Why Does Subendocardial Ischemia NOT Localize on the ECG?

Jerry W. Jones, MD FACEP FAAEM

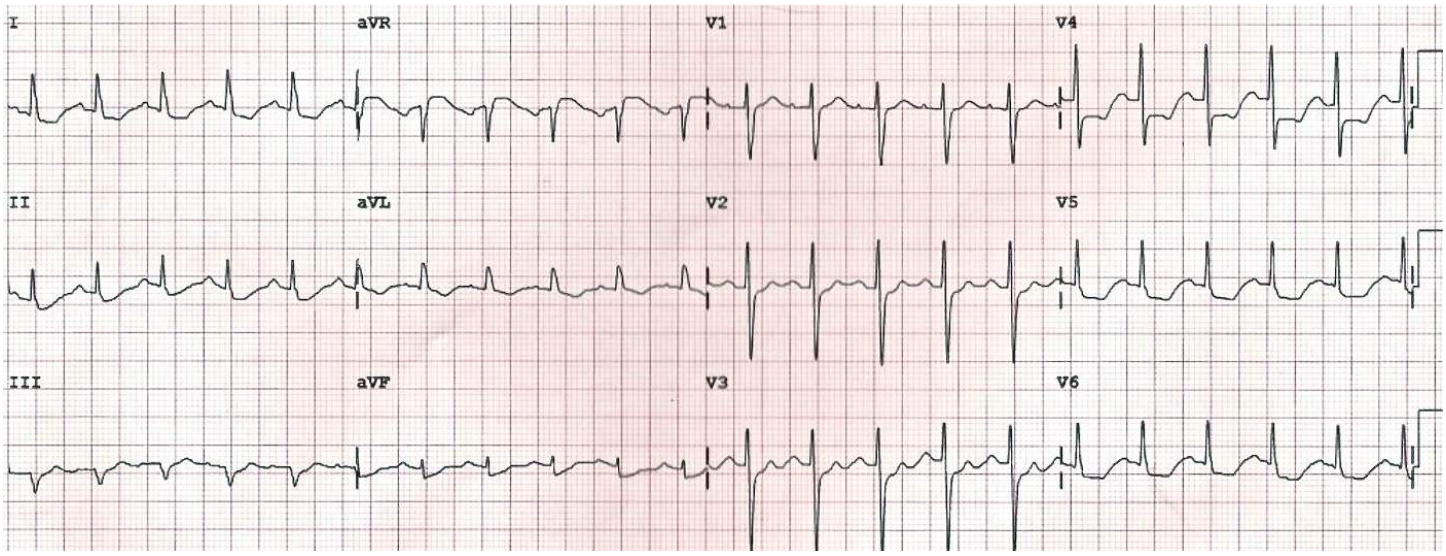


Figure 1 | Subendocardial Ischemia

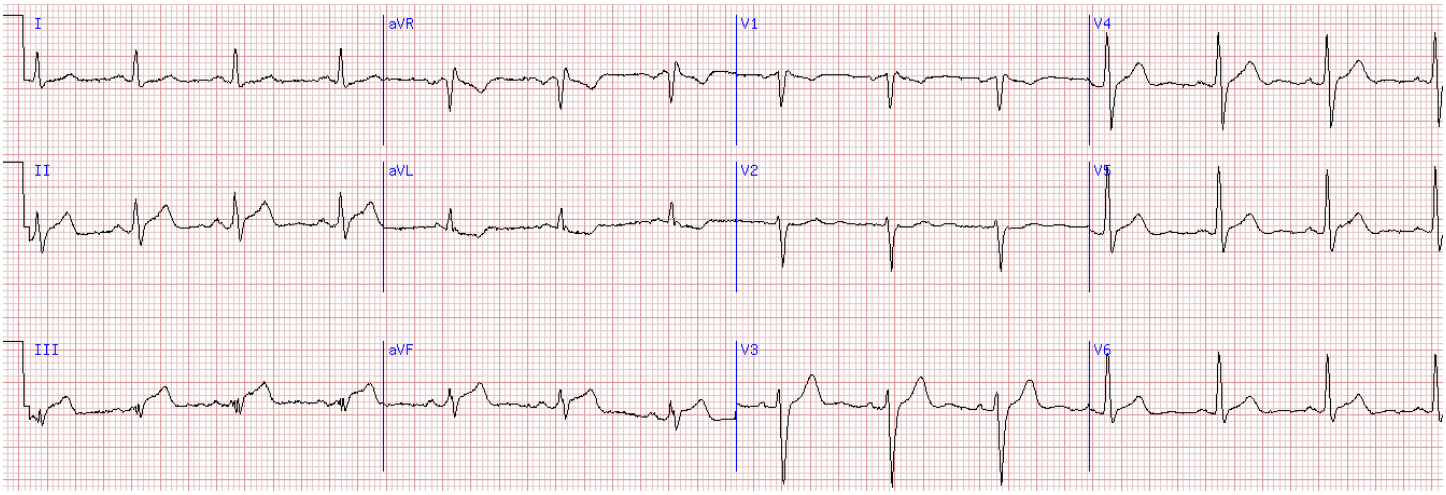


Figure 2 | Reciprocal Changes

One of the most frequent – and consequential – mistakes made by beginners in electrocardiography is the confusion caused by ST depression when present on the ECG of a patient with chest pain. Too often medical personnel are led to believe that ST depression represents subendocardial ischemia (which, for some unknown reason, is often assumed to be chronic) and that ST elevation represents an acute MI.

Let me begin by disabusing you of some bad information:

1. **Subendocardial ischemia may be *acute* or it may be *chronic*.** You should NEVER assume that it is chronic without previous ECGs with which to compare. Here's a major problem to consider: *acute* subendocardial ischemia and *chronic* subendocardial ischemia will likely *look exactly the same!* And the subendocardium can proceed to *infarction* (non-transmural) and STILL look like subendocardial *ischemia*. Never allow yourself to become overconfident while managing a patient with chest pain and ST depression.

2. **Subendocardial ischemia may be represented by ST depression or ST elevation.** It is not the *type* of ischemia that determines whether the ST segment is elevated or depressed – it is the *location* of the recording electrode and the *direction* of the injury current.

3. **ST elevation does NOT indicate an acute myocardial infarction.** It represents acute transmural ischemia. An infarction is indicated by – among other things – Q waves, but *not* ST elevation.

4. **The ST depression of subendocardial ischemia does NOT tell you where the ischemia is located specifically.** It just tells you that it is *subendocardial*.

5. **The ST depression of a reciprocal change is very good at localizing an acute transmural ischemia.** It may do so BEFORE there is any ST elevation detected.

OK… so why does the ST depression of subendocardial ischemia not localize to a specific vascular territory while the ST depression of a reciprocal change does localize with very good reliability? Let's look at a section of the left ventricular wall during an acute subendocardial ischemia…

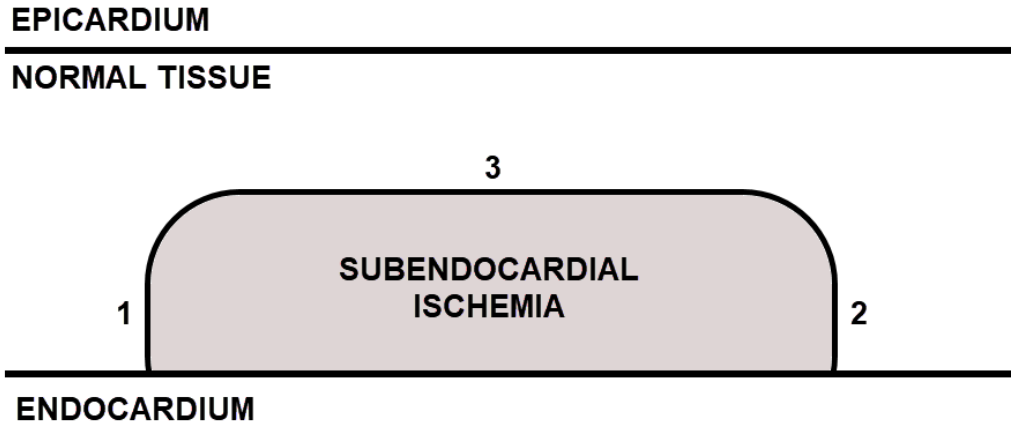


Figure 3 | Diagram of Left Ventricular Wall

As you should know, *ALL ischemia begins in the subendocardium – not* in the epicardial area. In Figure 3, there is a diagram of acute ischemia (shaded area) developing in the subendocardium. It is surrounded by normal, non-ischemic myocarddium. As you can see, the subendocardial ischemia has not yet become transmural, i.e., it has not risen to the epicardial surface.

This is the actual approximate shape of most developing subendocardial ischemia. There are three interfaces between the ischemic myocardium and the non-ischemic (normal) myocardium. The left (1) and right (2) borders are perpendicular to the endocardial and epicardial surfaces while there is a longer, transverse border (3) that is parallel to the epicardial and endocardial surfaces.

As I said earlier, whether ischemia presents as ST depression or ST elevation is not based on two different kinds of ischemia – it's ALL ischemia – it depends on the location of the recording electrode and the direction of the injury current.

Here are two very basic rules to remember for this concept to become understandable:

1. Injury currents – during Phase 2 of the action potential (which corresponds to the ST segment on the ECG) – always travel from NORMAL tissue to ISCHEMIC tissue,
2. A positive current traveling TOWARD a recording electrode will result in ST elevation on the ECG while a positive current traveling AWAY from a recording electrode will present as ST depression on the ECG.

In electrocardiography, there are only three currents we need be concerned with:

1. The current of depolarization, which has a POSITIVE head and a NEGATIVE tail
2. The current of repolarization, which has a NEGATIVE head and a POSITIVE tail
3. An injury current, which – like the depolarization current – has a POSITIVE head and a NEGATIVE tail.

Now let's take another look at out subendocardial ischemia diagram and see what the different currents reveal. We will be dealing ONLY with the injury currents initiated at the ischemic/normal interfaces since there is nearly no net depolarization or repolarization current during the Phase 2 "plateau" which is represented on the ECG by the ST segment.

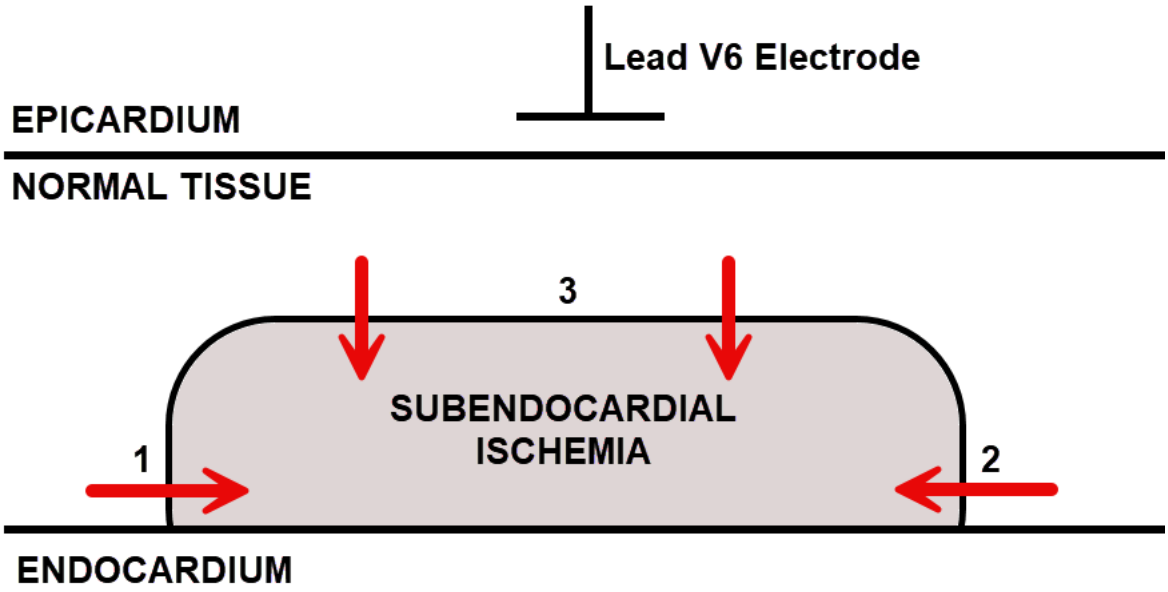


Figure 4 | Injury Currents + Recording Electrode (V6)

Remember: injury current vectors have POSITIVE heads and NEGATIVE tails. If an electrode sees a POSITIVE head coming TOWARD it, it will record a POSITIVE deflection (in the case of the ST segment, that would ST elevation); if it sees a NEGATIVE tail, it will record a negative deflection (and in this case, ST depression).

Look at Figure 4 and tell me what the V6 recording electrode on the left lateral chest wall sees. It sees the injury current vectors traveling AWAY from it; therefore, it sees only the negative tails. Thus, it records ST depression.

But what about the two lateral interfaces (1 and 2)? There are two reasons those interfaces – which are very important because that is where the most voltage change occurs – do not leave much, if any, impression on the ECG tracing:

First, both currents are essentially *traveling perpendicular* to the V6 electrode. Currents traveling perpendicular to an electrode are essentially invisible to that electrode. So, we wouldn't expect much of an impression on V6. But there is another issue…

Second, those injury currents are *traveling directly toward each other*. They are going to collide head-on and extinguish each other.

> **PEARL |** When two vectors travel in opposite directions *or* travel directly toward each other, they are going to be undetectable to a lead's recording electrode.

So, what are we left with after considering all the injury vectors present here? The only vector leaving an impression on the V6 recording electrode is the vector traveling from the normal myocardium directly away from the V6 electrode through the transverse border and toward the subendocardial ischemia. *It is pointing toward the subendocardium of the left ventricle and the ventricular cavity itself*.

And what information can we extract from that? Can we determine which area of the left ventricle is involved? NO! Whether the subendocardial ischemia is inferior, inferolateral, anterior (LAD) or anterior (LCx), all indicators would simply point toward the NON-SPECIFIED subendocardium. It CANNOT localize to the specific vascular region *when viewed from electrodes placed on the exterior chest wall*.

But what if we placed electrodes on the endocardial surface at different locations WITHIN the left ventricle? Now that is a completely different matter with a very different response!

First of all, we would again dispense with the lateral ischemic vectors because any electrodes placed internally would still have the same relationship to those vectors: they would be traveling perpendicular to the electrodes and be practically undetectable. But the injury current vector travinging across the transverse border (or interface) would be traveling directly TOWARD at least one of those internal endocardial electrodes. And, because the vector has a POSITIVE head and a NEGATIVE tail, the electrode would see a POSITIVE charge coming toward it and it would inscribe ST elevation that would be specific to its location on the endocardium.

In THAT situation, subendocardial ischemia *could* localize – but don't expect this! How often do you place electrodes directly on the endocardium just to do an ECG?

"But Dr. Jones," you say. "In your books and your Masterclasses, you make the point that Lead aVR acts as a 'virtual' intracavitary electrode! It usually demonstrates ST elevation during subendocardial ischemia. Doesn't that localize the subendocardial ischemia?"

It localizes the ischemia to the subendocardium but no further. In my example above, please recall that I referred to placing electrodes *directly on the endocardial surface* which allowed INTERNAL localization of the subendocardial ischemia. Now, where is the electrode for Lead aVR located… on the RIGHT SHOULDER! All it sees is a summed vector telling it that there is a *mean* injury vector pointing inward. It cannot localize the vector to a specific vascular territory.

That is why the ST depression of subendocardial ischemia as detected by electrodes on the chest wall cannot localize the ischemia to a specific vascular area.

But why are *reciprocal changes* able to localize?

Let's be a bit more specific about reciprocal changes: *they localize to a vascular area that is **opposite** to the leads in which they manifest*.

You are not seeing a local injury vector when you look at a reciprocal change. You are seeing a localizing injury vector in a vascular area *opposite to the lead with the ST depression* of the reciprocal change. This is because 1) the electrodes for the leads manifesting the reciprocal change are overlying normal, non-ischemic myocardium and 2) this is occurring during Phase 2 of the action potential (ST segment) which is normally quiet electrically, allowing the electrode to see the vector occurring across from it. So, reciprocal change is essentially a mirror image of a localizing transmural (or almost transmural) ischemia occurring opposite it.

I hope you have a better understanding of WHY you cannot tell anything more from the ST depression of subendocardial ischemia than the fact that the ischemia is subendocardial. So when you see "inferior subendocardial ischemia" or "anterior subendocardial ischemia" on an ECG report – know better than to belioeve it!