LBBB… or LBBB + Anterior Fascicular Block?
Discussion

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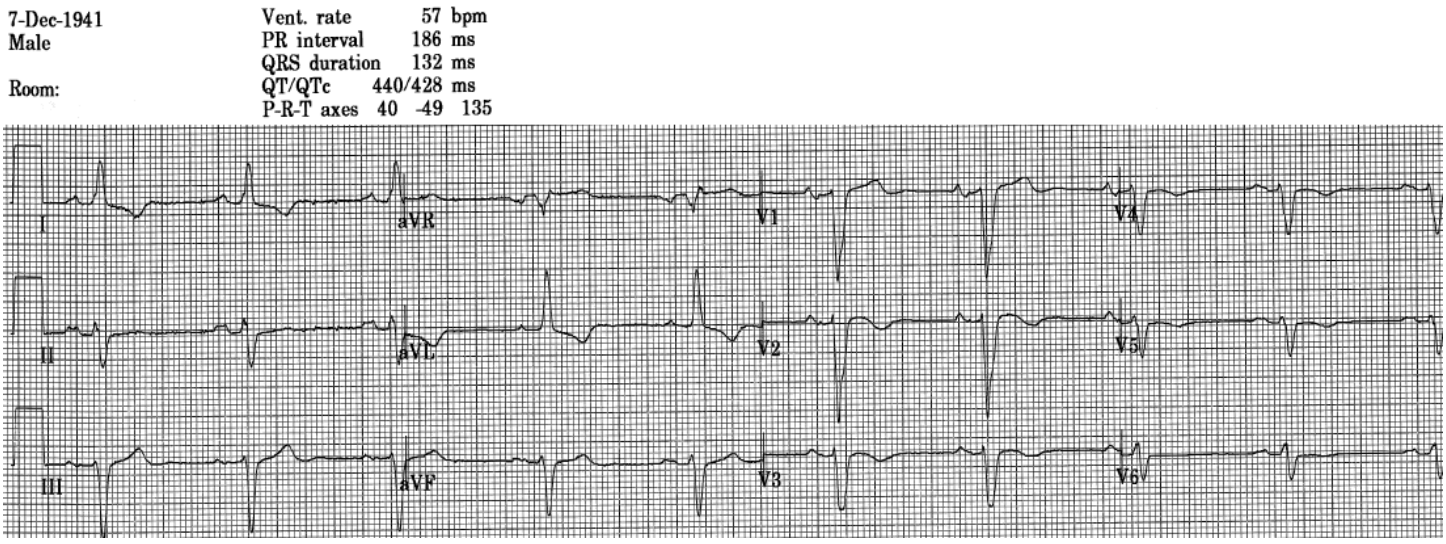


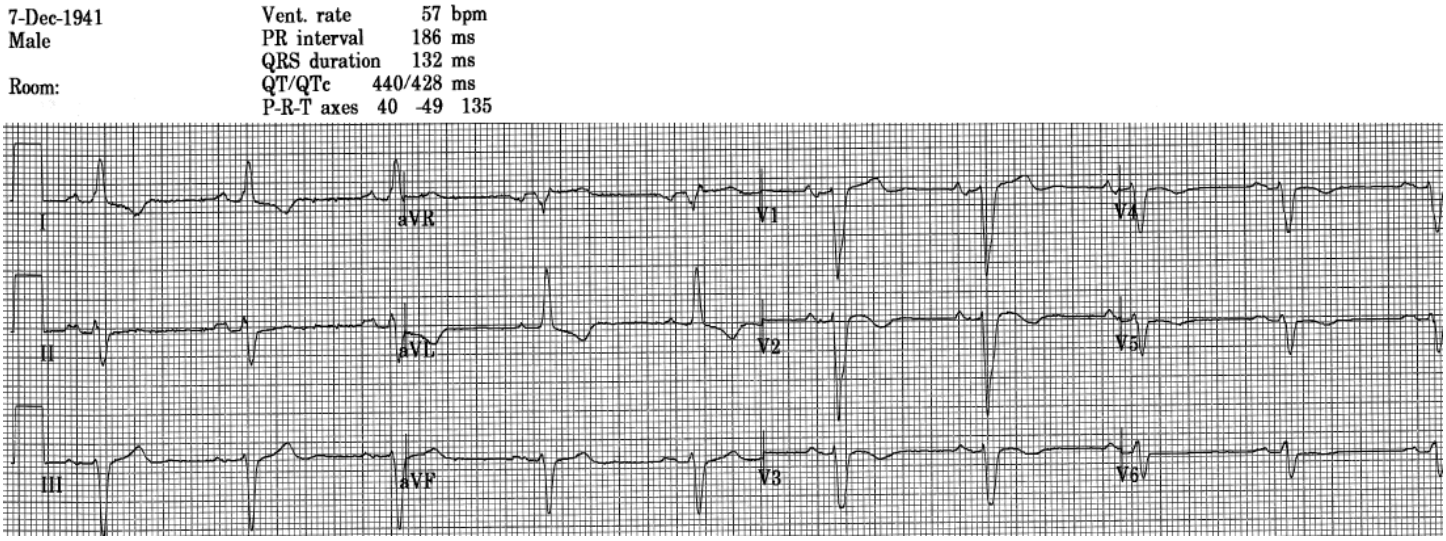
This is another randomly selected ECG from my collection. We see a *very common pattern* here – something that looks like left bundle branch block (LBBB) – but also anterior fascicular block. And the LBBB pattern really isn't "classic." A "classic" LBBB has wide, monophasic, notched R waves in Leads V5 and V6 and that's certainly NOT the case here.

First… there is NO anterior fascicular block present! Yes, I KNOW that it looks just like an anterior fascicular block (what used to be called *left anterior hemiblock*), but it really isn't. How can I be so sure? When an anterior fascicular block is present, the ventricles are being activated from left-to-right. When a left bundle branch block is present, the ventricles are being activated from right-to-left. How does that present differently on a 12-lead ECG?

With a *true* anterior fascicular block, the posterior and septal fascicles are still working just fine. Septal depolarization proceeds normally from left-to-right, posterior-to-anterior and (usually) slightly downward. How does that appear on the ECG? There will be narrow rS complexes in the inferior leads and in Leads V1 and V2. However, each left-sided lead will still see the septal vector traveling away from it and will inscribe *a very small initial q wave – the septal q*.

As you can clearly see, there are no septal q waves on this ECG because ventricular activation is from right-to-left and anterior-to-posterior. We've all probably seen LBBB aberrancy with much wider QRS complexes. In this case, the impulse traveling across the septum from the right ventricle likely entered the posterior fascicle immediately and conducted the rest of the way through the His-Purkinje system, though in a retrograde manner.

Now let's address the lack of R waves throughout the precordium. The presence of a true anterior fascicular block can result in a ***clockwise rotation***. Let's pause here for a bit of explanation…

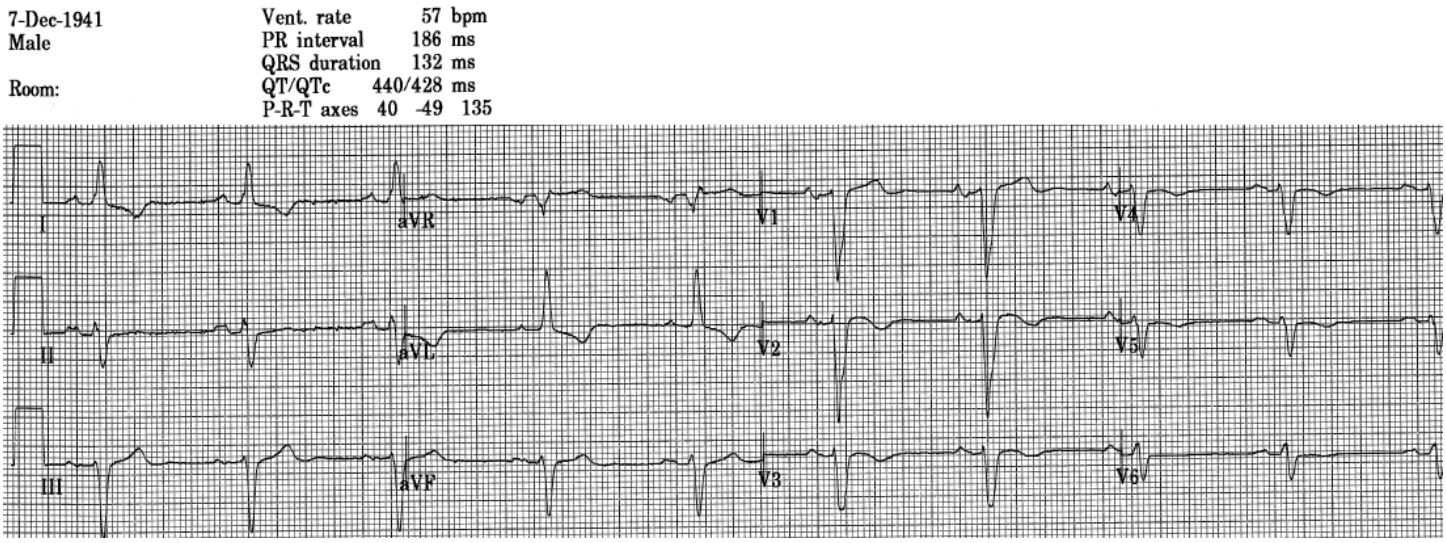


In the *frontal* plane, we talk about the mean QRS *axis* (ÂQRS). You are all familiar with that. In the *horizontal* (or *transverse*) plane however, we speak in terms of *rotation*. It's just easier. We *could* speak of a horizontal axis, but it's a bit more complicated than axis in the frontal plane. The lead axes in the horizontal plane are not all 30° apart – they vary. Therefore, we simply look to see where the rS complexes that are characteristic of the right-sided leads turn to RS or Rs complexes characteristic of the left-sided leads. The *transition point* normally occurs at or between Leads V3 and V4 (inclusive). If the rS complexes extend past Lead V4, there is a left axis deviation in the horizontal plane but we call it instead a *clockwise rotation*. If the QRS complexes have an R taller than the S (R/S ratio > 1) in Lead V2 or V1, there is a right axis deviation in the horizontal plane, but we refer to that as a *counterclockwise rotation*. Some of you may refer to it as *anticlockwise*. (Just imagine looking up from the patient's feet.)

Actually, it's all somewhat *relative*. If a patient normally manifests a transition in Lead V5 but today the transition is in V4, you could say they have had a counterclockwise rotation *relative to their previous ECG*.

Getting back to our ECG… the late, retrograde activation of the anterior fascicle has resulted in a rather profound left axis deviation. ***This always happens when the anterior fascicle is activated retrogradely AFTER the posterior fascicle***. The cause can be a true anterior fascicular block or right-to-left activation of the ventricles as in LBBB. It doesn't matter – the main thing to remember here is that *the anterior fascicle is activated AFTER the posterior fascicle in both cases!*

This certainly results in a clockwise rotation of the *electrical axis* of the heart (the heart itself really doesn't have to move). However, even with a full anterior fascicular block, we usually see a lot more R wave in Leads V5 and V6, even thought the R/S ratio may be slightly less than 1.0. I suspect something else has happened here. Any ideas?



A previous MI could certainly have caused this. Now, let me be clear: most cases of "poor R wave progression," as we see in this ECG, are NOT due to a previous MI, but rather to anterior fascicular block, COPD or other causes of left axis deviation. We've already ruled out true anterior fascicular block and, since there is no P pulmonale, rightward shift of the P axis in the frontal plane (there is an upright P wave in Lead aVL) or inverted T waves in the inferior leads, we can rule out advanced COPD also. This makes a previous anterior MI more likely as a cause of the lack of R waves in the precordial leads. What else on this ECG might suggest a previous MI?

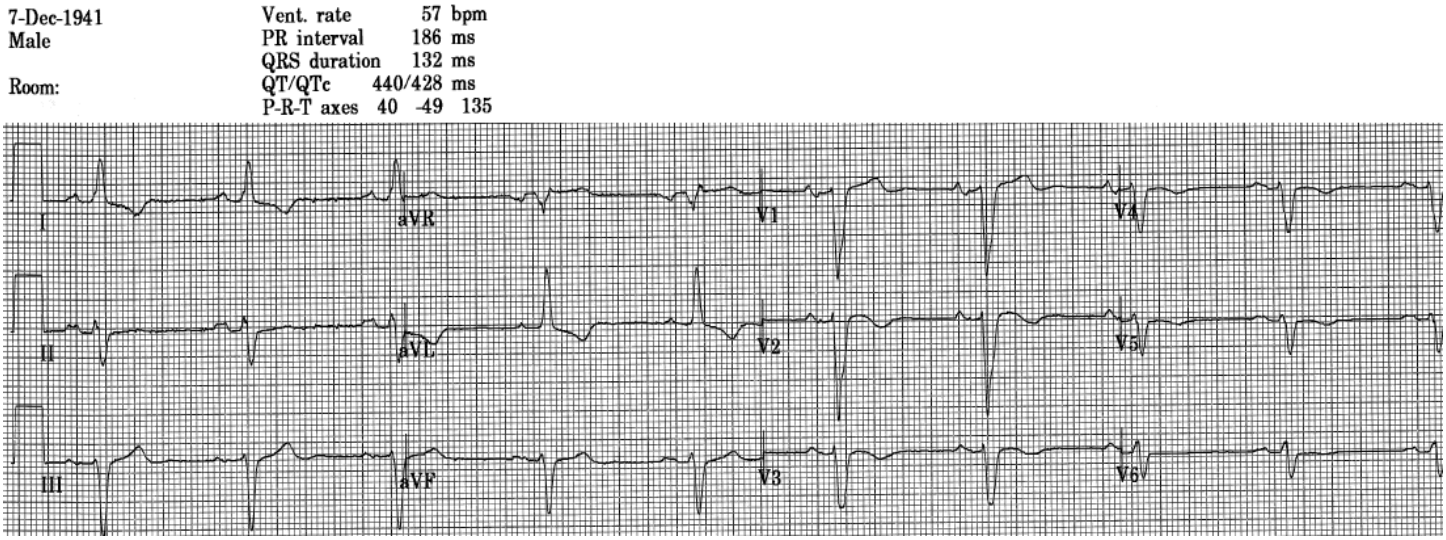
The LBBB! Here is one more possibility that could explain both the loss of R waves in the precordium and the anterior fascicular block-like pattern in the inferior leads: an occlusion of a Type 3 ("wrap-around") LAD. An occlusion distal to the first diagonal branch would spare the basolateral area leaving Leads I and aVL with tall, intact R waves, but attenuating the R waves in V3 – V6 and Leads II, III and aVF. However, that would NOT *necessarily* explain the LBBB in this 70 y/o male (ECG was recorded in 2011) (the left bundle branch is perfused by the first septal perforator). However, *age* and *coronary vascular disease* in general could cause that.

What else is likely present? You aren't necessarily going to see it on this ECG but I can almost promise you it's there! Let me give you a not so subtle hint:

About 95% of all patients with complete LBBB (QRS duration $\geq$ 120 msec) also have *left ventricular hypertrophy!* So there is likely hypertrophy of the left ventricle as well. Could LVH alone explain the findings on this ECG? No… there is right-to-left activation of the left ventricle and LVH alone doesn't cause that. LVH causes fibrosis within the left ventricular wall which

delays conduction by disrupting the His-Purkinje fibers and THAT can eventually result in right-to-left conduction, but the LVH must have advanced to that extent.

What about the T wave inversions in Leads V2 – V5 along with the subtle ST depression in Leads V4 and V5? Most likely that is due to chronic subendocardial ischemia. Please note that I



did not say "anterolateral" subendocardial ischemia – just *subendocardial ischemia*. The ST depression and T wave changes of subendocardial ischemia are actually non-localizing. So, does that mean there is NO subendocardial ischemia in the anterolateral region. No, it doesn't mean that at all. However, that region may NOT be as involved as the surface 12-lead ECG might suggest and other areas may be involved with little or no representation on the ECG. Lead II has a flat ST segment and an isoelectric T wave. That, too, is suggestive of subendocardial ischemia. Just remember that **ST depression due to ischemia does not localize!**

Reading an ECG is just simply reporting the changes that you see. ***Interpreting*** an ECG takes that basic information and gives you a much better picture of the patient's *present* condition, their *past* medical history and maybe even some insight into what the *future* holds for them.

I hope you gained some insight into what you are capable of determining from a 12-lead ECG.

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