Anterior Fascicular Blocks – What You Probably Don't Know!

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What exactly IS an **anterior fascicular block** and how do you diagnose it? And after you diagnose it, what does it mean for the patient? What does it mean for your *management* of the patient?

For years I struggled with wondering "What IS a *left anterior hemiblock*? And why is it different from left axis deviation? (Actually, it's just *one cause* of left axis deviation.)

Let's begin with what it is not. Do not use the term *hemiblock* anymore. It has been outdated for many, many years. It is an anterior *fascicular* block. And you no longer must mention the word *left*, either. How many *right* anterior fascicular blocks have you heard of? Let me answer that for you: ***NONE!***

In this article I am going to discuss how and when to diagnose an anterior fascicular block, what it means for the patient and how it may – or may not – affect your management of the patient.

Let's look at a classic example of an anterior fascicular block...

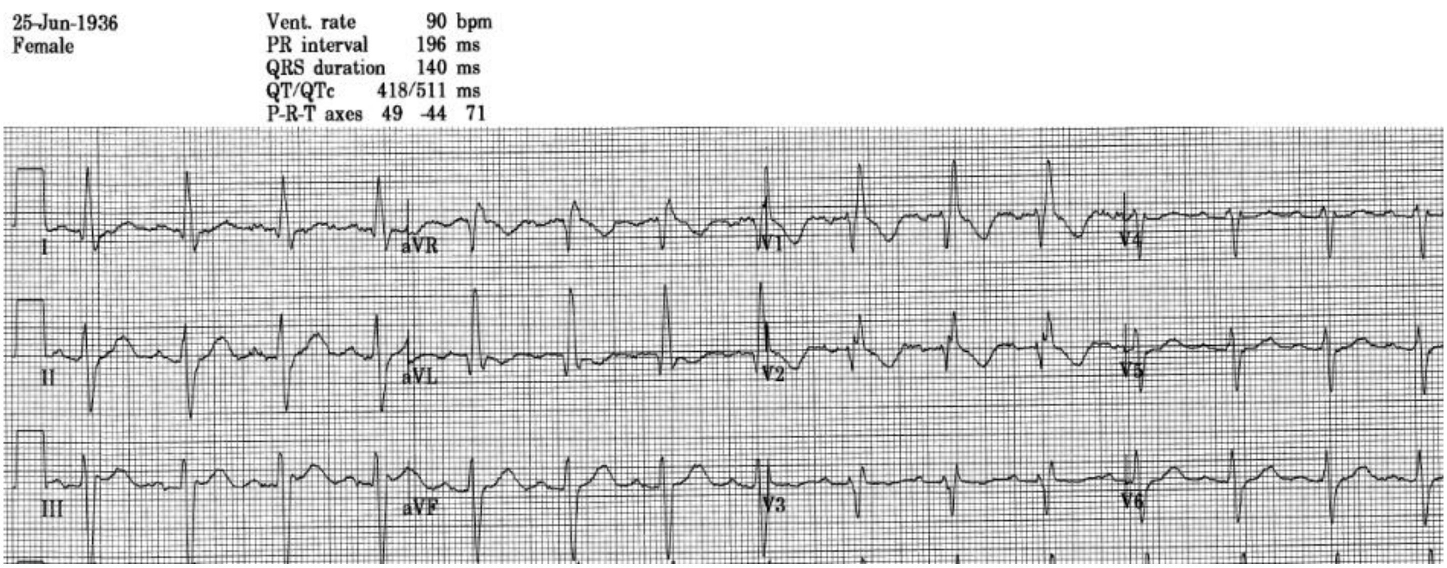


Figure 1

This is a classic example of an anterior fascicular block. In this ECG, it accompanies a complete right bundle branch block. While this is very common, contrary to popular belief, they do not have to appear together. You will see examples of anterior fascicular blocks *without* a right bundle branch block.

An anterior fascicular block is not an unusual finding. They certainly are not *rare* like their cousins – *posterior fascicular blocks*. But do you know what is even *more frequent?* The *mimics* of anterior fascicular blocks! The diagnosis of anterior fascicular block is a *very precise* determination and too often the diagnosis is made when the condition is not present. I am going to teach you how to recognize both.

Here are the *precise* criteria that determine the presence of an anterior fascicular block:

1. A small q in *both* Leads I *and* aVL. A qR in both leads! This is very important!

2. A small r in Leads II, III and aVF (an rS morphology in *all* three leads, especially Lead II).

3. A normal or slightly prolonged QRS, usually less than 120 msec. In Figure 1 the QRS duration is prolonged even further by the presence of the complete RBBB. That's OK.

4. There should be a *left, superior axis* – usually between −30° and −60°. Note that many texts say that the axis must be equal to or more negative than −45°. Most of the time it will be more negative than −45°, but not always! (FYI, −50° is more negative than −45°, while −30° is less negative than −45°.)

   There definitely will be a left axis deviation (more negative than −30°), but the use of −45° as a criterion needs explanation. The use of −45° became a "rule" because it is located exactly between 0° and −90°. By using −45°, false positive diagnoses were reduced. But this is not based on *physiological* reasons – it is arbitrarily based more on *simple practicality*.

   The *real* determinant of anterior fascicular blocks is the presence of **left axis deviation** *and* the specific – and I want to emphasize ***specific*** – **morphologies** in Leads I and aVL and in Leads II, III and aVF.

   PEARL | *The pattern matters more than the exact axis value!*

Let's look back at the ECG in Figure 1. The first thing you should check is whether there is a *left axis deviation* present. How do you do that? By looking at Leads I and II. Please forget about using Lead aVF when determining the presence of a left axis deviation. We stopped using Lead aVF in the 1960s – *over 60 years ago!* Yet even today, I see online ECG "classes" that still use that *outdated* criterion. Maybe if people refreshed their knowledge of electrocardiography more often than every 60 years, we could make faster progress.

Well, we see that the while the QRS in Lead I is positive, the QRS in Lead II is negative resulting in a left axis deviation. It isn't quite −45°, but that shouldn't bother us. Now we check the morphology of the QRS complexes. In Leads I and aVL (we need *both* leads here) we see a tiny q and a tall R. That tiny q is the septal q which informs us that the onset of left ventricular depolarization has begun normally with a left-to-right depolarization of the interventricular septum. This impulse comes from the posterior fascicle (or perhaps from a *septal* fascicle – if one is present). Under *normal* circumstances, the anterior and posterior fascicles depolarize *simultaneously*, thus canceling each other, so we do not detect any axis deviation. But that's not the case here – we have a profound left axis deviation.

Now let's look at Leads II, III and aVF. We see the required rS complexes in all three leads. This IS an anterior fascicular block. Remember: ***morphology before axis!***

Let's look at another example…

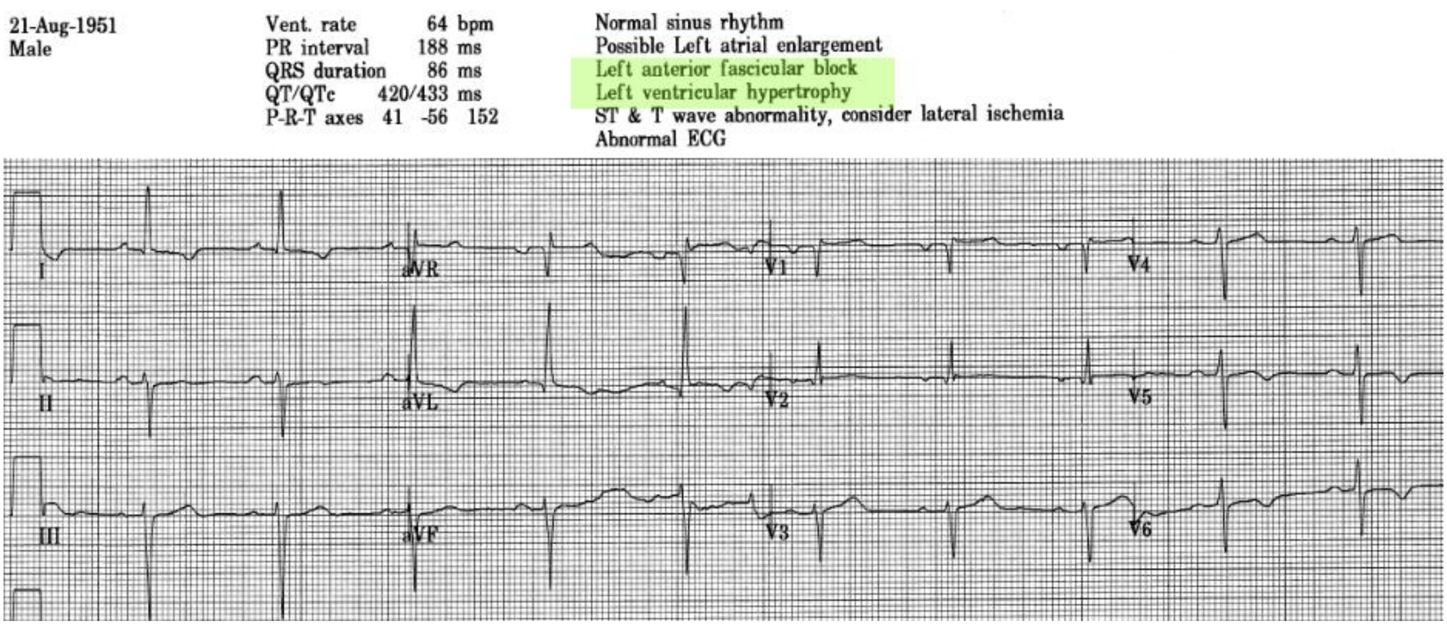


Figure 2

Here is what appears to be an example of an anterior fascicular block without a right bundle branch block. That's what I thought as I was sifting through my ECG collection. Then something caught my eye. As I mentioned earlier, although anterior fascicular blocks are relatively common, there is something even more common: the *mimics* of anterior fascicular blocks!

There is *one more criterion* for anterior fascicular block: *no other cause of left axis deviation* (most commonly LVH, old inferior MI, paced rhythm, dilated cardiomyopathy).

The precordial leads certainly show no evidence of LVH but look at Leads I and aVL. While both leads meet the criteria for anterior fascicular block, they each manifest something that is NOT present in pure anterior fascicular block. Do you see it? Both leads manifest an early but distinctive ***secondary repolarization abnormality*** – the ST segment that slopes downward into an inverted T wave followed by an abrupt return to the baseline. We see these changes in four situations: complete bundle branch blocks, ventricular ectopy, some cases of ventricular hypertrophy (but not all) and some cases of ventricular pre-excitation (but not all). Note that I did not include anterior fascicular block!

How can we know if *both* LVH and anterior fascicular block are present?

There is no real reason that simple left ventricular hypertrophy should replicate the same QRS morphologies as anterior fascicular block. What will be common to both conditions, however, is a tall R wave in Lead aVL and a deep S wave in Lead III.

PEARL | *The lead axis for Lead aVL is −30° making it the boundary between a normal axis and left axis deviation. Lead III is as close to being opposite Lead aVL as it can be, so when there is a tall R wave in Lead aVL there will always be a deep S wave in Lead III. Therefore,* ***a deep S wave in Lead III*** *is just as suggestive of an anterior fascicular block or left axis deviation as a tall R in Lead aVL.*

Here is a very important point to remember: the diagnosis of anterior fascicular block is based on a qR morphology in Leads I and aVL and an rS morphology in Leads II, III and aVF. Since LVH is part of the issue here, we are going to insist that the mean QRS axis in the frontal plane (ÂQRS) is −45° or more negative (i.e., between −45° and −90°). Voltage alone (i.e., the height of the R wave in Lead aVL) isn't really going to help us here, but there are two things to remember:

1. Anterior fascicular block will *never* present with a secondary repolarization abnormality (sometimes called a *strain pattern*), and
2. Regular LVH will rarely have a mean axis that is more negative than -45° without a concomitant anterior fascicular block.

So, in Figure 2, the *QRS morphology* of anterior fascicular block is present and the *mean QRS axis is −-56°*, therefore, anterior fascicular block is present. But… both Leads I and aVL have *repolarization abnormalities*, so LVH is *also* present.

Let's look at another ECG (next page) …

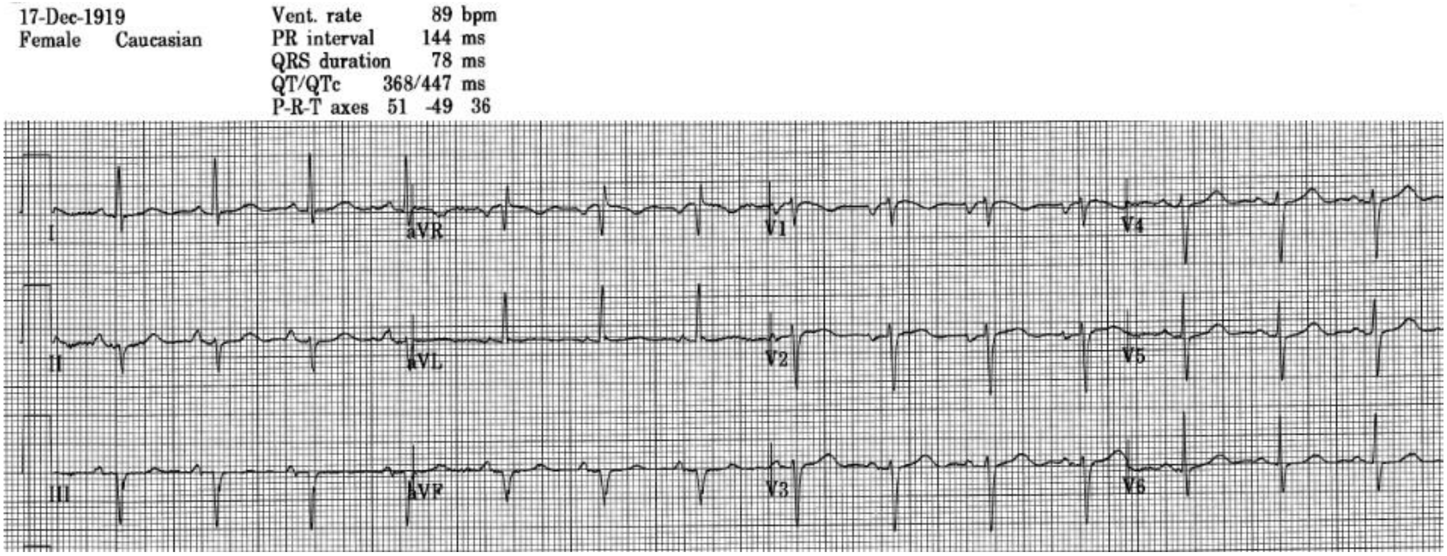


Figure 3

Superficially, this looks like an anterior fascicular block – but let's look more closely …

While there is an rS morphology in Lead II (yes, there is a tiny r there), there are no r waves in Leads III or aVF. That *disqualifies* this as an anterior fascicular block. Also, there are no qR complexes in Lead I or Lead aVL. That also *disqualifies* this as an anterior fascicular block.

However, there is a definite left axis deviation present. What is its cause? Leads III and aVF provide the answer here: this is a previous *inferior wall myocardial infarction*, which just happens to be a *major source of left axis deviations*.

PEARL | *While signs of LVH may manifest in both the limb leads and the precordial leads, signs of anterior fascicular block present only in the limb leads (frontal plane). But there is a consequence of the profound left axis deviation that accompanies an anterior fascicular block: a delayed transition in the precordial leads, sometimes extending past Lead V6*.

While Figure 3 may *look* like an anterior fascicular block, in this case it is not. Although the mean axis is −49°, remember that *it is the QRS morphology that determines the presence of anterior fascicular block – not the height of the R waves or the mean QRS axis*.

Another possible anterior fascicular block – or not… (next page)

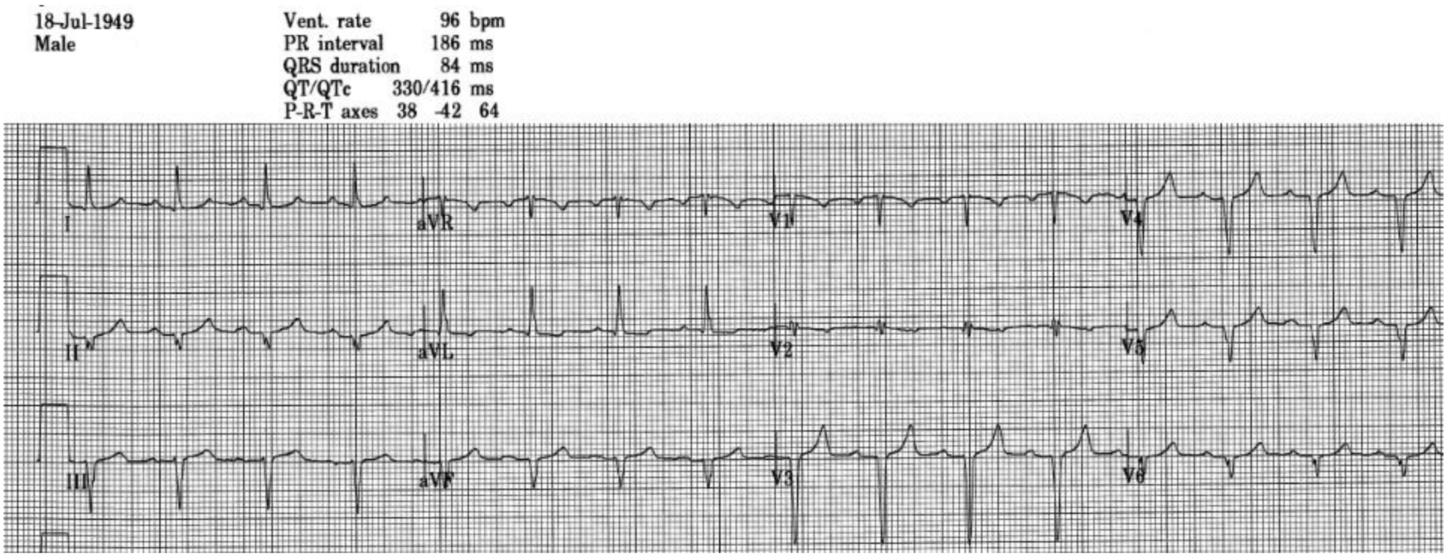
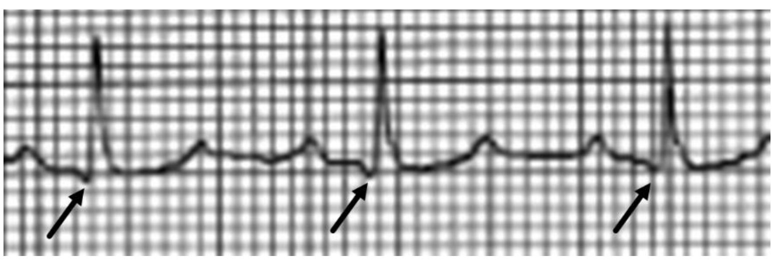


Figure 4

Another ECG that looks very much like an anterior fascicular block… but is it?

Remember: we diagnose anterior fascicular block ***based on the QRS morphology in the frontal plane (limb) leads – not on axis or voltage*** (R wave height or S wave depth).

First, do *both* Leads I and aVL manifest an qR morphology? Actually, yes they do, but Lead I is a bit difficult to see if you aren't trained. Let's enlarge Lead I:



This enlarged view of Lead I from Figure 4 demonstrates very small q waves. The q waves in Lead aVL are much more visible. OK, that qualifies for an anterior fascicular block – but there's more to decide.

Looking at Leads II, III and aVF, we perhaps see a small r in Lead III – but I really doubt its significance. Here's why: Lead III is very susceptible to a wavering baseline due to movement of the diaphragm, much more than Leads II and aVF. As you can see, the tiny r wave appears and disappears. Regardless, there are no r waves in either Lead II or Lead aVF. They have qs waves. This is an old inferior MI – *not* an anterior fascicular block.

The mean axis deviation is −42°, rather profound… but not an ultimate determinant of anterior fascicular block. Again, a consequence of left axis deviation – especially a marked left axis like this one – is a delayed transition in the precordial leads. We can certainly see that here. In fact, the transition is occurring somewhere after Lead V6. It strikes me that this is a bit extreme – even for an axis of −42°. Virtually every lead contains a q wave and most have qs morphologies. With an obvious history of inferior wall MI, I would seriously consider this "poor R wave progression" in the precordial leads a manifestation of a previous *anterior* wall myocardial infarction – and a rather extensive one! While a simple anterior fascicular block can definitely delay the transition point past Lead V6, there are usually some r waves present.

PEARL | *What cardiac structure should concern you when you see infarctions of both* ***inferior*** *and* ***anterior*** *walls of the left ventricle? The septum! And you should be especially concerned if one of the infarctions is old and the other in progress! This is a set up for a septal rupture. In such cases, the septum has lost virtually all its blood supply. If this patient were to suddenly develop shock – without ventricular fibrillation – a septal rupture would be the first thing to cross my mind!*

Let's look at another ECG and decide if an anterior fascicular block is present…

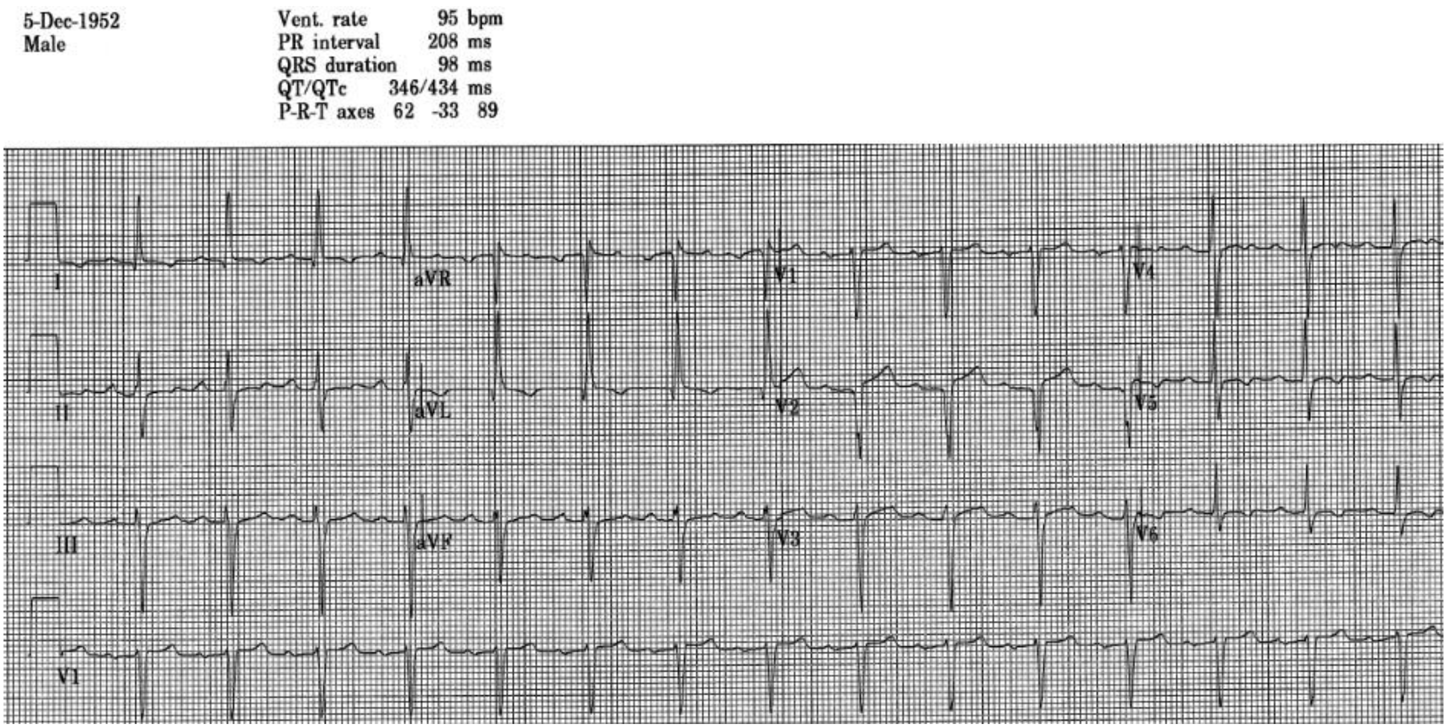


Figure 5

Again, this looks like an anterior fascicular block – but is it really? Leads I and aVL both manifest qR morphologies and Leads II, III and aVF all manifest rS morphologies. There is also a left axis deviation. Although the axis is only −33°, we know that the determination of anterior fascicular block is based on morphology with a left axis deviation and that the left axis is not required to be −45° or less. But is there anything else on this ECG we should be noticing?

YES! First, there are *secondary repolarization abnormalities* in both Leads I and aVL. Pure anterior fascicular block NEVER has secondary repolarization abnormalities! Second, the transition occurs in Lead V4 – not delayed at all! So, is there an anterior fascicular block present here – or is it all left ventricular hypertrophy? I don't think we can reliably state that it is one OR the other. It would be safer to say that *both are likely to be present*. Again, the criteria for anterior fascicular block have definitely been met. *That alone establishes the diagnosis of anterior fascicular block*. But signs of LVH — which are *never* present in anterior fascicular block — are also clearly visible, indicating the presence of LVH.

Let's look at one more ECG that makes a very important point…

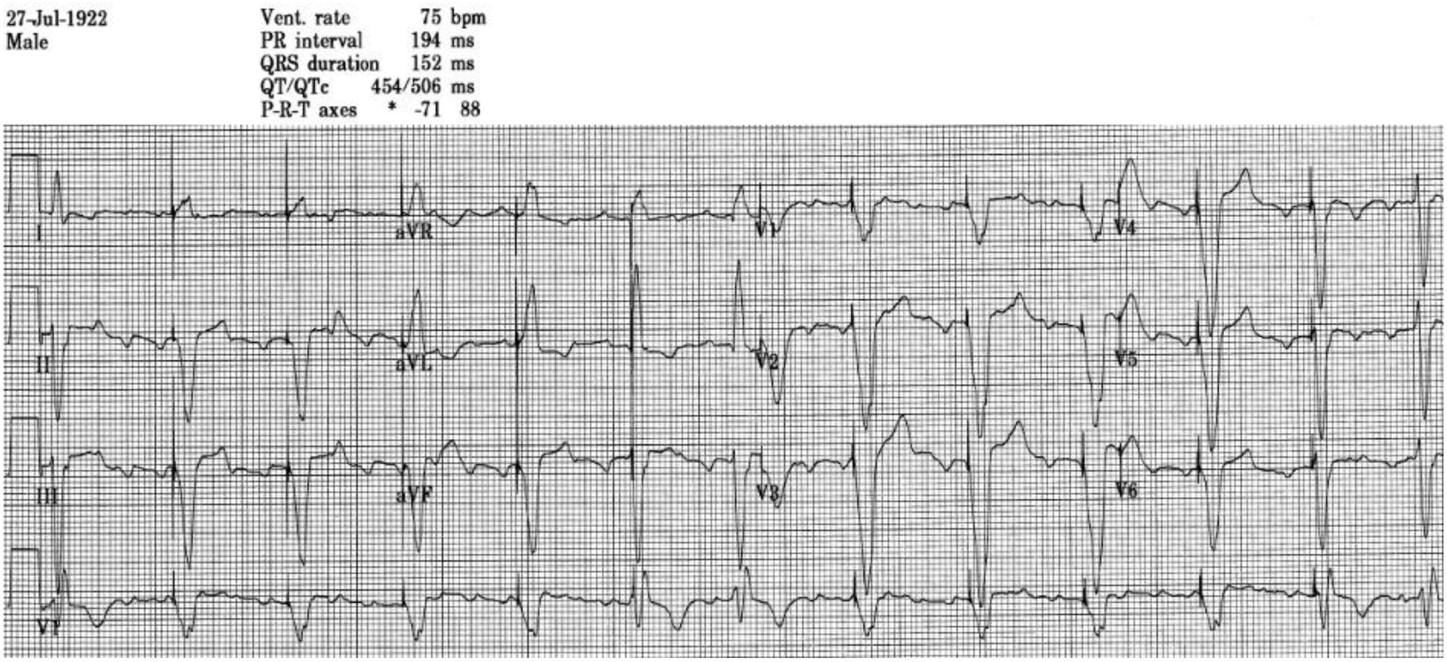


Figure 6

Here we have an obvious *paced ventricular rhythm*. The pacer wire tip is in or very near the right apex, producing exactly what we would expect to see: a left bundle branch block-*like* pattern. The ECG can look very much like this without the pacemaker and with only a true left bundle branch block.

Why did I call this a left bundle branch-***like*** pattern and not simply a left bundle branch block? ***Because there is no LBBB present!***

PEARL | *A bundle branch block can only be present when an impulse enters the ventricles via the bundle of His. It must encounter the block during transmission through the bundle branches and Purkinje fibers which only happens with an antegrade entry into the ventricles via the bundle of His. That does not happen when the impulse arises in the ventricles ectopically (PVC or paced beat) or enters via an accessory pathway*.

Anterior Fascicular Block, the Patient and YOU

Patients will have no idea that an anterior fascicular block is present. It will prolong the QRS complex no more than about 20 msec – often less. So when should you be concerned about it?

The main concern is when it appears in the presence of a right bundle branch block. This constitutes a bifascicular block and many of these patients will eventually progress to complete heart block requiring a dual chamber pacemaker.

Should you be alarmed if you encounter this combination? First, unless the patient has been complaining of *dizziness* or *syncope*, most cardiologists take the "let's wait and see" approach. However, the patient should be well-informed to seek medical care *immediately* if those symptoms develop. Another symptom to watch for is the acute or subacute onset of *weakness* and/or *fatigue*. This may reflect a profoundly bradycardic ventricular rate suggestive of complete heart block.

What You Need to Know and Remember…

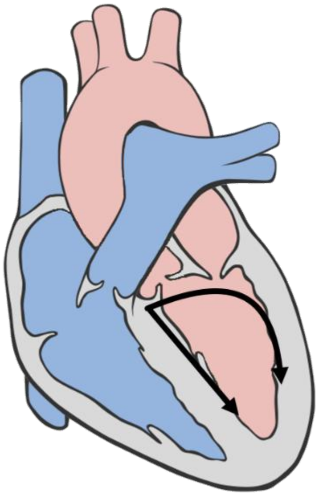
1. The diagnosis of **anterior fascicular block** is a ***morphological*** diagnosis. *It is not based on* ***voltage*** *or* ***axis***. There must be qR complexes in BOTH Leads I and aVL and there must be rS complexes in EACH of Leads II, III and aVF – all of them!

2. An axis of −45° or more negative is *not* a *requirement* for anterior fascicular block. It is an arbitrary rule that is *actually quite good – but not absolute* in every case. A left axis deviation, however, *must* be present because of the required rS complex in Lead II.

3. The most common mimic of anterior fascicular block is probably LVH. Occasionally, they are both present at the same time. Just remember: ***There is no reason that LVH should meet all the morphological criteria of anterior fascicular block – so if all the criteria are present, then so is anterior fascicular block. However, if secondary repolarization abnormalities are also present in Leads I and aVL, LVH is present also.***

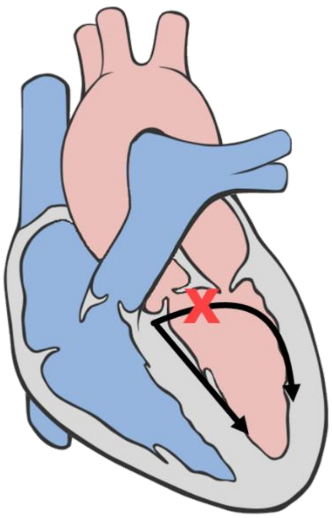
4. Always look to see if anything else could explain the morphological configuration. While a number of conditions can mimic anterior fascicular block, few – if any – can replicate it *exactly* according to its criteria.

Here are some diagrams demonstrating how an **anterior fascicular block** occurs...

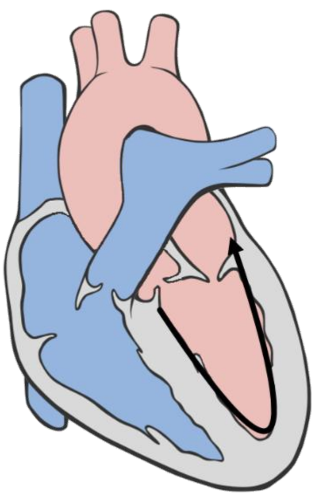


In this illustration we see the posterior fascicle coursing down the left side of the septum and the anterior fascicle crossing over to the anterolateral walls of the heart. Conduction is simultaneous so the vectors for both fascicles cancel each other with no effect on the axis.

Both fascicles are travelling directly to the base their respective papillary muscles. Their job is to activate the papillary muscles first so they will be able to provide countertraction for the mitral valve leaflets, preventing them from everting into the left atrium during the powerful left ventricular contraction.



In this illustration, we now see a block (red "X") has developed in the anterior fascicle. The activation of the anterolateral walls of the left ventricle must now take place *after* the inferomedial wall which is activated first by the posterior fascicle.



This illustration shows how the impulse from the posterior fascicle travels down to the base of the posteromedial papillary muscle, then crosses cell-to-cell through working myocardium to the base of the anterolateral papillary muscle. It then travels retrograde up the anterior fascicle, ending in the basolateral wall. This results in a profound left axis deviation since there are no opposing rightward forces during this time.

The only time the impulse travels outside conducting tissue is during the crossover from the posteromedial to the anterolateral papillary muscle. It is this time spent traveling through working myocardium that adds 10 to 20 msec to the QRS duration. It is certainly possible to have an anterior fascicular block with a normal QRS duration.