Delays and Blocks Involving the Bundle Branches

Part 2 - *Left Bundle Branch Block*

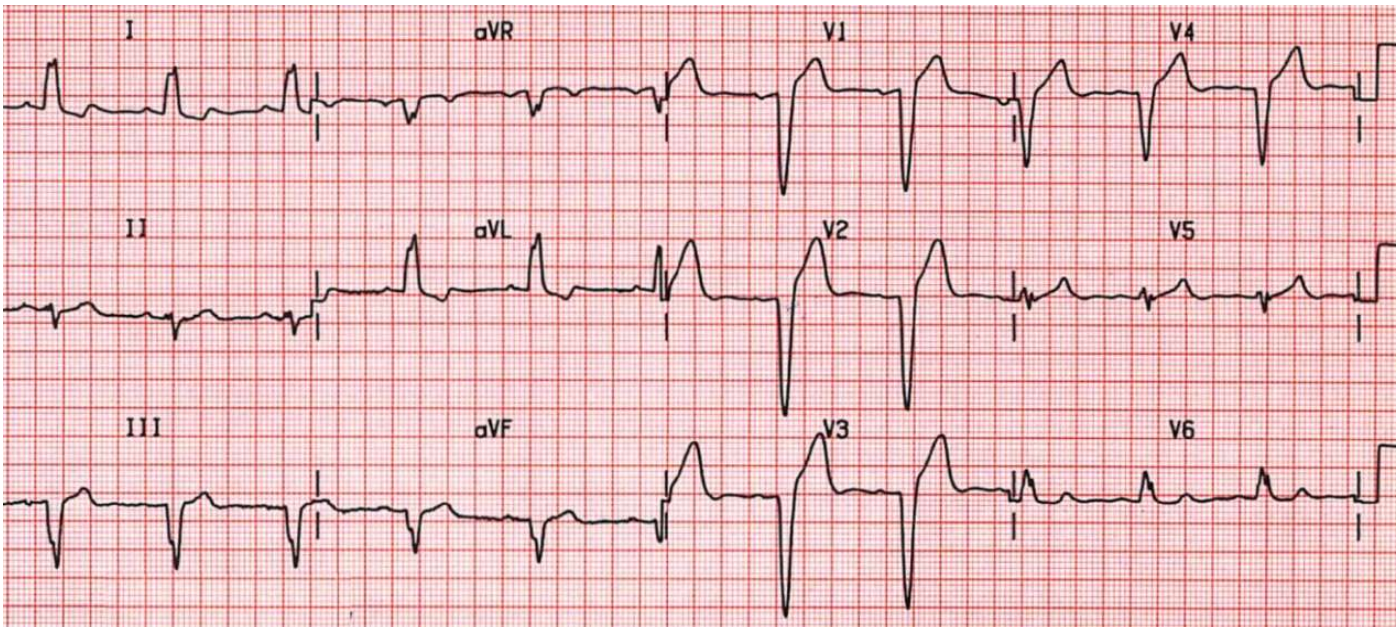


Figure 1

The tracing (Figure 1) demonstrates a **left bundle branch block** (**LBBB**). Because the QRS interval is equal to, or greater than, 0.12 seconds (120 msec), we say that it is a ***complete*** **LBBB** (**cLBBB**). But I'm going to let you in on a little secret – there may be *no block at all!* I'll explain why later.

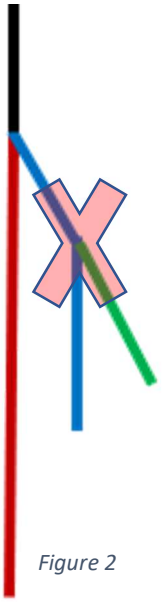


Figure 2

Let's see what is *classic* about this cLBBB…

First, as I said, the QRS interval is ≥ 0.12 seconds. That is an *absolute requirement*. "What's so special about 0.12 seconds?" you ask. Nothing, really. It's part statistically-based and part arbitrary. But it's a convention used world-wide.

When we are looking for bundle branch blocks, the first lead we look at is always **Lead V1**. It's the *most important lead for distinguishing between the right ventricle and the left ventricle* on the 12-lead ECG (right and left atria, too).

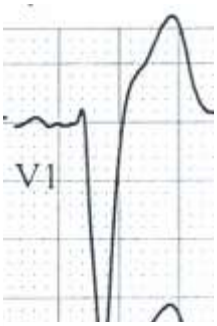
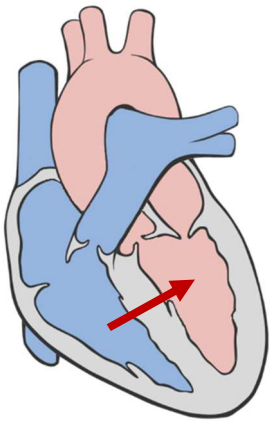


Figure 3

LBBB can exhibit one of two morphologies in Lead V1: a **QS** morphology (shape) or an **rS** morphology. The ECG (Figure 1) above manifests a QS morphology – there is no tiny r wave present in Lead V1. Here on the left (Figure 3) is a cLBBB with an rS morphology in Lead V1. We'll talk more about this tiny r wave later.

Although Lead V1 is always very important in determining whether a right or left bundle branch block is present, other leads are also important in supporting your diagnosis.

Let's look at what happens when the left bundle branch is :blocked"…



First, the ventricles are activated ***in succession – not simultaneously***. The left ventricle – *whose conducting system is blocked* (Figure 2) – must be activated from the right – *after the right ventricle has been activated*.

So, the left-sided leads (I, aVL, V5, V6) see the depolarization vector heading directly for them. That produces a QRS morphology in each of those leads that is *very characteristic* – **wide (≥ 0.12 second) QRS** represented by a **monophasic R wave** that is **notched**. Let's look at the ECG again…

Figure 4

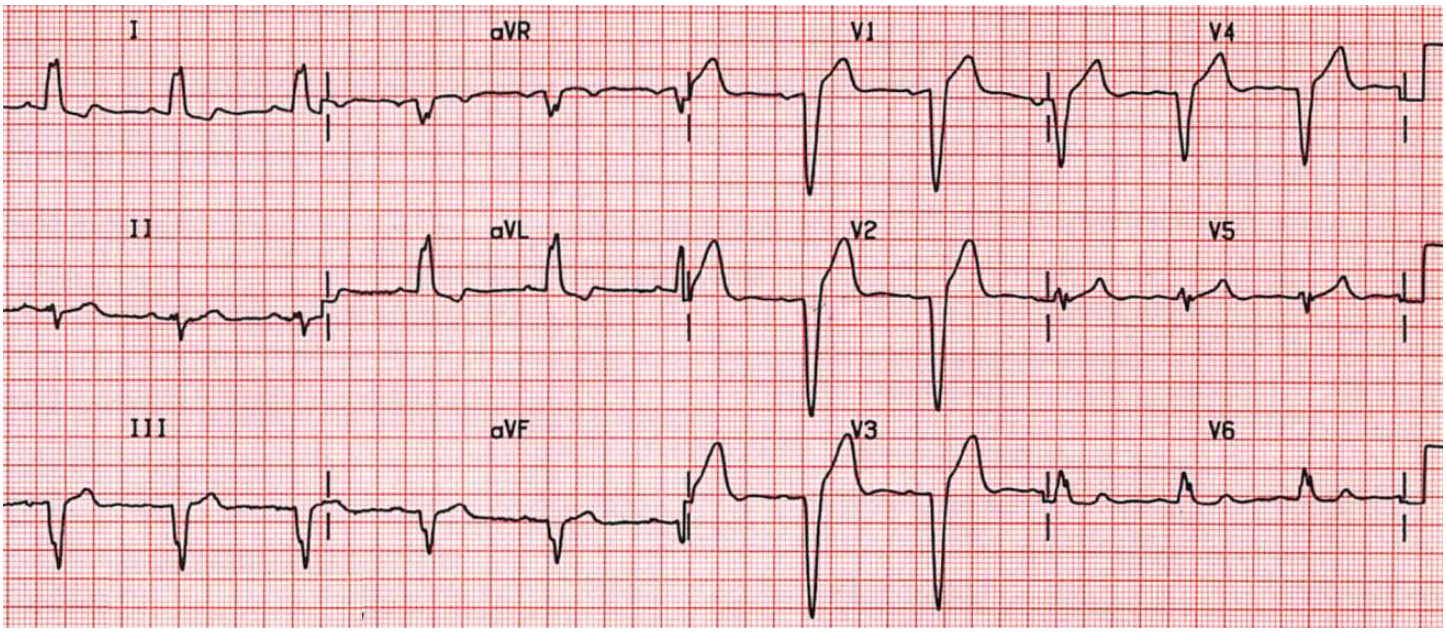


Figure 5

Look specifically at **Leads I**, **aVL** and **V6**. *Wide*, *monophasic* and *notched*!

Lead I is a **wide**, **monophasic** (only on one side of the baseline) **R wave** that is **notched** at the top. *Classic* for cLBBB! **Lead aVL** looks the same – *classic!* **V5** doesn't look anything like we expected! Well, we can't win them all! (*Don't worry about it! It looks the way it does because it's the transitional lead.*) **Lead V6** looks just as we expected – *classic!* Of Leads V5 and V6, when we are deciding on the presence of a bundle branch block – *or not!* – Lead V6 is by far the *most important* of the two. And remember: *Leads I and V6 should always look very similar* (i.e., same *polarity* – positive or negative, or same *biphasic morphology*). If not – then you have missed something! Go back and find out what you missed.

So, this is a *classic* cLBBB…

QRS duration ≥ 0.12 seconds (120 msec)
QS or rS morphology in Lead V1
Notched, monophasic R wave in Leads I, aVL, V5 and V6 (all the left-sided leads)

Now, you may have noticed that the QRS complexes in all the LEFT-SIDED LEADS that manifest the changes of complete LBBB (cLBBB) have another strange finding: a rather odd-looking *repolarization pattern*. When anyone says ***repolarization pattern***, they are referring to the appearance of the **ST segment** and **T wave**. If I want to draw someone's attention to the ST segment and T wave, I'll say, "Let's look at *repolarization*" (it's shorter and sounds smarter!).

As for the LEFT-SIDED LEADS I, aVL and V6 (remember: V1 is also a RIGHT-SIDED LEAD – V2R), in those leads we can see that the ST segment immediately begins to slope downward from the J point (the end of the QRS complex) into an inverted T wave. But look closely at the T wave – it is certainly NOT symmetrical. The *downward slope* is very *gradual*, but the return to the baseline (*upward slope*) is rather *abrupt*! This is *classic* for a **repolarization abnormality**!

The downward slope usually begins at the baseline because that's where the J point is *usually* found. Just remember that *the downward slope* ***always begins at the J point*** **– whether it is *on the baseline* or *slightly below it or above it***.

Don't make the mistake of thinking this represents *subendocardial ischemia* or a *non-STEMI*. You don't need to disturb the cardiologist in the middle of the night for this (*nor in the middle of the day*, for that matter). While this pattern is called a repolarization *abnormality*, it's an "abnormality" because most normal QRS complexes do not have such repolarization changes. But in actuality – *it is expected when a bundle branch block is present*. In fact, if you think you are seeing a bundle branch block – *but there is no repolarization abnormality* – then you are ***NOT*** looking at a bundle branch block. If a complete bundle branch block (in this case a left bundle branch block) is present, there ***MUST*** also be a repolarization abnormality present! Let's take a closer look…

A repolarization abnormality is what we call a ***secondary change***. That means that *repolarization* is abnormal because *depolarization* (the QRS) was *abnormal*. Just look at that QRS in Figure 5. That is NOT a normal QRS! ***Abnormal repolarization*** is ***secondary*** to ***abnormal depolarization***.

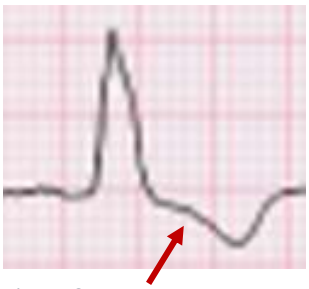
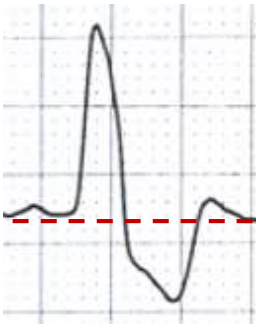


Figure 6

This snippet (Figure 6) is from a Lead V5. Here we see that the J point ends about 1 mm below the baseline. *It is not at all unusual for a repolarization abnormality to begin about 1 mm below the baseline*. Note that the downward slope of the inverted T wave is *very gradual* while the return to the baseline is *relatively abrupt*. Also note that there is a *slight upward convexity* present (arrow). Although that convexity is very typical, it's not *always* there.

Occasionally, with conditions that result in repolarization abnormalities, the return of the T wave to the baseline actually "shoots past" the baseline and there is a positive ending to the T wave. We call that ***overshoot***. Here is an example of "overshoot" (Figure 7)…



The end of the T wave has gone beyond the baseline and finished in positive territory. *We only call this **overshoot** when a repolarization abnormality is present*. Repolarization abnormalities ALWAYS occur with 1) **complete bundle branch blocks**, 2) **PVCs** and 3) **pre-excited QRS complexes**. They SOMETIMES occur with *ventricular hypertrophy*, particularly in its *advanced* stages. (Did you notice that *none* of those conditions have a *normal* QRS?)

Figure 7

Here's a ***PEARL*** for you…

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*You may have noticed that Leads V1 through V4 have QS complexes with elevated J points. **This is not an acute MI!** While this pattern is very typical of cLBBB, it is not directly caused by the left bundle branch problem. cLBBB **directly** causes the **wide, monophasic, notched R waves in Leads I, aVL, V5 and V6**.*

*The changes in V1 – V4 (usually just V1 – V3) are **reciprocal changes** to the ST depression and T wave inversion in the left-sided leads.*

*If you have a patient with chest pain and their ECG looks like this one, I recommend the **Sgarbossa Criteria with the Smith Modification** for determining if an acute MI is present.*

Just be aware that the J point elevation in V1 – V3 during cLBBB is usually more than the J point depression in the lateral leads. They do not reflect each other exactly.

I have been using the term left bundle branch *block*, but many examples of cLBBB really aren't blocks at all – they are just instances where *the left bundle branch is conducting more slowly than the right bundle branch*. Here's how that happens…

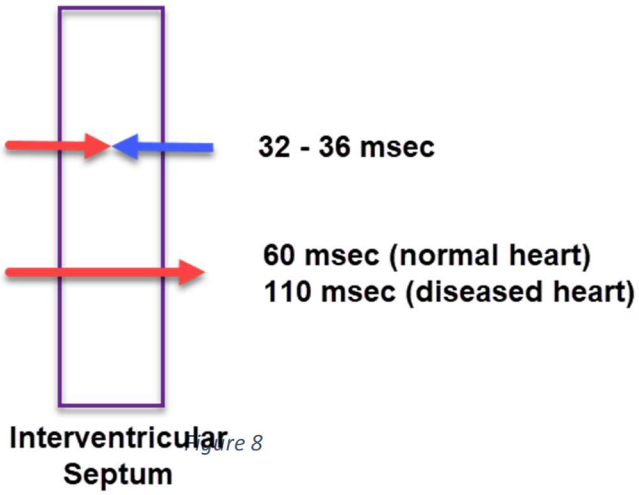

Figure 8

Normally, the septum is depolarized from BOTH directions because BOTH bundle branches are working just fine (Figure 8). Therefore, the depolarization of the septum represents the initial depolarization of both ventricles and it is very rapid, taking somewhere between just 32 and 36 msec.

But when one of the bundle branches is "blocked" (in our case, it's the LEFT bundle branch), the depolarization of the left ventricle occurs AFTER depolarization of the septum, which now can occur only in ONE direction: *from right-to-left*. This will take about 60 msec in a *normal* heart and about 110 msec in a *diseased* heart.

But what if – instead of a *block* of the left bundle branch – we have a left bundle branch that conducts very slowly, for any of a number of reasons. And let's say that the right bundle branch conducts fast enough to arrive at the mid-septum and then begin activating the septum from right-to-left *before* the impulse traveling down the left bundle branch can reach that area. Following that, it begins the activation of the left ventricle *before* the left bundle branch has a chance to get started.

Here's another ***VERY IMPORTANT PEARL*** for you…

QRS complexes in cases of left bundle branch block and right bundle branch block look the way they do NOT because one of the bundle branches is BLOCKED, but rather ***because one ventricle was activated BEFORE the other!*** *THAT is why there may appear to be a complete left bundle branch block when there really is NO BLOCK AT ALL! You will see this happen a lot with fascicular blocks (but that's in another article at the end of this series).*

So, when you see a "complete LBBB," you may actually be seeing a functional, but slowly conducting, left bundle branch and *not* a true block. How do we know this might be the case?

Here is an actual example: A patient with a baseline cLBBB developed episodes of a very lethal ventricular tachycardia called *bundle branch reentrant tachycardia* in which the right bundle branch is one limb of a reentrant circuit, and the left bundle branch is the other limb. The treatment – which is *very* effective – consists of *ablation of the right bundle branch*. That's right! The right bundle branch is *destroyed* to prevent another episode of the dysrhythmia from possibly killing the patient. In this patient's case, that would mean a *complete heart block* (cLBBB + cRBBB = complete heart block), so the electrophysiologists were prepared to insert a permanent pacemaker. But guess what happened when they ablated the right bundle branch? The patient's heart kept on pumping but now with a cRBBB! *The left bundle had been functional all this time* but, since it conducted more slowly than the right bundle branch, it never had a chance to demonstrate its functionality!

What causes a cLBBB?

Well, *infarction* can definitely cause it. A small infarction that occurs in the *proximal* portion of the left bundle branch *before* it bifurcates into the fascicles could easily cause a cLBBB. If the infarction were any lower, it would have to be *massive* in order to disrupt all the fascicles (Figure 2).

Probably the most common cause of cLBBB is *fibrosis*. Fibrosis can be due to aging – most people with cLBBB are older. It can be also be due to *ventricular hypertrophy*. Much of the hypertrophy

is not due to enlarged myocytes but rather to the deposition of fibrin in and among the muscle fibers which contributes quite a lot to the enlargement of the ventricular wall.

Here's another ***PEARL*** for you…

You are probably already aware that the ECG is NOT the best method for diagnosing left ventricular hypertrophy (LVH). Sensitivity is very low. But what if I told you of a sign that was very sensitive and very specific? Close to 95% of all patients with cLBBB have some degree of left ventricular hypertrophy. If you see a patient with cLBBB, you can say that he or she also has LVH and you would be wrong only about 5 times out of 100.

Congenital cLBBB is *very rare*, so when you see cLBBB on an ECG, you can assume that the patient has a diseased left bundle branch. Even if it is still functional, it is diseased because it cannot conduct as it should.

At the beginning of this article, I mentioned that the QRS in Lead V1 can appear as two different *classic* morphologies: a **QS complex** or an **rS complex**. But only one at a time – you won't see both in the same tracing! Why is that? A cRBBB has only ONE *classic* morphology (rSR').

A **QS morphology** really is the *most classic* of the two morphologies in Lead V1. The **rS morphology** is caused by something called a *proximity effect*: the proximity of the V1 electrode to the surface of the heart allows it to detect a small anterior force at times, but only *under certain circumstances*. Now, a *normal* QRS in Lead V1 *also* has an rS morphology – but the "r" wave in a *normal* rS is caused by a specific, *real* depolarization vector (called vector #1). When you are looking at an ECG with a cLBBB and you see a small "r" wave in Lead V1 (rS complex), just think of that little "r" as an *illusion*. ***There is never a normal r wave present in Lead V1 during cLBBB!***

I hope you have learned something from this introduction to left bundle branch block. In the next installment in this series, I will discuss **right bundle branch block**.

One last **PEARL (a *lagniappe* – a little something extra!)**…

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*A **PRIMARY** repolarization abnormality is due to effects occurring at the **cellular** level – ischemia, hypoxia, acidosis, electrolyte abnormality or medication/drug effect. The QRS is usually normal – it's the repolarization itself that is abnormal.*

*A **SECONDARY** repolarization abnormality is due to a more **macroscopic anatomic effect** – causing abnormalities in the **path** of the depolarization AND repolarization impulses, such as widening of the ventricular wall, fibrosis, infarcted areas, infiltrative disease and more. Both **depolarization** (QRS complex or even P wave) as well as **repolarization** (ST segment and T wave) **will appear abnormal**.*
