

Ectopy or Aberrancy? Or Something Else?

Discussion

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24-May-1924
Female

Vent. rate 96 bpm
PR interval * ms
QRS duration 72 ms
QT/QTc 368/464 ms
P-R-T axes * 57 35

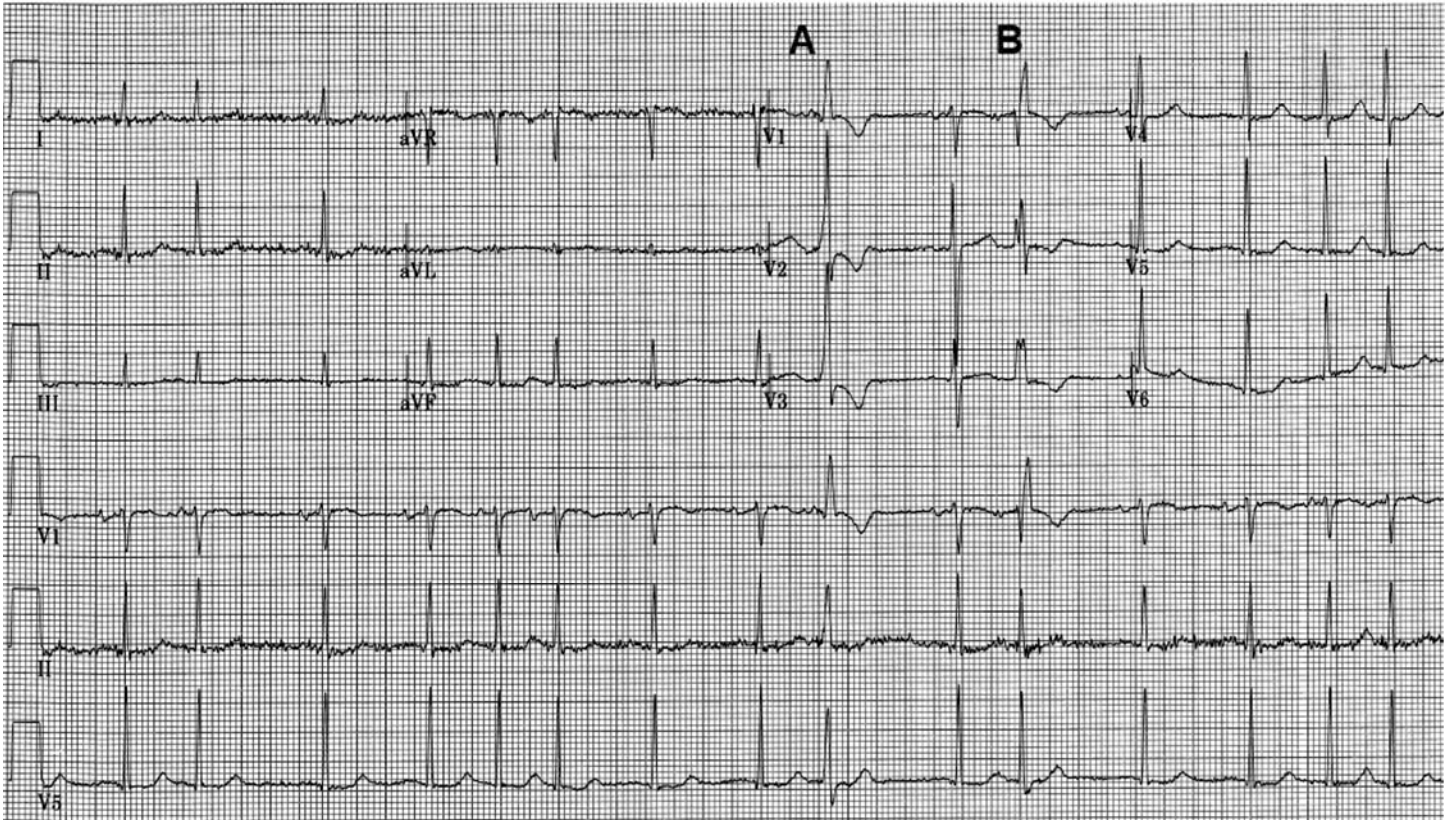


Figure 1

One of the most difficult issues for beginners in electrocardiographic interpretation involves deciding whether a beat is **ectopic** or **aberrant**. That is the topic of the discussion in this monograph. I think you will find there is something here for both newbies and more experienced electrocardiographers alike.

An *ectopic beat* is an impulse that did not arise in the sinus node. Ectopic impulses can arise just about anywhere: in the atria, the AV node/junctional area or in the ventricles. They can originate in regular working myocardium or in specialized conducting fibers.

This ECG is perfect for this discussion because it presents us with two (*spoiler-alert!*) aberrantly-conducted beats – one is a classic RBBB and the other a non-classic RBBB. We will begin with a short discussion about atrial fibrillation and the Ashman-Gouaux phenomenon, commonly

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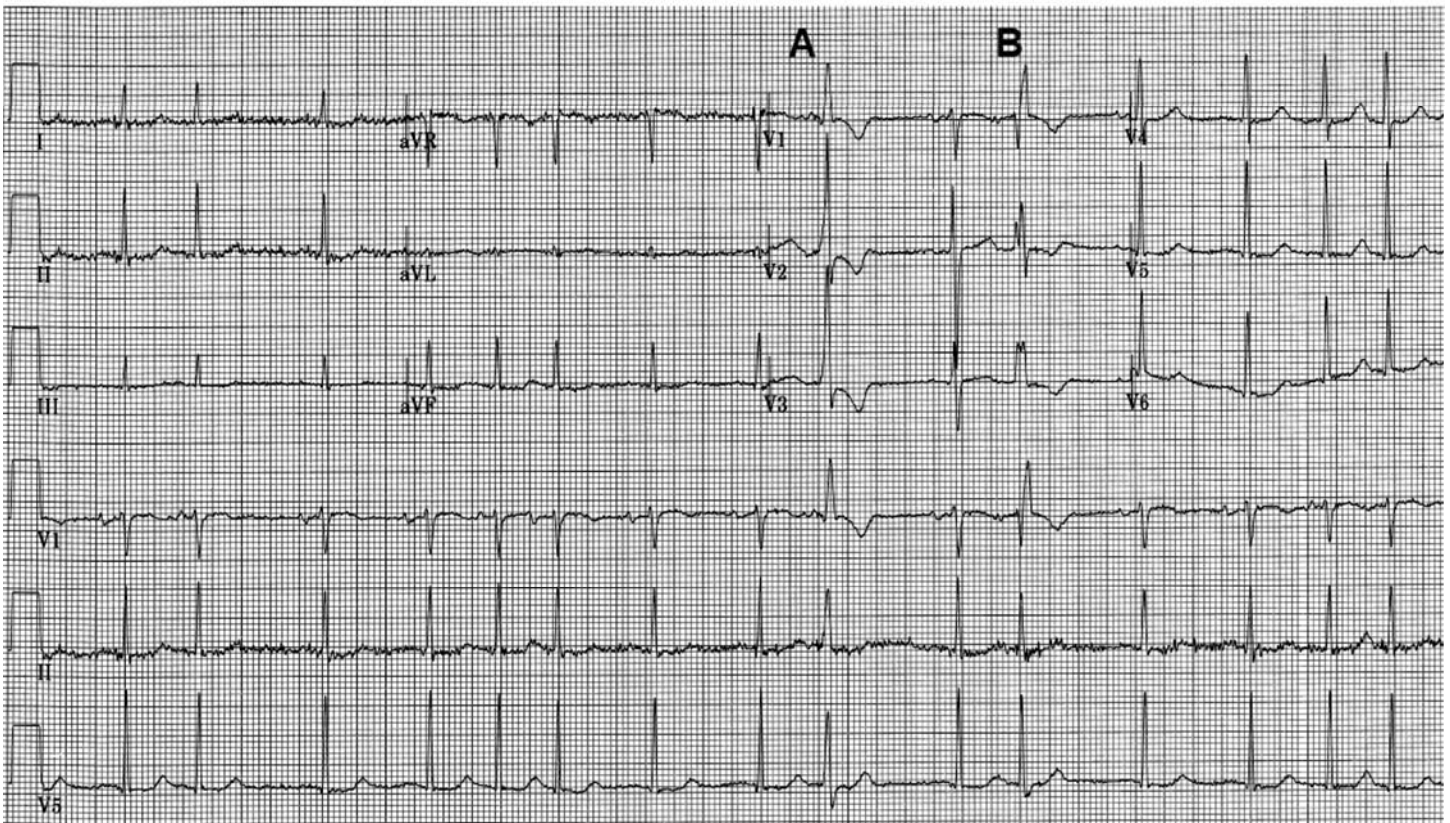


Figure 1 (repeated)

known as the Ashman phenomenon because so few people can pronounce Gouaux correctly (Goo-OH).

Drs. Ashman and Gouaux, while doing some research in New Orleans, La. noted that during atrial fibrillation, if a long R-R interval was followed by a short R-R interval, the QRS ending the short R-R interval was frequently abnormally widened. This led to a major discovery...

What they discovered was that **each R-R interval determined the length of the refractory period of the *following* R-R interval**. What better environment for many occurrences of long-short R-R intervals than a heart in atrial fibrillation?

But this concept is not limited to atrial fibrillation – it can also occur during other rhythms. This ECG is neither atrial fibrillation nor is it a true sinus rhythm. What do you think it is?*

If we look at the aberrantly-conducted QRS labeled “B” in Lead V1, we see a classic rSR’ of complete RBBB. The r wave is very tiny, but it’s there! Now let’s look at Lead V2. Here, the QRS *looks like* a RBBB, but this is not so classic – it consists of an R-R’-S; that is, an R wave followed by an R’ wave followed by an S wave. It doesn’t matter how many R-primes (R’) are present: **if the QRS interval ends with an S wave of any size – it is not a RBBB morphology!** (Exception: Leads I, aVL, V5 and V6 may end with S waves, **but NOT any right-sided leads!**)

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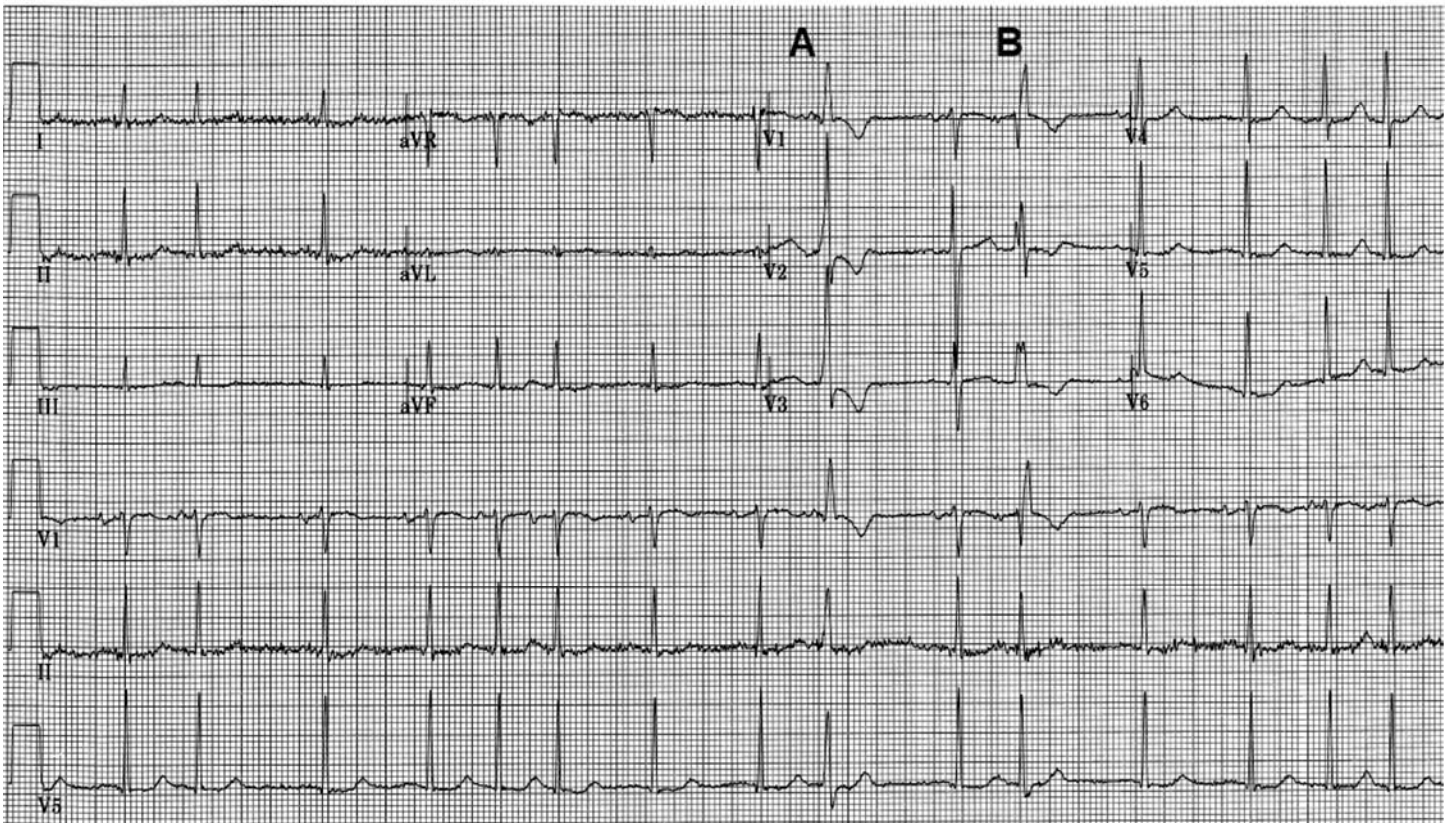


Figure 1 (repeated)

While Lead V2 often continues the RBBB morphology with perhaps a small amount of variation, it occasionally doesn't, and this is one of those times. Leads V1 and V2 are considered "right-sided" septal leads with regard to precordial leads V1 – V6. Lead V3 is more of an *apical* lead and shouldn't be considered a right-sided precordial lead. As you can see, the aberrantly-conducted QRS ("B") in Lead V3 is a **monophasic R**. This is also a variant morphology for RBBB, but it is certainly not classic. More importantly, ***there is no terminal S wave!***

If we look below QRS "B" in the V5 rhythm strip, we see a classic morphology of RBBB that typically appears in Leads V5 and V6: a tall R wave with a wide, slurred s wave. The R/S ratio in Leads V5 and V6 should be > 1.0.

So, what has happened here? The R-R interval that began with beat "A" and ended with the next QRS resulted in a relatively long R-R interval. Then beat "B" appeared, resulting in a much shorter R-R interval. By appearing when it did, the impulse *may* have arrived during the ***relative refractory period*** of the right bundle branch and conduction in the right bundle branch was slowed long enough for the left ventricle to complete depolarization and then cross the septum

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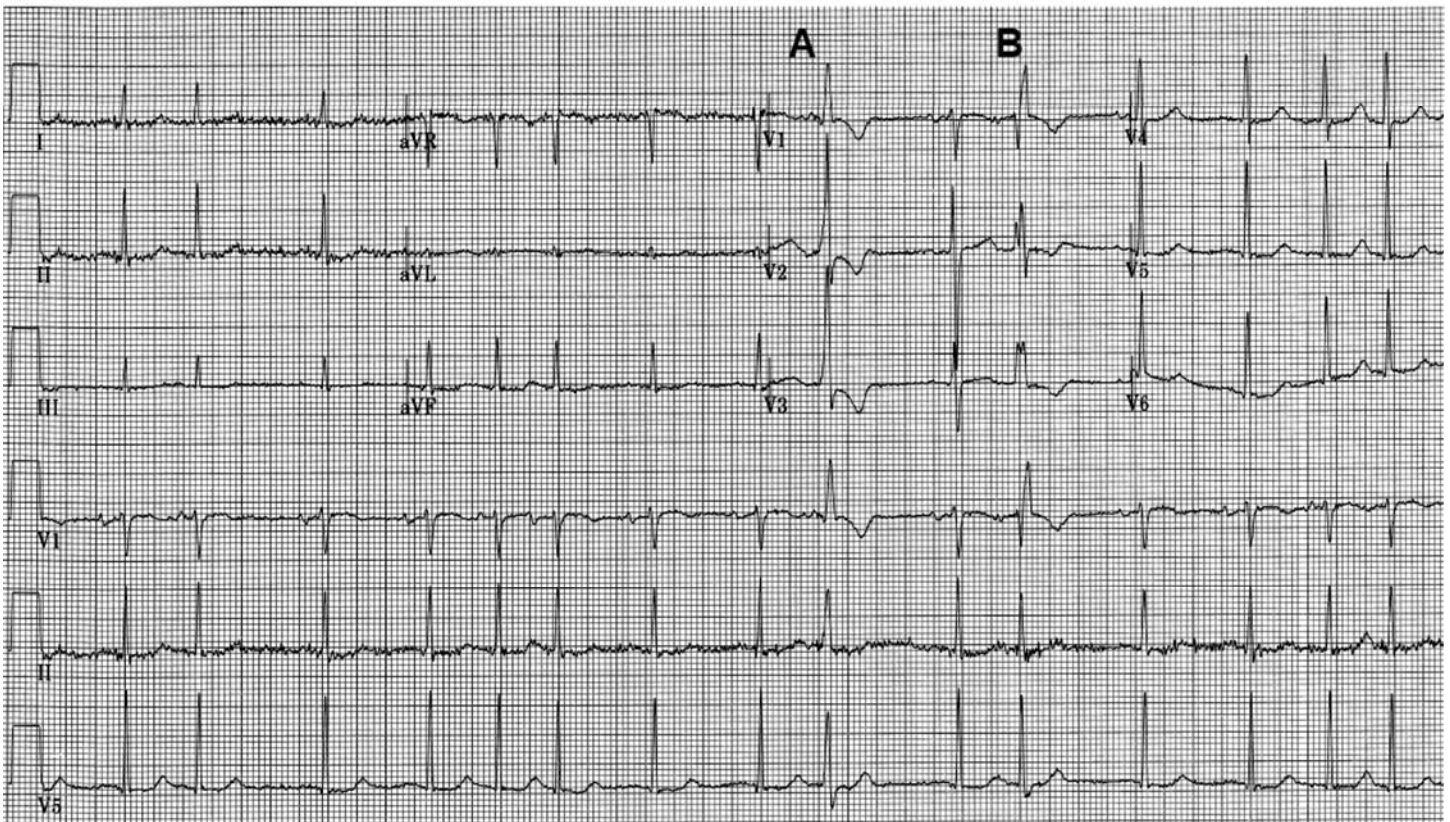


Figure 1 (repeated)

to the right ventricle, which was then depolarized *after* the left ventricle. Alternatively, it is *also* possible that the impulse arrived in the right bundle branch during the **absolute refractory period** and conduction in the right bundle branch failed completely.

Remember: there cannot be a terminal S wave in a *classic* RBBB in the right-sided, septal leads (V1 and V2). The R' represents depolarization of the right ventricle. A terminal S wave – in this case – would suggest that the left ventricle is being depolarized *again* without any repolarization (indicated by the T wave) having occurred yet! Impossible!

Now let's look at aberrant QRS "A" in Lead V1. It is a monophasic R wave. If you enlarge the tracing considerably, you might detect a very tiny q wave preceding the R wave. For the purpose of discussion, let's consider it a *monophasic R wave*. The monophasic R wave is followed by an inverted T wave (as expected in any bundle branch block that ends with a positive deflection).

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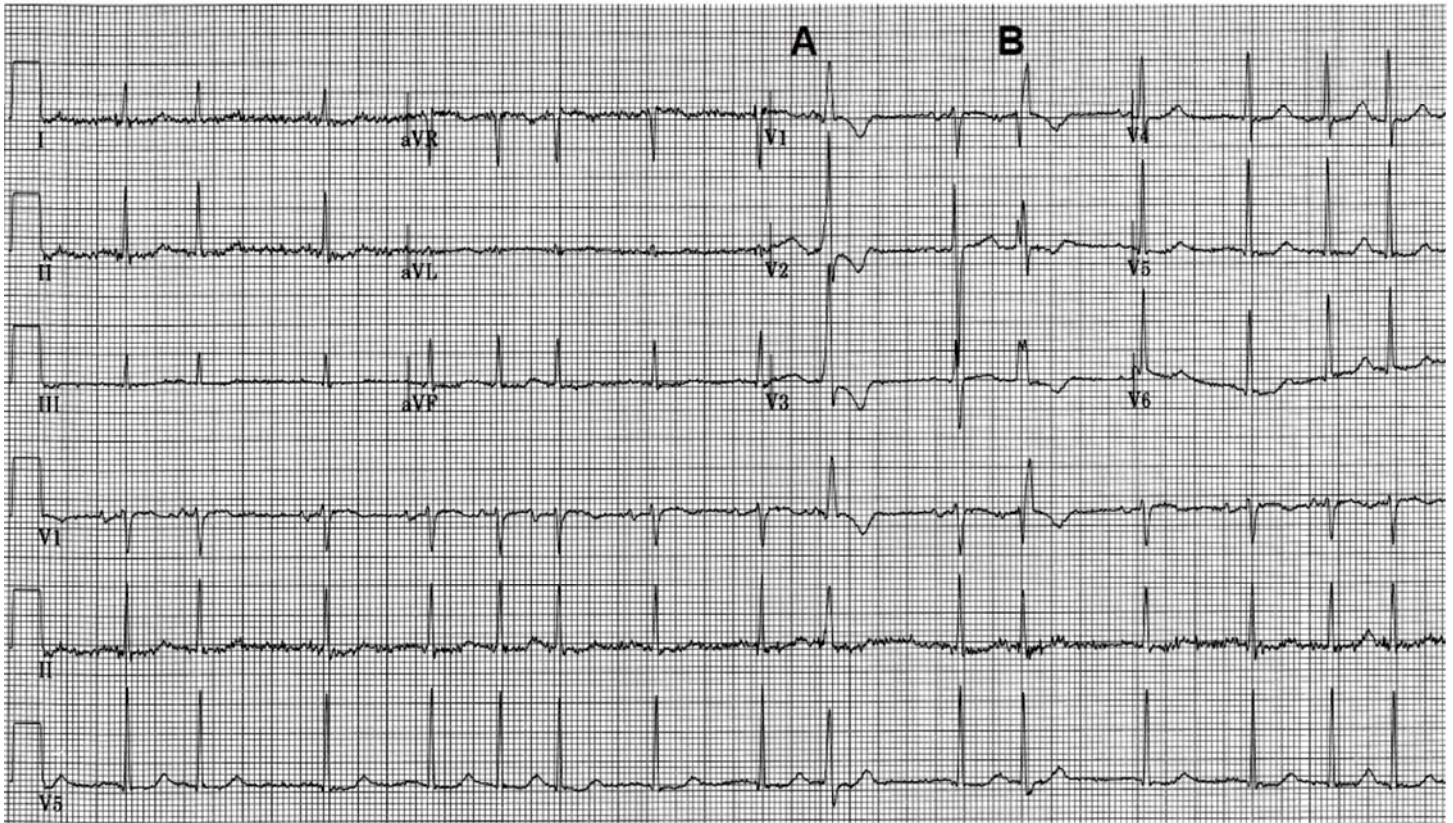


Figure 1 (repeated)

Now let's look at what is happening in Leads V2 and V3. In those leads we also see a tall R wave. But both also terminate with an easily visible S wave. And then, both QRS intervals are followed by an inverted T wave. These T waves are significantly inverted.

As a general (but not absolute) rule, a T wave following an aberrant or ectopic beat is usually opposite the terminal deflection. When it appears *otherwise*, you must consider the presence of *another pathological process*, such as ischemic heart disease.

Here are examples of classic RBBB from Lead V1 (Figure 2) and classic LBBB from Lead V6 (Figure 3).



Figure 2



Figure 3

Look at the ST segments and T waves of both complexes. In RBBB (Figure 2), the ST segment begins sloping down from the J point which is usually located on or within about 1 mm below the baseline. In LBBB (Figure 3), the ST segment begins sloping down from the J point which is usually located *below* the baseline*. This is called a **secondary repolarization abnormality**; but in spite of its name, it is not really *abnormal* in the presence of a bundle branch block. It is expected over **right-sided leads** in RBBB and over **left-sided leads** in LBBB. Note that the polarity of the T waves is *opposite* the last deflection of the QRS.

*Although the J point is often depressed in secondary repolarization abnormalities, quite frankly, this is *too much* J point depression. We typically allow up to 1 mm of J point depression in bundle branch block patterns with an inverted T wave – *but no more*. Even this example suggests the inclusion of a *primary* repolarization abnormality *in addition to* the *secondary* repolarization abnormality. (YES! You can sometimes see BOTH in the same deflection.)

Now let's focus on the repolarization abnormalities for QRS "A" in Leads V2 and V3. The T wave is indeed inverted, but the terminal deflection of the QRS complexes is an S wave. If this were an s wave less than 1 mm, I wouldn't be too concerned, but these are significant, easily-seen S waves! This most likely represents a **primary repolarization abnormality**.

What's the difference between a **primary** and a **secondary** repolarization abnormality? A **secondary repolarization abnormality**, as seen in *bundle branch blocks*, *premature ventricular complexes* and *some* instances of *ventricular hypertrophy* and *ventricular pre-excitation*, is due to *alterations in the path* that repolarization must take due to the fact that *depolarization also took an abnormal path*. A **primary repolarization abnormality** is due to *pathology at the cellular level*, such as *ischemia*, *acidosis*, *substance toxicity* and *severe electrolyte derangements*, to name a few. Primary repolarization abnormalities result in a T wave that is concordant with the main or terminal deflection of the QRS. Secondary repolarization abnormalities *always* follow an abnormal QRS; primary repolarization abnormalities *usually* don't (though they CAN, as in the example in Figure 3!).

Both these instances represent aberrant beats. Aberrancy – about 99.9% of the time – refers to bundle branch block. RBBB aberrancy is more common because the right bundle branch has a longer refractory period than the left bundle branch. That is why a premature beat is more likely to encounter the *relative* (or even *absolute*) refractory period of the right bundle branch.

Because aberrant beats begin normally, the first half of an aberrant beat – *especially if classic!* – will be conducted normally while the second half of the QRS will be wider and "aberrant." Ectopic beats are typically wide and abnormal from the very beginning of the QRS.

There is a caveat about the Ashman phenomenon, however: *Although a long-short R-R interval is a great "set-up" for aberrant conduction – it is also a great set-up for reentrant ectopy. Do not automatically assume that a wide QRS complex that ends a long-short R-R interval during atrial*

fibrillation is always an aberrantly-conducted supraventricular beat. If the coupling intervals are exactly the same, the abnormal-appearing beat is likely a reentrant ectopic beat.

*BTW, the rhythm in this ECG is a **wandering atrial pacemaker**.

Now for a *lagniappe* (pronounced “lan-yap,” Louisiana creole for “a little something extra”)...



Figure 4 (Limb leads from Figure 1)

You may have already noticed the baseline artifact in Leads I, II and aVR. This is NOT a tremor artifact; a tremor artifact would be present in ALL leads. If you have excellent eyesight, you may even have noticed that there is a lesser amount of the same artifact present in Leads aVL and aVF. Lead III is essentially the only frontal plane lead without the artifact.

What does this mean? Let's address the three leads with the *most* baseline artifact – Leads I, II and aVR. Before we go any further, I want to remind you of a few things about Einthoven's Triangle...

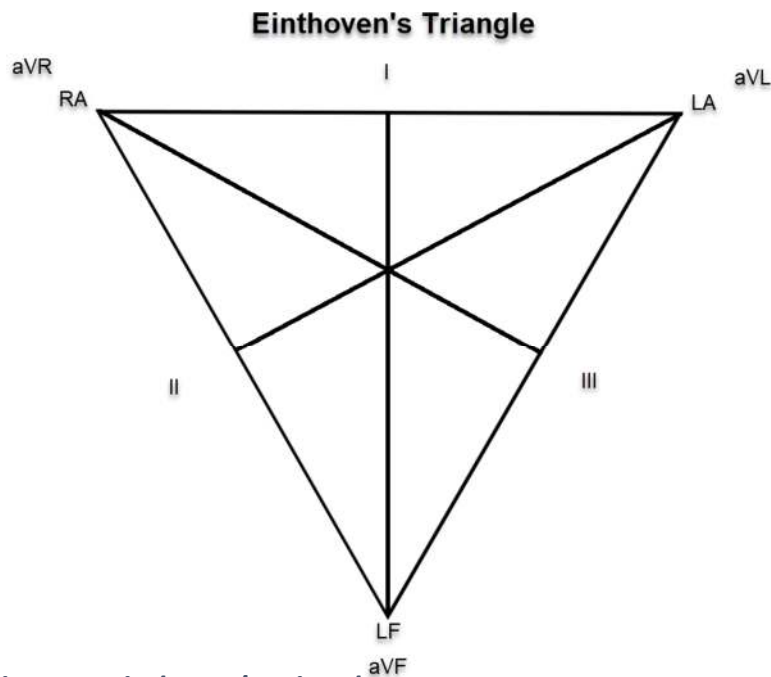


Figure 5 Einthoven's Triangle

see.

What I really want you to notice is that **Lead aVL is the left arm electrode**; **Lead aVR is the right arm electrode**; and **Lead aVF is the left foot electrode**.

Leads I, II and III are calculated by determining the difference in voltage between two of the three electrodes. For Leads I, II and III it takes TWO electrodes to determine the lead.

The AUGMENTED leads (aVR, aVL and aVF) each have a positive pole which is their **assigned electrode (RA, LA, LF)** and a virtual negative pole – which you can't

NOTE: EVERY lead on the ECG is a BIPOLAR lead. THERE IS NO SUCH THING AS A UNIPOLAR LEAD! THERE NEVER HAS BEEN ANY UNIPOLAR LEADS! Yes, I KNOW the word unipolar has been used for years... BUT IT HAS BEEN USED INCORRECTLY! Even the man who coined the term "unipolar lead" stated *unequivocally* that *these were NOT actually unipolar leads*. The latest recommendation is to NOT use "bipolar" or "unipolar" in relation to the ECG leads.

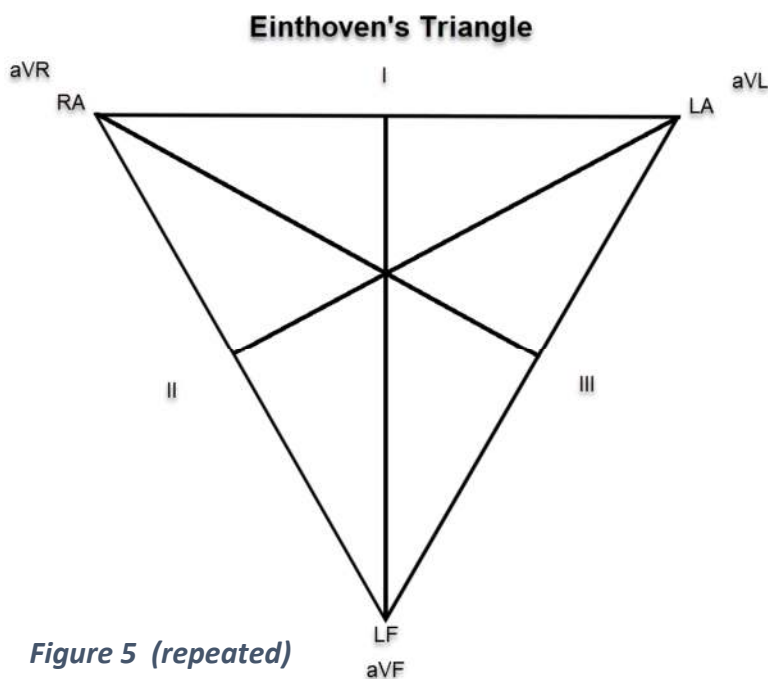


Figure 5 (repeated)

When THREE leads manifest an artifact, **the involved electrode will be the electrode associated with the affected AUGMENTED lead**. On this ECG, the artifact appears in Leads I, II and **aVR**. aVR is the affected AUGMENTED lead and it is represented by the RIGHT ARM electrode.

If the right arm electrode is the only one involved, then why are Leads I and II also affected? What do Leads I, II and aVR all have in common? **They ALL use the right arm electrode**.

Lead III was the only frontal plane lead that was NOT affected by the offending electrode because *Lead III is not calculated using the right arm electrode*.

Therefore, the culprit electrode will be the positive pole of the affected AUGMENTED lead and also the two standard leads on each side of it will be affected because that electrode is also used in their own derivations. So, in our case here, a loosely-attached RIGHT ARM electrode affects not only **Lead aVR** but also **Leads I** and **II** which are on each side of it – all three sharing the right arm electrode.

But why the lesser degree of artifact in Leads aVL and aVF? Not as bad as in Lead aVR – but still noticeable (with a bit of scrutiny). What is their connection? Remember, the AUGMENTED leads are really BIPOLAR leads and that all three of them ***share the same negative pole***: called **Wilson's Central Terminal**. There is much less effect on Leads aVL and aVF, but the effect is still discernible if you look closely enough.

So, when someone presents you with an ECG on which three of the frontal plane leads have an obvious artifact – just note the affected augmented lead and THAT will tell you which electrode is the culprit. It's almost like a parlor magic trick – your colleagues will be amazed!