Most people quickly recognize right bundle branch block (RBBB) because of its distinctive appearance. Beginning in the early 1950’s when an ICU nurse commented to Dr. Henry Marriott that the QRS in Lead V1 looked like “rabbit ears” to her, this “block” has been saddled with the moniker of “rabbit ears.” But therein lies a problem: those rabbit ears are not always present! You certainly don’t need them to diagnose a right bundle branch block. Before we go any further, let’s see what causes a right bundle branch block...

The His bundle exits from the AV node and, as it enters the ventricles, it divides into two branches – the left bundle branch (which we discussed in the last installment) and the right bundle branch. The left bundle branch subdivides again into the anterior fascicle and the posterior fascicle. We will discuss them in the next installment.

The right bundle branch is responsible for the activation of the right ventricle. The two bundle branches are normally activated almost simultaneously, providing an effective, physiologic heart beat.

But it also serves another function. Many people mistakenly think that the left bundle branch is responsible for depolarization of the interventricular septum (henceforth, the “septum”). But that’s not entirely true! The right bundle branch is also responsible for the depolarization of the septum. If the left bundle branch were the only source of septal depolarization, it would take 60 msec in a normal heart and 110 msec (or more!) in a diseased heart to fully depolarize it. But
with the active participation of the right bundle branch in the depolarization of the septum from the right (it starts just a couple of msec after the left bundle branch), it only takes about 36 msec to complete the depolarization of the septum.

But what happens when the right bundle branch is blocked*? Well, in that case, the left bundle branch is the only source of septal depolarization.

*Like the left bundle branch, the right bundle is not always actually blocked, but rather it sometimes conducts slower than the left bundle branch.

The left ventricle is fully depolarized first, followed by the right ventricle. Since there are no conducting fibers that cross the septum from left-to-right, conduction is cell-to-cell. By the time the depolarization impulse reaches the right ventricle, the left ventricle has been depolarized and a deflection – an R wave – has already been inscribed on the ECG tracing. Now it’s the right ventricle’s turn to depolarize. It does, and the ECG machine inscribes a second R wave on the ECG tracing. But there seems to be a BIG difference in those R waves!

The first R wave – representing left ventricular depolarization – is quite small. In fact, it’s so small that we designate it with a lower case “r.” The second R wave – representing right ventricular depolarization – is much larger. It gets to keep its upper case “R” designation.

It is well known that the walls of the left ventricle are very thick – and that includes the septum which is just another wall (or surface) of the left ventricle. The walls of the right ventricle are about 1/3rd the thickness of the left side. Here is an illustration that demonstrates the difference…

As you can see (Figure 3), the walls of the left ventricle – including the septum – are very thick. That means there should be a lot of voltage being recorded on the ECG tracing. Remember: the vertical axis of the ECG tracing measures voltage, so the taller (or deeper) the deflection, the more voltage is involved. And that usually means more myocardial muscle mass. So why is the R’ wave (we use the “prime” symbol to designate the second R wave) of the right ventricle so much larger than the r wave of the left ventricle?

The opposite walls of the left ventricle are massive, but… they are all depolarizing at about the same time. Now, do you see where this is going? The opposing walls of the left ventricle are depolarizing in opposite directions, so much of the current generated will be cancelled. Thus, the remaining amount of current available to be seen on the ECG tracing is much less than that
of the right ventricle. The current is really there – and there is a lot more of it than in the right ventricle – but the cancellation of forces has resulted in a much smaller deflection.

Even though the depolarization of the septum is performed by the left bundle branch alone, it is still a left-to-right depolarization, which is physiological. That’s good news! That is the normal direction of septal depolarization. There will still be a small “septal r” in Lead V1 and small, physiologic “septal q waves” can still be seen in the left-sided leads (sometimes in the inferior leads).

Are there any consequences of RBBB? Usually, nothing worrisome. Some people are born with congenital RBBB and they seem to do just fine. It is usually not associated with the degree and severity of disease that accompanies LBBB. However, there can be problems...

The left anterior descending artery (LAD) is the main (though not only) supplier of blood to the bundle branches. If a RBBB occurs during the course of an acute anteroapical (formerly anteroseptal) MI, then the occlusion is in the proximal LAD with a very large area of myocardium at risk and a high mortality. That is because the septal perforating branches of the LAD, which are usually the first branches from the artery, supply the blood to the right bundle branch.

There are two types of RBBB – complete RBBB (cRBBB) and incomplete RBBB (iRBBB). By definition, a cRBBB has a QRS duration of 0.120 seconds (120 msec) or greater. An incomplete RBBB will have a duration between 0.101 seconds and 0.119 seconds (in other words, from 100 msec to 120 msec, but not including those values).

This is what an incomplete RBBB looks like:

Look familiar? We’ve all seen the tiny r’ at the end of the QRS in Lead V1. They seem to be ubiquitous. Here’s the deal: the QRS duration in this snippet is 110 msec – right between 100 and 120 msec. That officially makes it an incomplete RBBB. Often, we see this small r’ in Lead V1, but the QRS duration is less than 100 msec! That’s often because the ECG technician incorrectly put the adhesive electrodes for Leads V1 and V2 one or two interspaces too high. In those cases, assuming that an incomplete RBBB wasn’t already present, the QRS duration should not be more than 100 msec (0.10 seconds). There are a few situations where you are likely to see this rSr’ morphology...

1. The aforementioned incorrect electrode placement (all TOO common!)
2. Termination of the impulse in the outflow tract of the RV (That’s OK – it’s normal)
3. Slight delay of conduction through the right bundle branch (occasionally)
4. Acute Pulmonary Embolus!
I don’t generally get too concerned about iRBBB unless I suspect a pulmonary embolus.

Another way to determine if the adhesive electrodes were placed too high is to look at the P waves in Leads V1 and V2. If you see an rSr’ in Lead V1, but the P wave in Lead V1 is completely negative and the P wave in Lead V2 is either negative or biphasic, then you know the electrodes were placed too high. And again, the QRS duration should be no more than 100 msec. This is a very, very common problem. I recently went through about 30 consecutive ECGs in my collection before finding one in which the electrodes for V1 and V2 were applied correctly.

Now, let’s talk about rabbits! If you think that complete RBBB is defined by those rabbit ears, well, you are going to overlook a lot of cRBBBs! Here is what is needed to define a cRBBB...

1. QRS duration ≥ 120 msec (≥ 0.12 seconds)
2. Terminal R wave in Lead V1
3. Wide slurred S wave in Lead I and Leads aVL and V6 (but Lead I is enough!)

That’s it! There does not have to be an R’ at all! (I know... you probably feel like I just shot the rabbit! You’ll get over it.) If you want proof, take a look at the ECG snippet in Figure 5...

This is a cRBBB without rabbit ears! There is a terminal R wave – and since it is the only R wave, it is not an R’ - it is an “R.”

Take a good, long look at this QRS complex (inscribed in Lead V1, specifically). If you have recorded the ECG on a patient with respiratory difficulties, you are likely seeing the electrocardiographic sign of marked right ventricular strain. Should the patient be in distress from an acute PE or pulmonary hypertension, this QRS morphology portends a very high in-hospital mortality!

One last myth to burn (figuratively): a ventricular tachycardia with a triphasic, upright QRS in Lead I will demonstrate an initial R wave that is taller than the terminal R’ (OK... the left rabbit ear is taller than the right). That is true! So then, if the R’ is taller than the initial R wave, then there is a classic cRBBB present and the tachycardia must be due to an SVT with aberrancy. Right?

Do you mean a classic rSR’ in Lead V1 like this one?

This snippet is from a documented ventricular tachycardia! So, an ectopic rhythm from the left ventricle CAN produce a classic rSR’ in Lead V1. Reentrant tachycardias involving the bundle branches and/or the fascicles of the left bundle branch can produce a perfect, classic rSR’ in Lead V1 no wider than 120 msec!
Here’s a hint that should have made you suspicious that something was not quite right about that QRS complex. Look at the repolarization abnormality. Remember in the last installment on “Left Bundle Branch Block” how the repolarization abnormality for upright QRS complexes is characterized by an *upwardly convex descent* (here is Figure 2 reproduced):

It’s a subtle finding *but a very important one.*

Here are a few more examples of ventricular tachycardia with an rSR’ (or similar) in Lead V1...

I hope you learned something from this installment on “Right Bundle Branch Block.” The next installment in this series will be “Fascicular Blocks.”

I have resumed presenting the **Advanced ECG Interpretation Boot Camp** here in Houston, Texas. This will be followed by the **Masterclass in Advanced Electrocardiography** and the **Masterclass in Advanced Dysrhythmias.** People are already signing up. Don’t miss a great opportunity to take your ECG interpretation expertise to a much higher level!!

All are four-day, live and in-person classes which which feature in-class, active participation by all attendees. Watch for my announcements on my website!

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

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