Can you see the *invisible* ST elevation…?

First – a warning! There is a lot of learning to be acquired from this ECG and it is primarily at the *intermediate* to *advanced* level.

On a 12-lead ECG, an acute epicardial ischemia (aka, acute MI) can present as ST *elevation* or ST *depression*. Wait a minute – an acute MI can present as ST *depression*? How does that happen?

It happens like this (66 year old man with classic substernal chest pain)…

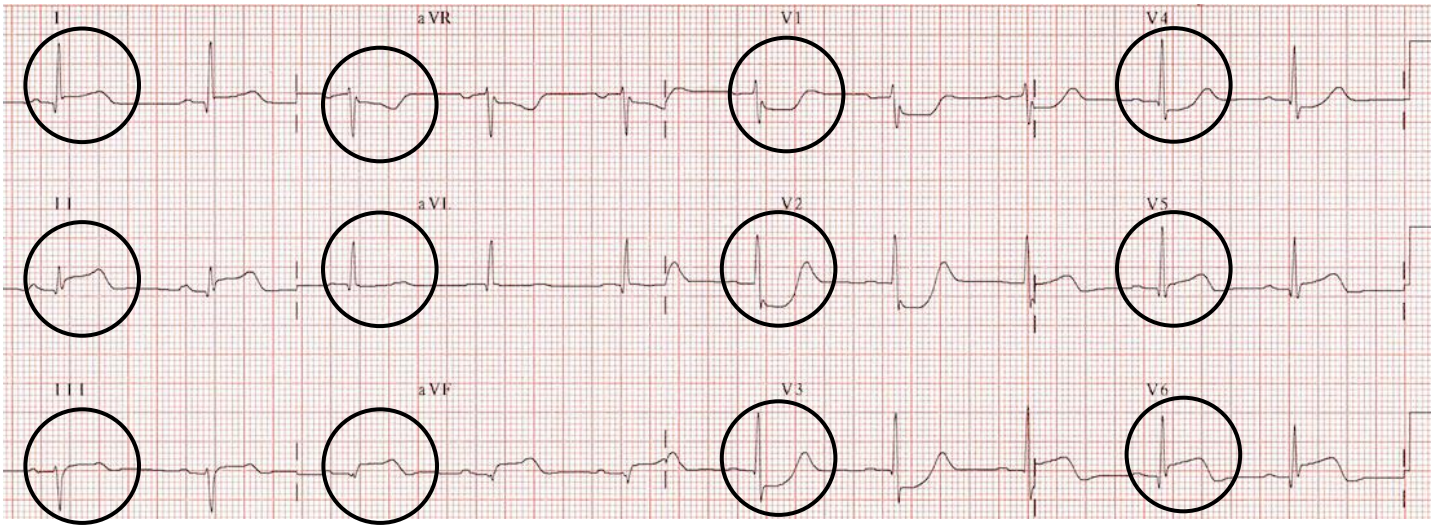


Figure 1

I have circled all the examples of ST elevation and ST depression due to an acute occlusion myocardial infarction (OMI).

"Wait a minute!" you exclaim. "That's every lead! You've circled all twelve leads!" To which I reply, "Congratulations! What an *amazing* observation!"

The ST deviation in every lead that I circled is due to a *single* coronary artery occlusion. The occlusion is in a *dominant left circumflex artery proximal to the obtuse marginal branch*.

The ST elevations represent the site of actual acute epicardial ischemia and the ST depressions represents the *specific* reciprocal changes to that epicardial ischemia.

This *one occlusion* has resulted in an *inferior* MI (II, III, aVF), a *basolateral* MI (I, aVL), a *posterior* MI (V1-V4) and an *apicolateral* MI (V5, V6). The ST depression in Lead aVR is due to *reciprocity* with both the basolateral and inferior leads since it is 150° away from the positive poles of both Lead I and Lead II.

Let's start with the inferior MI. There is ST elevation in Leads II, III and aVF which basically defines this as an inferior MI. We're probably all in agreement with that.

Next, let's look at the basolateral MI. There is ST elevation in Leads I and aVL.

Once again you exclaim, "Wait a minute! There's no ST elevation in Lead aVL!" to which I reply, "Oh, YES there *is*… you just aren't experienced enough to see it yet. But I assure you it's there! And you WILL be able to see it momentarily!"

Now, let's look at the posterior MI. It has become evident during the last few years that what we have always called "posterior" MIs are actually infarctions of the left *lateral* ventricular wall. Because of the heart's *rotated* position in the chest, what we have always viewed as the *lateral* wall in textbooks and journal articles is actually located in the *posterior* thorax. The correct term for a posterior MI is *lateral MI*. That has created a tremendous amount of confusion; therefore, I will compromise (for the time being) and call it a *posterolateral* MI.

A posterolateral MI does not appear directly on the 12 lead ECG. But because it is located in the horizontal plane, its reciprocal leads (V1 – V3 and sometimes even V4) do appear. So, this is an infarction that we can diagnose strictly from its reciprocal changes. (Haven't I always reminded you that reciprocal changes are very important in corroborating the presence of an acute MI?)

So that leaves us with the apicolateral infarction diagnosed by the ST elevation in Leads V5 and V6. There is a lot to say about the ST elevation in Leads V5 and V6…

> First, ST elevation in V5 and V6 can be caused by *all three coronary arteries*. In fact, all three coronary arteries (LAD, LCx and RCA) can cause an *inferior MI* along with *ST elevation in V5 and V6* (YES, even the left anterior descending artery can do that!). This particular occlusion occurred in the LCx which is slightly more likely to result in ST elevation in Leads V5 and V6 than occlusion of the RCA, but the percentage difference isn't great enough to use that fact diagnostically.
>
> Second, the apicolateral area of the left ventricle is probably *one of the best perfused areas of the heart*. It receives blood from the RCA, the LAD, the LCx and the ramus intermedius (when present).
>
> Here's the problem you must consider: if there is ST elevation in V5 and V6, *why* did that happen? With so many vessels capable of perfusing the area, why did an occlusion of just one of those vessels result in ST elevation (transmural myocardial ischemia)? Could it be because all the other vessels are *too diseased to be of any assistance*? Or could it be due to the fact that the LAD may be a short Type 1 LAD; the RCA is not dominant, so there are no posterolateral arteries coming from the RCA; and finally, the ramus intermedius may simply be absent (it's present in only about 30% of people).

As you can see, there's a lot more to *interpreting* an ECG than just *reading* it!

Let's return to the issue of ST elevation in Lead aVL that many of you were probably not able to see. How do I know that there really is ST elevation there? First of all, because there is ST

elevation in Lead I and that happens when there is a basolateral MI – which is also included in the territory for Lead aVL. Lead aVL *must* be seeing this same epicardial ischemia, but why isn't it *showing* it? For the answer to that question, let's turn back to the inferior infarction and specifically, the ST elevation in Lead III.

Did you notice that, while there *is* visible ST elevation in Lead III, it isn't of the same magnitude as in Leads II and aVF? Did you remember the *reciprocity* that exists between the *inferior* leads (II, III, aVF) and the *basolateral* leads (I, aVL)? And especially between Leads III and aVL? A perfect display of simultaneous infarctions of the *inferior wall* and the *basolateral wall* would manifest ST elevation in Leads II, III, aVF, I and aVL.

Well, THAT isn't going to happen because Leads III and aVL are as far apart as two reciprocal leads can be in the frontal plane (150°). ST elevation in Lead aVL is going to cancel with the ST elevation in Lead III. If BOTH Leads had the *same amount of ST elevation*, they would cancel each other perfectly and *both* leads would appear normal, *despite the fact that both are infarcting*.

But what if one of the leads has *more* ST elevation than the other? Cancellation of forces could cause one ST segment to drop down to a level that is isoelectric with the baseline and the other might retain a small amount of ST elevation. Take another look at the ECG and see if you notice something like this occurring between Leads III and aVL.

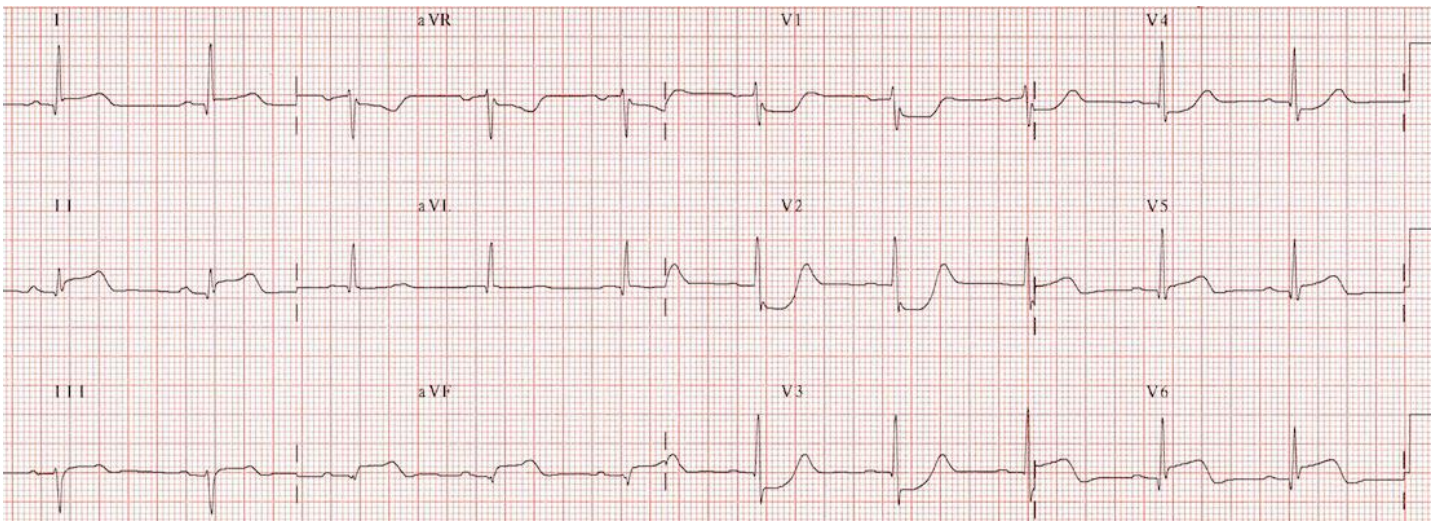


Figure 2

There it is! Now look once more at the inferior leads and the basolateral leads and then think about the reciprocity that exists between those sets of leads and especially between Lead III and Lead aVL. Are you starting to see the "invisible ST elevation" in Lead aVL now?

But wait! There's more…!

Let's take a closer look at Leads V1 – V3. As a matter of fact, let's focus on Lead V2 because it has a great QRS-T to analyze.

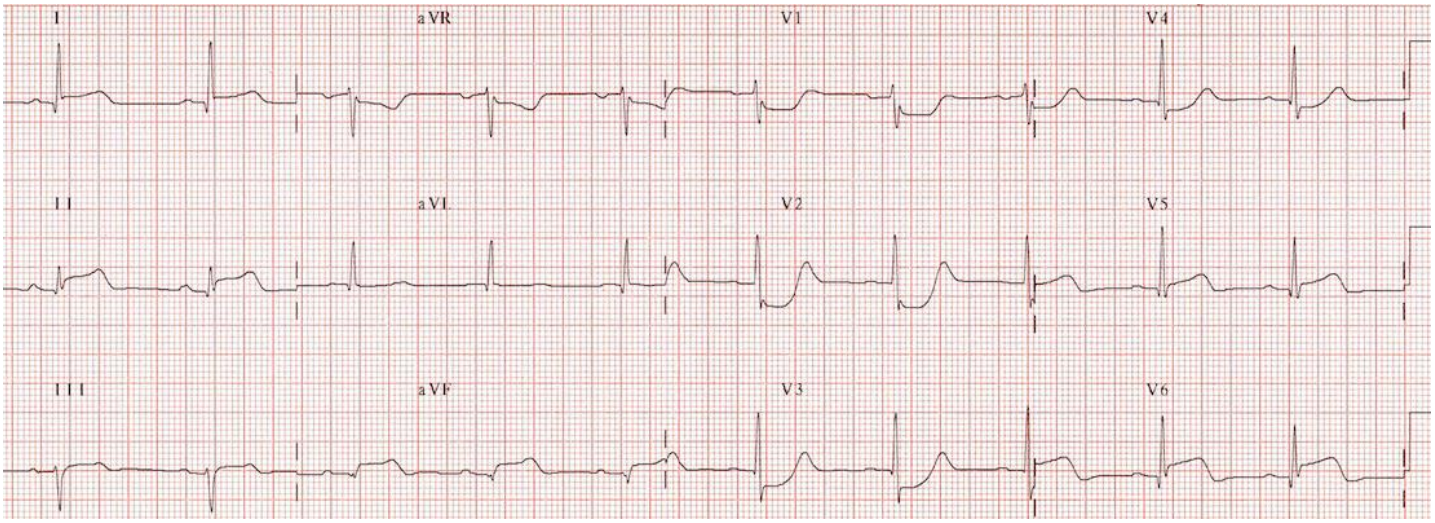
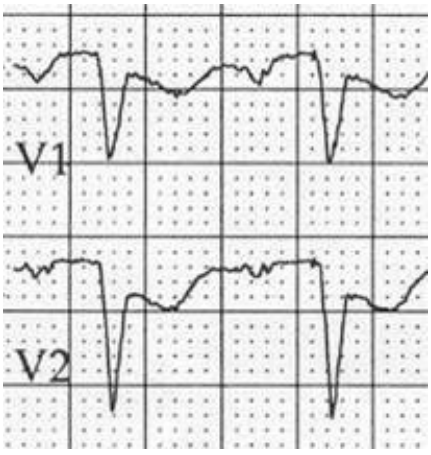


Figure 3

One thing that you must understand if you ever deal with myocardial infarctions is that **T waves do NOT invert – or even *begin* to invert – while the acute ischemia is on-going**. T waves begin to invert only when the imbalance of the supply-demand for oxygenated blood has resolved. Either reperfusion has occurred (spontaneously or by medical means) or part of the heart muscle has died. The human way of resolving this imbalance is by restoring the circulation (supply) to the ischemic area; nature's way of resolving the imbalance is by killing off the heart muscle (demand). Restoring Supply vs. Reducing Demand.

So, remembering that T wave inversion occurs only after the infarct is resolved (one way or the other), take a close look at Lead V2 (Figure 3). Do you see the T wave inversion? If you don't, it's because you have forgotten that you are really seeing a *reciprocal* lead. To see what a posterior lead *should* look like, we have to turn this QRS-T upside down. So, instead of seeing a tall R wave, a deeply depressed ST segment and an upright T wave, what we are *actually* seeing is a deep Q wave, marked ST elevation and an *inverted* T wave. That suggests that reperfusion has already begun posteriorly *but not inferiorly*.

Here is a snippet of Leads V1 and V2 from another patient during an acute posterolateral MI…



As you can see, no R wave means "no Q wave yet." ST depression is present meaning that "ST elevation" is present posteriorly. And the T wave is inverted, meaning the T wave posteriorly is still "upright," a much better situation in which to intervene.

Many ECG experts rely on the analysis I just demonstrated to develop an idea of the status of the posterior MI. I myself did exactly that for years.

Figure 4

But eventually, I began to notice a problem. It became especially evident when I began examining ECGs of patients with posterolateral MIs in which posterior leads were recorded immediately after the 12-lead ECG (which usually isn't done). Here is the SAME ECG as in Figures 1 to 3 with posterior leads (V7, V8 and V9) recorded in place of V4-V6…

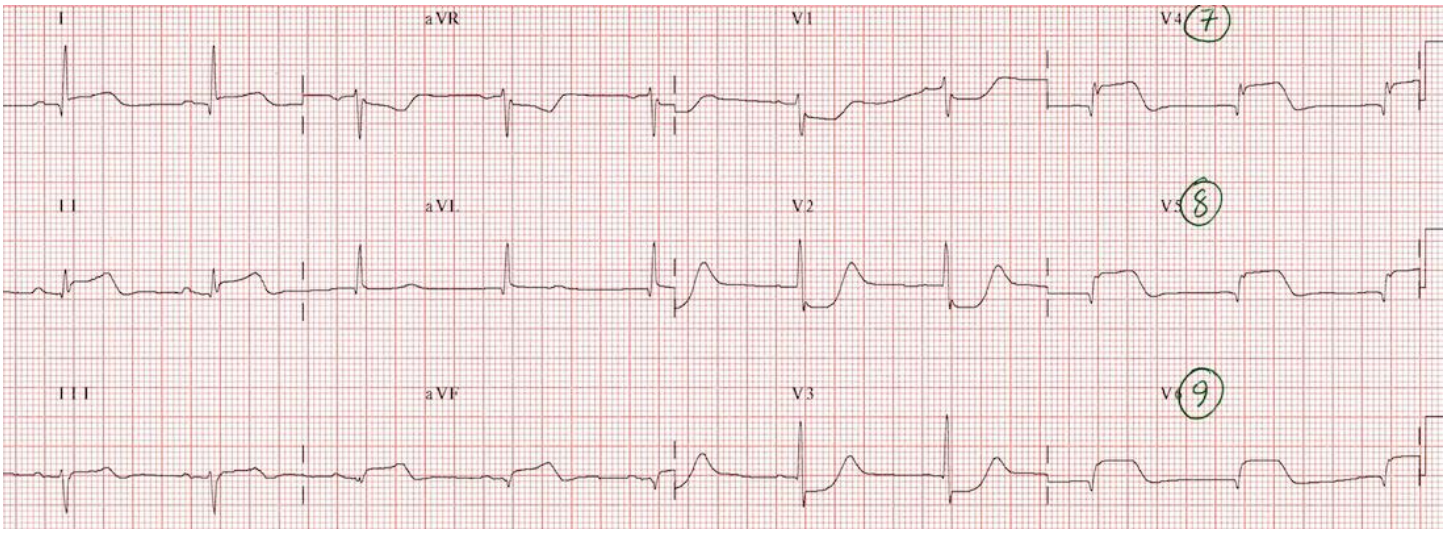


Figure 5

Do you notice anything? What I began to notice was that often the posterior leads done at almost exactly the same time *failed to show reperfusion changes even though the reciprocal leads (V1-V3) did!* Are the directly recorded posterior leads different because of the increased distance to the surface of the heart? Or is there simply no reperfusion actually taking place?

I don't have the answer since I am unable to do a controlled study and also because I have not found any articles regarding this issue specifically. If any of you know of any studies or articles addressing this topic, please let me know.

My approach (in my latter years as an ER attending) was that when V1 – V3 manifested T wave inversion, I assumed that no reperfusion had begun. If V1 – V3 manifested an upright T wave (along with ST depression), I recorded posterior leads. It is acceptable to use V1 – V3 (and sometimes V4 is affected, also) to make a diagnosis of a posterolateral MI *without recording posterior leads*.

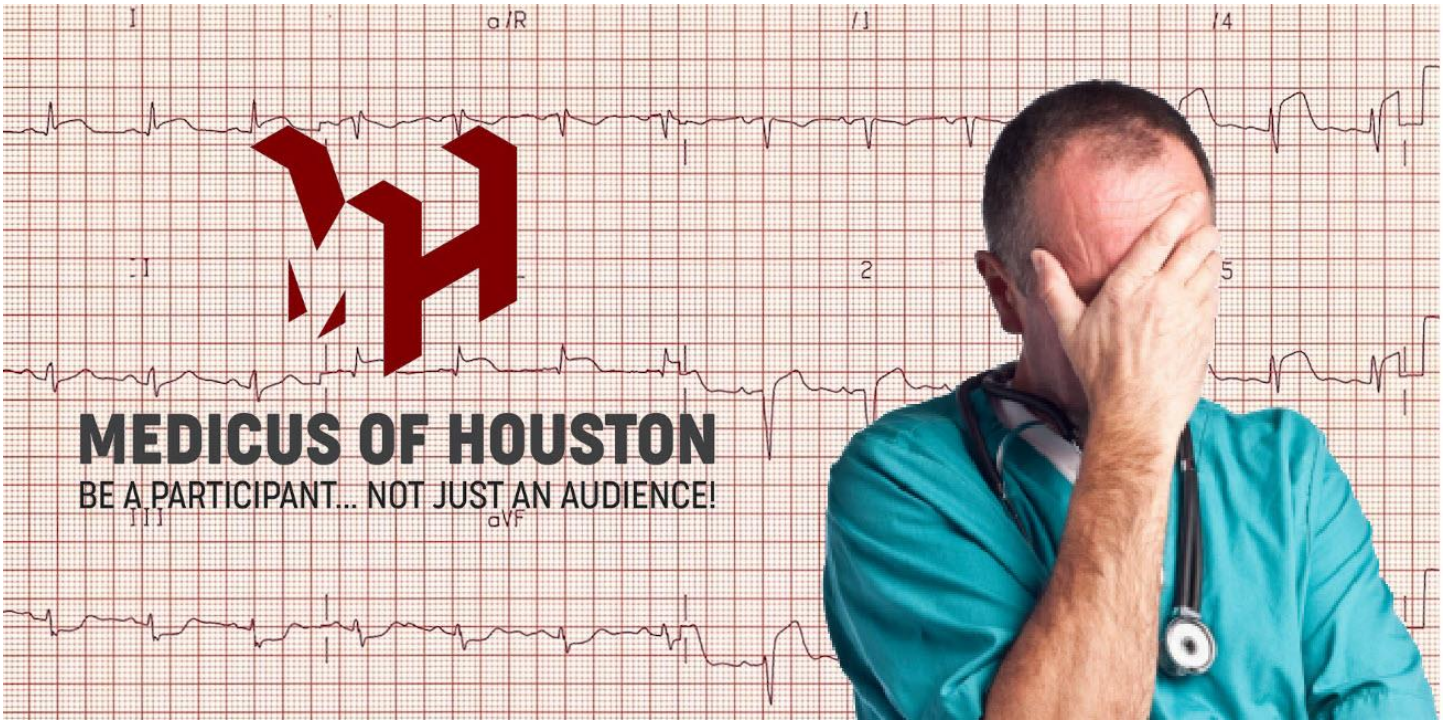
There were basically two instances when I recorded posterior leads: 1) when there was ST depression with an upright T wave in V1 – V3, and 2) when I suspected an acute coronary syndrome but saw no ST changes on the 12-lead ECG. Remember…

You cannot rule out an acute myocardial infarction based on just one ECG, just 12 leads or just one troponin (from blood drawn less than 4 hours after the onset of symptoms).

Is it really necessary to know this information (upright vs. inverted T wave in V1 – V3) in order to properly manage the patient? No, but I always wanted to be on top of things like this because there were sometimes long delays in transferring a patient into the chest pain unit or any of the critical care units (ICA, CCU) during which time the patient was my responsibility.

Are you also aware of the specific dysrhythmic and hemodynamic complications that may manifest in *each* of the coronary arteries when occluded? Even different *locations* of the occlusion within a single coronary artery can result in a different set of complications. As the old adage says, "An ounce of prevention is worth a pound of cure." Perhaps I will create a post on that topic in the future.

Your management of this patient will not be any different. And remember: you only need 0.5 mm of ST elevation in the posterior leads to qualify as acute ST elevation. As you can see, this unfortunate patient is in a lot of trouble!



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