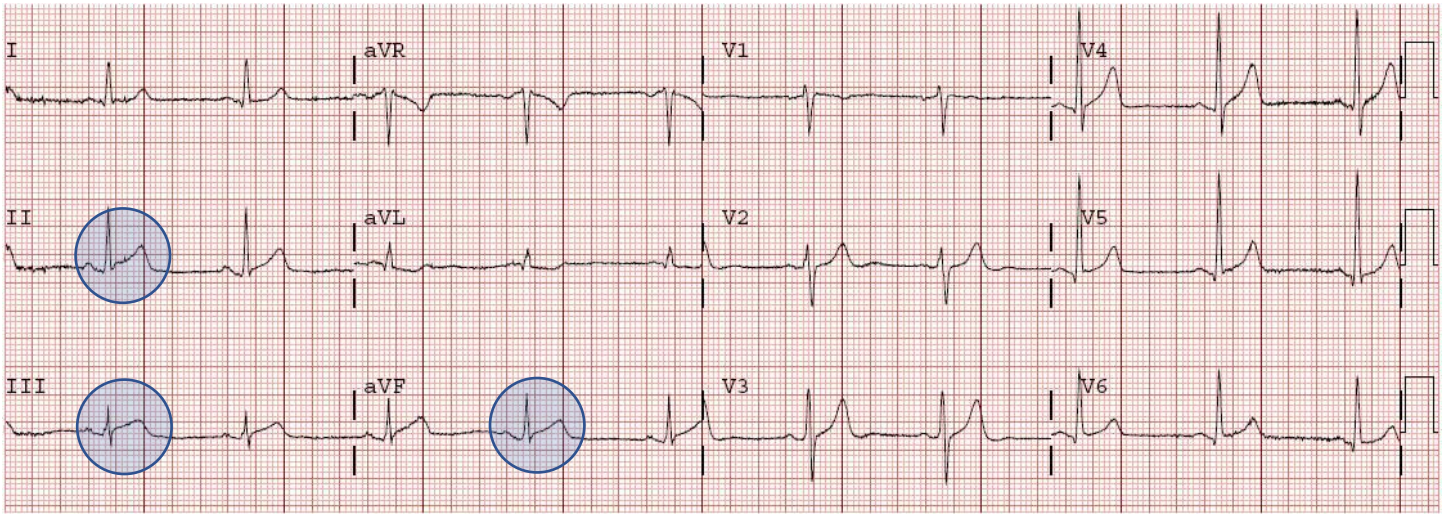
How Do We Diagnose an Acute Inferior MI?

A 56 year old male with a history of hypertension, 2 pack/day smoking habit and hypercholesterolemia presents in your emergency facility. He complains of the sudden onset of squeezing pain in the center of his chest with radiation to both shoulders. He also feels very nauseous and is visibly diaphoretic.

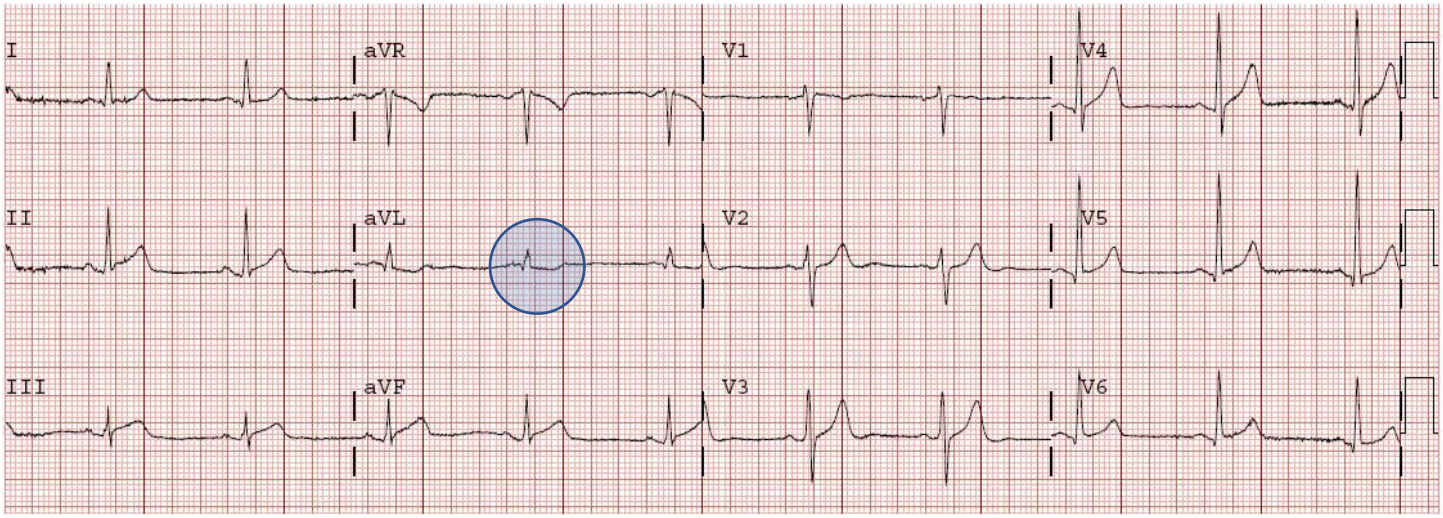
SPOILER ALERT: This man is having ***an acute inferior MI*** (no, I'm not trying to mislead you!).

My question is: how do we diagnose an *acute* inferior MI (or, more exactly, an *acute* inferior epicardial ischemia)? There are three strong hints.

First, we look for ***ST elevation of 1 mm or more*** in the inferior leads – Leads II, III and aVF. In this man's ECG, we see a subtle elevation in those leads – most notably at the J-point (the point at which the QRS interval ends and the ST segment begins). This elevation of the J-point appears to be 1 mm, so this alone would allow us to make the diagnosis of an acute inferior MI. But those elevations really are somewhat subtle and could easily be missed by someone with little experience interpreting ECGs.



Second, we would like some supporting evidence of an acute inferior MI. The first thing I would look for is a ***reciprocal change*** to the ST elevation in Leads II, III and aVF. But where would I look?



Look for reciprocal changes to ST elevation in II, III and aVF in Leads I and aVL. Actually, we can simplify this by just looking at Lead aVL – for now, don't concern yourself too much with Lead I. The reason for this is that we want reciprocal leads to be *as close to 180° apart as possible* for maximum benefit. So… what do we see? We see a subtle – but definite – downsloping ST depression in Lead aVL. ***There is our reciprocal change!***

Now a lot of very good physicians think that Leads II, III and aVF are reciprocal to Leads V1, V2 and V3. But they are wrong for at least two reasons:

> ***First***, *all leads that are reciprocal to each other must be in the same plane!* Leads II, III and aVF are in the *frontal* plane and Leads V1-3 are in the *horizontal* plane. Those two planes are perpendicular which makes them essentially invisible to each other.
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> ***Second***, the *inferior* leads should be reciprocal to *superior* leads while the *anterior* leads should be reciprocal to *posterior* leads. You've probably never heard of the "superior" leads because there is actually no such term as "superior leads." But there ***IS*** one lead that is definitely located superior to Leads II, III and aVF, and that is **Lead aVL**. And… aVL just happens to be in the *same (frontal) plane* as the inferior leads.
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> The positive electrode for Lead aVL is located on the left shoulder, so it has a very good view of the basolateral area of the left ventricle (we used to call that the "high lateral" area). The positive pole for Lead aVL is 150° away from the positive pole of Lead III and that's as far away as two positive poles can be in the frontal plane.
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> "But Dr. Jones," you say, "I've *seen* ST elevation in the inferior leads along with ST depression in Leads V1-3. Doesn't that prove that those groups are reciprocal to each other?" The ST depression in Leads V1-3 are definitely reciprocal changes – but they are *reciprocal to the ST elevation in Leads V7-9* which are usually not recorded on most ECGs. And, if you would look at Lead aVL, you will likely see ST depression there also which is the *real* reciprocal change to the ST elevation in Leads II, III and aVF.
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> Now someone else says "But Dr. Jones, I've seen **ST elevation in Leads V1 – V4** with **ST depression in the inferior leads**. Surely that proves that the inferior leads are really reciprocal to Leads V1-3!" Anytime there is an occlusion proximal to the *first diagonal branch of the left anterior descending artery*, you will get ST elevation in V2 downward and, if the occlusion also happens to be proximal to the *first septal perforator branch* also, there will be ST elevation in Lead V1 as well. But remember: the first diagonal branch *also* perfuses the basolateral wall of the left ventricle. The left shoulder electrode, which is the

positive pole for aVL, is looking directly at that area. So, when you get ST elevation in V1 – V3 (and sometimes further), you will typically *also* get ST elevation in aVL. So that "reciprocal ST depression" in the inferior leads is *actually* reciprocal… but it's reciprocal to the ST elevation in aVL and not directly reciprocal to the ST elevation in Leads V1-3. But what if I don't see any ST elevation in aVL? The answer to that question is in the next sentence…

Always remember that the reciprocal change can occasionally appear on the 12-lead ECG ***BEFORE*** *the ST elevation!* So, if our 56 year old man only had an isolated ST depression in Lead aVL, given his presentation, that would make me very concerned that an acute occlusion of the right coronary artery (RCA) was in the process of occurring. Now, just because *I* might be convinced does not mean that a *cardiologist* is going to be convinced. This is a case where you should begin your workup and immediate treatment and repeat the ECG about every 15 – 20 minutes. For more information on how we are trying to move away from diagnoses based on "millimeters of ST elevation" to diagnoses based on a more critical inspection of the ECG, do a search for "Occlusion Myocardial Infarction" or "OMI." You can also download a great monograph on this topic from ***Dr. Smith's Blog*** (https://hqmeded-ecg.blogspot.com/).

So we have determined that ST elevation in Leads II, III and aVF and/or ST depression in Lead aVL – in the appropriate clinical setting – may indicate an acute inferior MI. So, what's the third big hint?

Third – look at the T waves in the inferior leads. They are very, very large for the QRS complexes… not especially tall – but very wide, best seen in Lead III. Those are ***hyperacute T waves***. Hyperacute T waves occur in the very beginning of an acute MI. They are caused by subendocardial ischemia that is beginning to extend toward the epicardium but hasn't reached it yet. The ischemia has not yet become *transmural* – and what better time to try to stop an infarction!

So, just be aware that an acute inferior MI may present as ***hyperacute T waves***, ***ST elevation with reciprocal changes*** – or, just a ***reciprocal change (with the STE occurring later)***.

Some of the changes on this ECG may look very subtle to you if you are just beginning your study of electrocardiography. But they do *not* look subtle to me at all. Study the changes until you would recognize them in an instant and always remember **Jones's Rule**:

Jones's Rule

Any ST depression on the ECG of a patient with symptoms that are compatible with an acute coronary syndrome represent a reciprocal change to an ST elevation somewhere until proved otherwise!