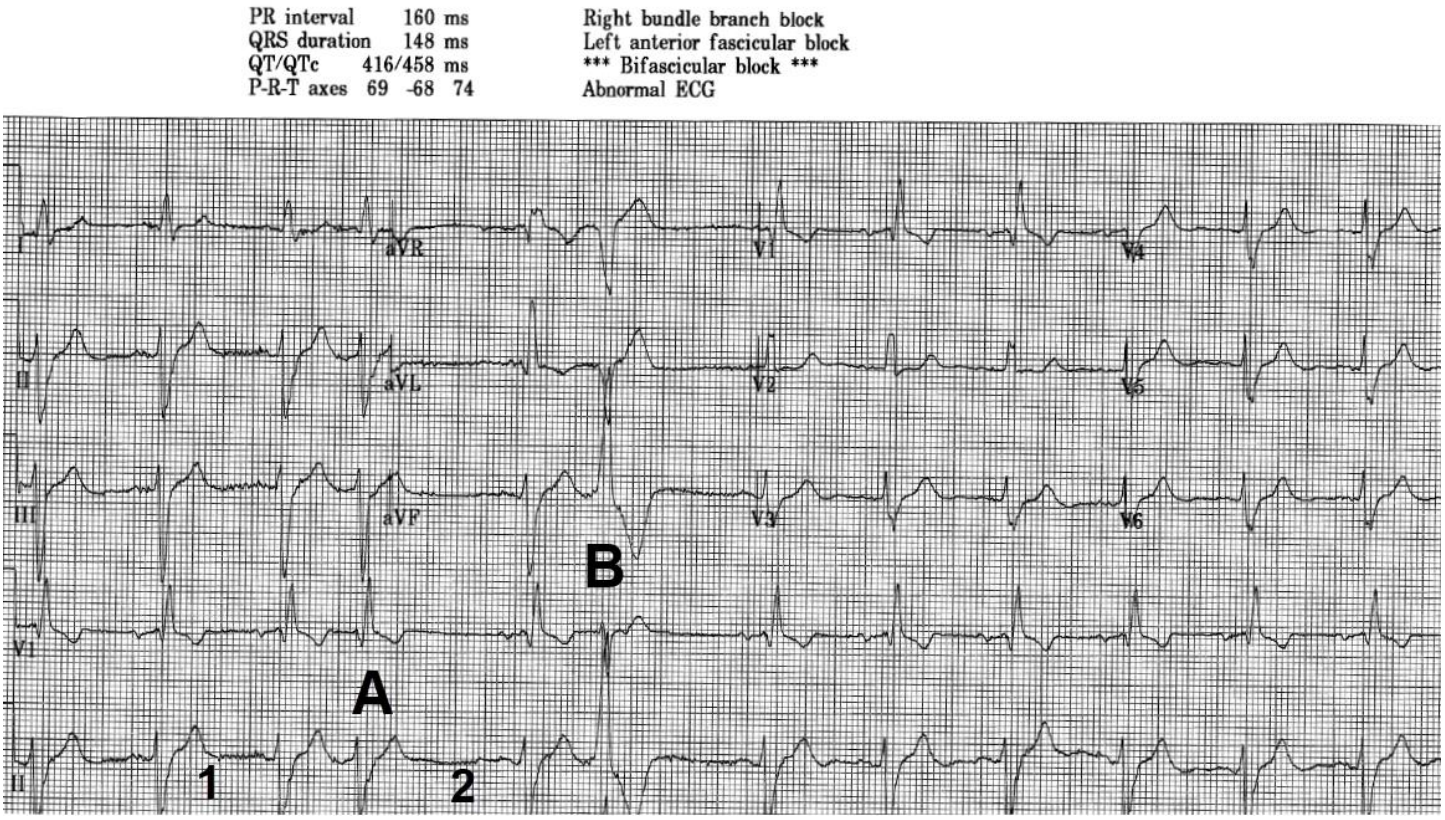

Figure 1

1. Does the bifascicular block – specifically, the complete RBBB (cRBBB) with LAFB – present any danger or threat to the patient? (The latest recommendations are *not* to use the term "bifascicular block" but to state the specific blocks instead.)

   Answer: *Whenever there is a block involving the right bundle branch and one of the fascicles of the left ventricle, there is a possibility of an eventual block of the remaining fascicle resulting in a complete, 3rd degree AV block. The incidence is low and cardiologists generally just follow the patient without intervention.*

   PEARL | In the presence of a cLBBB or a cRBBB with a block in one of the fascicles of the left ventricle (anterior or posterior), if there is also a first degree AV block be aware that the first degree AV block may *not* be as benign as you may think. In each block mentioned, there is only one remaining fascicle providing entry into the ventricles. If that fascicle becomes diseased or damaged, it will conduct more slowly and result in a *prolonged PR interval* or a *first degree AV block*. And, of course, there is the possibility that it may stop conducting at all leading to a sudden complete AV block! Remember: *AV block* does *not* necessarily imply any involvement of the *AV node*.

2. There are two premature complexes on this ECG. I've labeled them "A" and "B."

a. What type of premature beat is "A" and why do you think so?

b. What type of premature beat is "B" and why do you think so?

Answer: *The first premature beat (A) is a premature junctional complex (PJC). There is no P wave evident and the preceding T wave appears no different than the other T waves, so there is little chance of a P wave hiding in a T wave.*

The second premature beat (B) is a much more problematic. It could be a premature junctional complex – though it could be a premature ventricular complex (PVC). Personally, I lean more towards premature junctional complex. Why?

It would be very unusual for a PJC to be followed so closely by a PVC – with the same coupling interval! So why does one PJC (A) conduct normally through the His-Purkinje system while the second (B) conducts with what appears to be a complete bundle branch block in every lead except Lead V1 – in which it demonstrates a likely origin in the right ventricle since the QRS is negative looks nothing like a bundle branch block?

The answer may lie in the R-R intervals that precede the premature beats. I've labeled them "1" and "2." Do you notice anything? The R-R interval #2 is much longer than R-R interval #1. What we have here is a very good demonstration of the Ashman-Gouaux phenomenon (aka "Ashman phenomenon"). Ashman and Gouaux discovered that each R-R interval determines the length of the refractory period of the succeeding R-R interval. (This applies to the His-Purkinje system, i.e., bundle branches only!) In other words, each R-R interval length is indirectly proportional to the length of the refractory period of the following R-R interval. The shorter R-R interval (1) enabled PJC (A) to fall just outside the refractory period while R-R interval (2) created a much longer refractory period that caused PJC (B) to fall within the absolute portion of its refractory period.

But why does the aberrantly-conducted QRS in Lead V1 look so "unlike" a complete bundle branch block. The answer could be something called "normalizing fusion." There is a fixed cRBBB present, so that means that impulses have been entering the ventricles through the left ventricle. In this instance, the right ventricle was activated at the same time as the left ventricle which simulated "normal conduction." There are two ways this can happen – 1) a PVC can fortuitously appear in the "blocked" ventricle at exactly the same time as the sinus beat, or 2) equal delays in the left and right bundle branches could produce the same result. That would simulate normal ventricular conduction and a relatively normal-appearing QRS would result. However, the "normal" appearing beat would appear normal in all leads – not just one lead.

I have only one more possibility left that may explain the QRS in Lead V1 – the depolarization vector recorded by Lead V1 may have had a direction and angle that produced a QRS that was very different than the other leads.

Beyond those possibilities, I have no further suggestions.

3. Is there anything that concerns you about premature beat "B"?

Answer: *In the more classic bundle branch block complexes of premature beat "B," the repolarization abnormality begins 2-3 mm below the baseline. For repolarization abnormalities that have an R wave followed by an inverted T wave, we only allow 1 mm of depression at the J point. Beyond that, one should consider myocardial ischemia in addition to whatever else is happening.*