

Delays and Blocks Involving the Bundle Branches

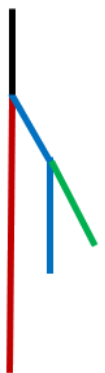
Part 1 – *Non-Specific Intraventricular Conduction Delay*

Usually, I begin with an ECG or a diagram, but for this monograph, I want to discuss a little vocabulary first so we will all be on the same page.

This topic is about **non-specific intraventricular conduction delay (IVCD)**. Often the “D” is said to represent “Defect,” but I personally prefer “Delay.” Note that I used the word **INTRA**ventricular and not **INTER**ventricular. That’s because “INTRA” means “within” while “INTER” means “between or among,” involving more than one area. Business that takes place only *within* the state of Texas is *intrastate* commerce while business that occurs *among* Texas, Louisiana, Oklahoma and Arkansas is *interstate* commerce.

Therefore, *intraventricular* conduction delay involves **only one ventricle**. So, when we refer to intraventricular conduction delay, we are speaking of the left bundle branch and/or its fascicles OR the right bundle branch – *but not both*.

Now, let’s talk about the word, **fascicle**. You have already learned that the left bundle branch divides into the *anterior* fascicle and the *posterior* fascicle. There may also be a *septal* fascicle, but we need not concern ourselves with it. They are all contained within the left ventricle. The right bundle branch does not divide into fascicles. Here’s the problem: when you read about bundle branch blocks and fascicular blocks, the left bundle branch and the right bundle branch themselves are *also* often referred to as “fascicles.” So, there are four fascicles (five, if we include the *septal* fascicle) in the heart. I have made it a rule NOT to use the word *fascicle* when referring to the *right or left bundle branch*.

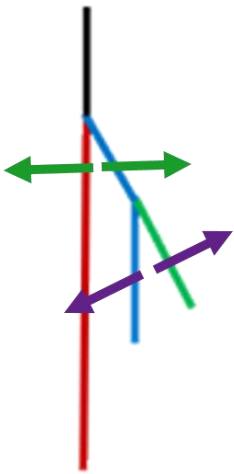


This diagram on the left represents the *bundle of His* (black), *right bundle branch* (red), the *left main bundle branch* and *posterior fascicle* (blue) and the *anterior fascicle* (green). The reason both the left main bundle branch and the posterior fascicle are blue is because many anatomists feel that the posterior fascicle is basically a continuation of the left main bundle branch while the anterior fascicle is a small branch off the main stem. Imagine the interventricular septum between the right bundle branch and the posterior fascicle. There are *no conducting pathways within the interventricular septum* – either longitudinally or transverse. All impulse conduction in

the interventricular septum is *cell-to-cell*.

We often think of the word “septum” as referring to a thin membrane or tissue. That is absolutely NOT the case with the interventricular septum. It is part of the left ventricle and is about as wide as its thick lateral wall. The initial portion – where the bundle of His enters the

ventricle – is a relatively thin membrane, but it rapidly develops into a thick, muscular wall of the left ventricle.



When an impulse arrives in the ventricles via the bundle of His, it divides and depolarizes the right bundle branch and the left bundle branch *simultaneously*. In the left ventricle, the anterior and posterior fascicles are also activated *simultaneously*. Activation of the right and left bundle branches results in impulses that are traveling in *opposite directions at the same time*. This causes most of the depolarization vectors to cancel themselves. We call this ***cancellation of forces***. The anterior and posterior fascicles are also depolarizing in opposite directions at the same time, so they, too, cancel each other. Now, when I say, “cancel each other,” I don’t mean that the depolarization waves simply disappear. The cancellation occurs when the ECG

machine inscribes the QRS interval on the ECG tracing. If you are looking at a QRS in Lead V1, you will (under *normal* circumstances) see something like this:



This is a *normal* QRS for Lead V1. See how *narrow* it is – about 80 msec in duration – which is quite normal. Bear in mind that this thin, little QRS represents a **HUGE** amount of ventricular myocardial depolarization – the entire right and left ventricles! But you can’t really SEE it because so many forces are cancelling each other on the ECG tracing. But... there *are* circumstances where you CAN see it! More on that in another installment...

OK. If you are a little lost at this point, go no further until you review what you just read.

This first installment is about ***non-specific intraventricular conduction delay*** (or ***defect***, if you prefer), so let’s get started! Sometimes the bundle branches and/or fascicles don’t conduct at the same speed. This is due to one or more abnormalities within the conduction system, and it is *never* normal! Such delays of conduction in parts of the conducting system cause the QRS to *widen* and *look a bit different*. By *conducting system*, I mean the *bundle of His, left and right bundle branches, anterior and posterior fascicles* and all the microscopic *Purkinje fibers* that transmit impulses to the myocardial cells.

Sometimes the widening is *less* than 0.12 seconds (120 msec) and sometimes it is *greater* than 0.12 seconds. In either case, the widening of the QRS doesn’t necessarily mean that a complete or incomplete bundle branch block is present. It isn’t just the *widening of the QRS beyond 0.12 seconds* that defines a bundle branch block – there must also be *specific QRS morphologies present in Leads I, aVL, V1, V5 and V6*. When we look for bundle branch blocks, we tend to focus on Lead V1 because it is the most important lead to inspect, but the others are very important, also.

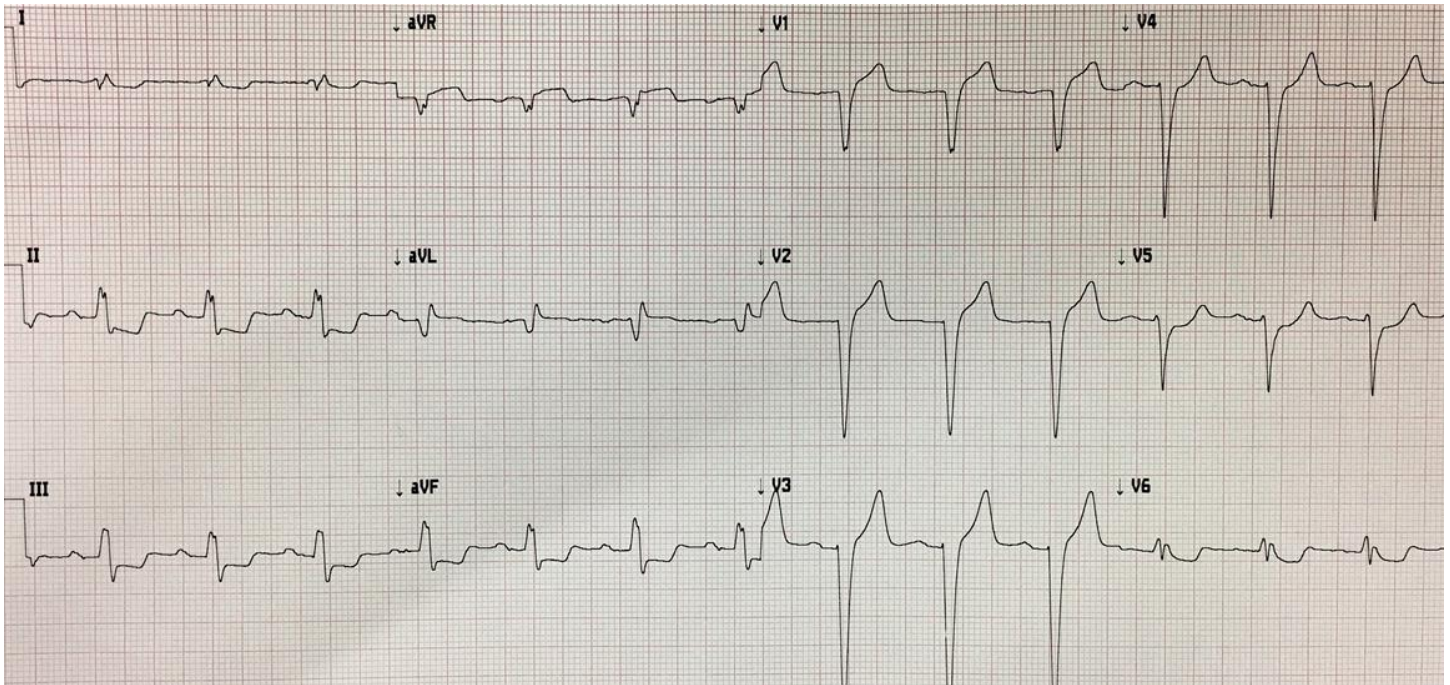
When the QRS widens between 0.10 seconds and 0.12 seconds it isn't *always* an incomplete bundle branch block. Sometimes the widening appears localized to just a few leads and sometimes it seems to affect all the leads. The key factor here is that it just doesn't look like an incomplete bundle branch block.

If the widening is greater than 0.12 seconds, the QRS complexes may or may not resemble a real bundle branch block – right or left. But here's the issue: while the QRS in Lead V1 may indeed be widened > 0.12 seconds, it may only resemble a right bundle branch block in that lead while it resembles a LEFT bundle branch block in other leads. It never qualifies as a true bundle branch block when all the usual leads are considered. Here is an example of a ***non-specific intraventricular conduction delay***...



As you can see, there is a generalized widening of the QRS but perhaps not quite 0.12 seconds. If you look closely, the initial deflection – whether it's an *upslope (R wave)* or *downslope (S wave)* – is very *vertical* and *smooth*. The widening appears to begin with the upslope of the S wave. This patient is in the subacute stage of a STEMI. Apparently, there is either stunned or infarcted myocardium that is causing a delay in one or more parts of the conducting system. If you look at Leads I, aVL and V1, you see a classic left bundle branch block (LBBB) pattern, yet the QRS is < 0.12 seconds in duration and there is an S wave (more properly, an s wave) in Lead V6. If this were a true LBBB, that should be a monophasic R wave with a notch on or near the peak. Lead V5 would look similarly but it has an even *deeper S wave*! This is a ***non-specific intraventricular conduction delay***.

Let's look at another one...



There is widening of the QRS *interval* (*correct* term for QRS *complex*) on this ECG to just past 0.12 seconds (probably close to 0.13 seconds). When we look at Lead V1, we see the typical QRS morphology of left bundle branch block (LBBB) – a wide QS complex. To go along with that we should see tall, notched, monophasic R waves in Leads I, aVL, V5 and V6 (especially in Lead V6). We don't. There are small, nearly equiphasic qr complexes in Leads I and aVL representing a remote (when we want to sound really smart, we say *remote* instead of *old!*) myocardial infarction. And Leads V5 and V6 certainly are not tall, wide, notched monophasic R waves! This ECG does NOT meet the criteria for a left OR right bundle branch block, yet the QRS duration is slightly prolonged. This represents a *non-specific intraventricular conduction delay*.

What causes a *non-specific intraventricular conduction delay*?

The usual suspects are

infarction,

ischemia,

left ventricular hypertrophy (LVH),

electrolyte disorders (especially ***hyperkalemia,***

medications and

acidosis.

There are probably a number of other causes, but these are the main ones with **prior myocardial infarction** being the *most frequent* culprit. Always think about these six causes of a non-specific intraventricular conduction delay when you encounter this phenomenon.

So, the next time you see QRS complexes that look like they should represent a bundle branch block – but just don't meet the criteria – you are most likely looking at a **non-specific intraventricular conduction delay**.

PEARL!

Beware! Too many people stop once they *note* a non-specific intraventricular conduction delay is present. **Don't fall into that trap! A non-specific intraventricular conduction delay (NS-IVCD) is an electrocardiographic symptom – not a diagnosis!** You haven't "diagnosed" anything until you at least consider the causes of a non-specific intraventricular conduction defect!

Want to improve your ECG interpretation skills? Are you *afraid* you are missing subtle myocardial infarctions? Do wide complex tachycardias make you *uneasy*? Do AV blocks and bundle branch blocks *confuse* you?

Join us for the **Advanced ECG Interpretation Boot Camp** here in Houston, Texas. Four days of intense study of 12-lead ECG interpretation and dysrhythmias. This course is for those with an intermediate skill at reading ECGs. If you understand the ECG portion of the ACLS, then you have an intermediate skill level. But these days, it takes *more* than an intermediate skill in ECG interpretation. The most common reason for medical malpractice litigation is *failure to diagnose*. And one of the most common missed diagnoses is *myocardial infarction* (and in some lists, it is Number 1!).

What makes this course so different? Why do people travel from Europe, Australia, New Zealand, South America and the Middle East to take this course in Houston, Texas?

It's the **active participation** in the interpretation of complex ECGs – up to **14 hours** of **in-class, active participation** during the 4 day course! Each participant gets to interpret ECGs while being guided and mentored by Dr. Jerry W. Jones, an internationally-recognized authority on electrocardiography instruction.

Come join us and be a PARTICIPANT... never just an audience!

<https://medicusofhouston.com/aeibc>