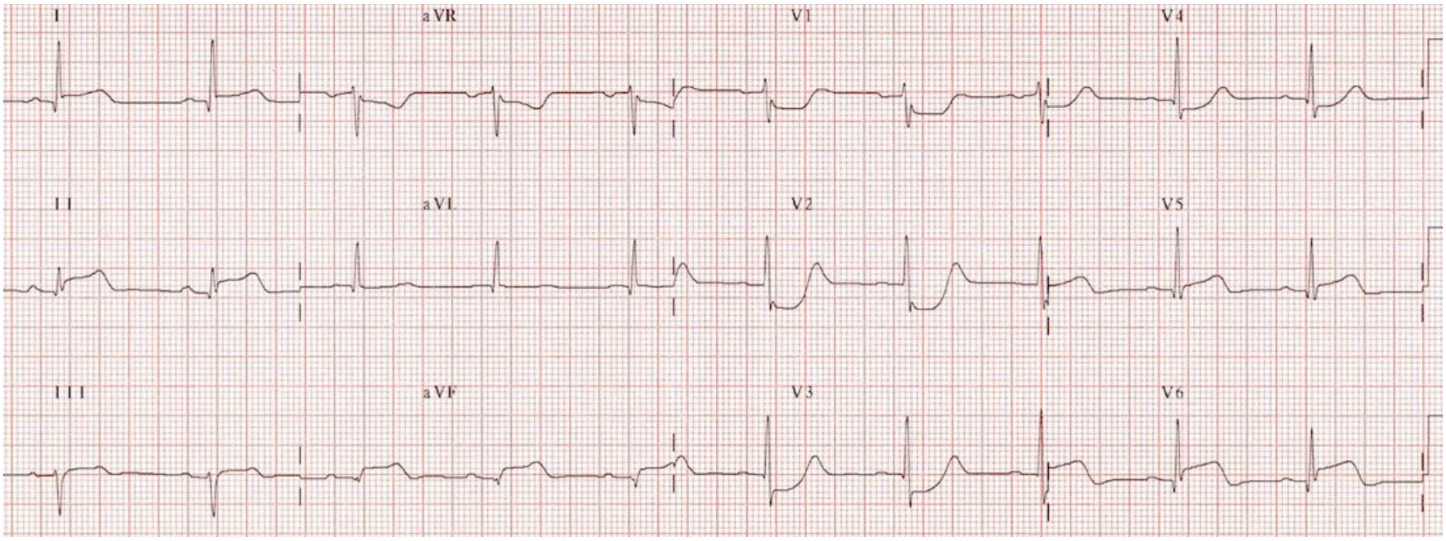
Reading an ECG vs. Interpreting an ECG

All introductory courses – written, live or on-line – teach you to READ an ECG. There's certainly nothing wrong with that! It's how we all got started, including myself. In fact, that's the way it HAS to be. You can't write a Pulitzer prize-winning novel until you've learned the alphabet! Obviously, you must be able to *read* an ECG in order to *interpret* it - but ECG interpretation takes you so much farther.

The problem with just READING an ECG is that it really doesn't give you all the information that is present… including a lot of life-saving information.

I'm going to "read" this ECG for you and then I will "interpret" it. Let's see what a difference it can make!



Reading a 12-Lead ECG

Rhythm: Sinus, regular, no obvious conduction abnormalities
P Wave: Upright in Leads I and II
PR Intervals: Normal
QRS Intervals: Narrow, less than 0.10 sec
QT Intervals: Grossly normal
ST Segments: STE in Leads I, II, III, aVF, V5 and V6; STD in Leads aVR, V1 – V4
T Waves: Low amplitude with wide bases (some begin at the "J" point)
Impression: Acute inferior MI, acute posterior MI, acute lateral MI

That's it. Does that tell you everything you might want (or need) to know about this patient and his/her ECG?

You think so? Very well. Let me ask a few questions based on this ECG…

1. What has happened here? Is there more than one artery involved?
2. If only one artery is involved, which one could produce all these findings?
3. Why does Lead I have STE while aVL has none?
4. Why do Leads II and aVF have such obvious STE while it is very subtle in Lead III?
5. What do Leads V5 and V6 have to do with an inferior MI?
6. How am I so sure that the ST depression in Leads V1 – V4 represents a *posterior MI* and not *anteroseptal subendocardial ischemia*?
7. Why can I not see any good P waves in Lead aVL?
8. Should I anticipate some additional adverse cardiac event?
9. Are you certain those are the only areas experiencing acute epicardial ischemia?

Still think just "reading" an ECG is all you need to know? Let's interpret the same ECG now.

Interpreting a 12-Lead ECG

Rhythm: Sinus, regular, no obvious conduction abnormalities
P Wave: Mean P axis is +60° making it isoelectric in aVL
PR Intervals: Normal
QRS Intervals: Narrow, less than 0.10 sec
QT Intervals: Grossly normal
ST Segments: STE in Leads I, II, III, aVF, V5 and V6; STD in Leads aVR, V1 – V4
T Waves: Low amplitude with wide bases (some begin at the "J" point)

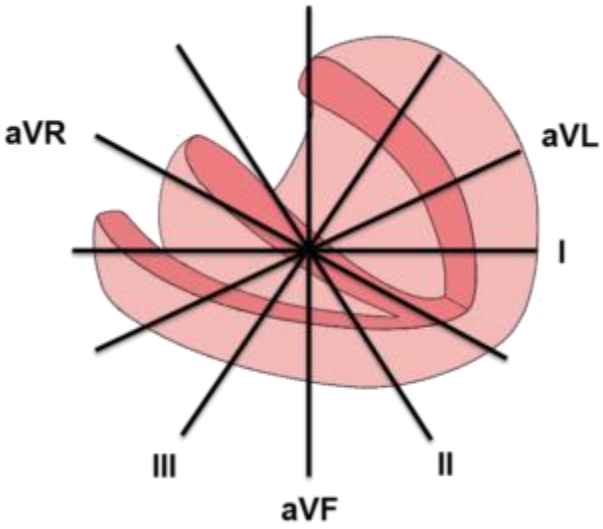
Interpretation:
There is evidence of acute epicardial ischemia in the inferior, posterolateral and lower lateral ("apicolateral") wall of the left ventricle. All three areas of the acute epicardial ischemia can be explained by a single, proximal occlusion of a dominant left circumflex artery. This is supported by the fact that the STE in Lead II > STE in Lead III. The ST depression in V1 - V4 is typical for a *reciprocal change* to a posterolateral wall epicardial ischemia. Had posterior electrodes been placed, V7 - V9 would likely demonstrate STE. If anterior ischemia were presenting so proximally (V1), it would likely present in Leads I and aVL as well, but it does not. Also, there would likely be ST depression in other leads also (and there is not, except for aVR which is expected).

One should remember that the dominant artery supplies the circulation to the AV node, so one should always be especially vigilant regarding delays or blocks at the level of the AV node when there is STE in the inferior leads. With an occlusion of the LCx – *whether dominant or not* – one should also be aware that there is a very significant possibility that the LCx is also

providing the circulation to the SA node and the right atrium. So, a *proximal* occlusion of a dominant LCx artery has the potential of resulting in AV conduction delays or blocks as well as serious atrial dysrhythmias, such as atrial fibrillation, SA block or SA arrest. With the loss of the atrial kick to the LV in addition to the fact that most of the left ventricular myocardium has been stunned due to ischemia, what could go wrong here?

This is a very extensive myocardial infarction involving the inferior, posterior and lower lateral walls of the left ventricle. That doesn't leave a lot of normal myocardium to act as a systemic pump! (Keep reading… this gets a lot worse!) The STE in V5 and V6 is due to the occlusion of the posterolateral branch or perhaps the obtuse marginal branch of the LCx artery. Either or both vessels can serve the lower lateral ("apicolateral") wall of the left ventricle. And it does not require a second occlusion – a proximal occlusion will deprive all the distal branches of blood unless there is some supportive collateral circulation located distal to the occlusion.

But why does Lead I have ST elevation and not Lead aVL? Don't they usually act together? Yes, they do! And here is a real surprise: there IS ST elevation in Lead aVL! Look closely… see it now? No? I'm glad you didn't relent and say "Yes!" because there really is no STE ***that can be <u>seen</u> (!)*** in aVL. And here's the reason why…



You surely noticed that of Leads II, III and aVF Lead III had the *least* amount of STE. In fact, it hardly qualifies as STE. It should have more. Now if you look on the Hexaxial Reference Grid (right), you will notice two things…

1. Leads III and aVL are 150° apart. That's as far apart as two lead positive poles can be in electrocardiography. If you try 180° apart, you are actually on the negative pole of the same lead. This degree of separation allows these two leads to act *reciprocally*. Reciprocity can present in different ways and have very different effects on the ECG (more about that in another post).
2. Also, Leads III and aVL are in the same (frontal) plane. To act reciprocally, the two leads *must be in the same plane*.

The STE in Lead III has completely cancelled the STE in aVL and the STE in Lead aVL has *almost* (but not quite) cancelled the STE in Lead III; therefore, we see just a minimal amount of STE in Lead III and absolutely none in Lead aVL. In this case, we can easily surmise that there was more STE in Lead III than in aVL. So, this brings *a new addition to our initial diagnosis*…

In addition to *acute epicardial ischemia* in the ***inferior***, ***posterior*** and ***apicolateral*** walls of the left ventricle, there is also *acute epicardial ischemia* in the ***basolateral*** (*high lateral*) wall of the LV supported by the STE in Lead I and the concealed (cancelled) STE in Lead aVL! This makes

the infarction even larger: now the ***entire lateral wall*** AND the ***inferior wall*** of the left ventricle are infarcting. There really weren't just **THREE** vascular areas involved – there were **FOUR**!

With the information you gain from being able to **INTERPRET** the 12-lead ECG instead of just **READING** it, you are armed with more advanced knowledge to arrive at a more complete diagnosis and anticipate and manage any other adverse cardiac events this unfortunate patient may experience.

At Medicus of Houston, I teach ***Advanced ECG Interpretation*** – in both the **Boot Camp** and the **Masterclass**.

Come join us and be a ***PARTICIPANT***… not just an audience!