Analyzing an Acute MI

"Jones's Rule" and Primary Repolarization Changes

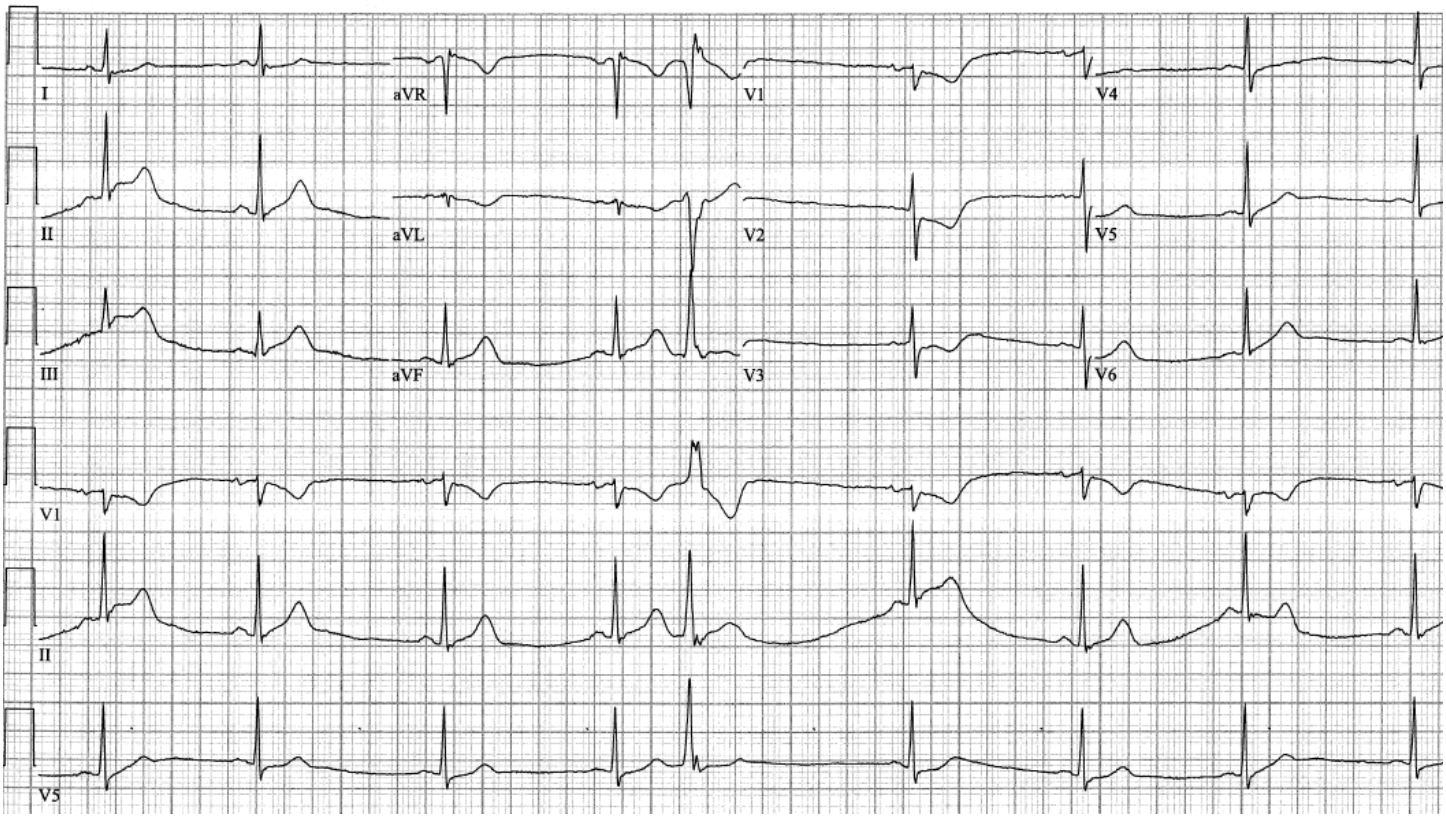

Figure 1

This is another 12-lead ECG randomly selected from my collection. There is a bit of baseline wander, *but that's life!* It was recorded on a 68 y/o Hispanic female in the emergency department who was complaining of chest pain. There is no other information about her.

First, the absolute basics. We can see by the upright P waves in leads I and II that sinus rhythm is present. The pos/neg biphasic P wave in Lead V1 is also supportive of sinus rhythm. The P wave in Lead V2 is basically isoelectric – but that's OK, as long as it isn't inverted or biphasic. There is 1:1 sinoatrial conduction and the P-R intervals are normal and remain the same. The rate is quite variable and may represent a sinus arrhythmia. Also, as we will see in a moment, there is also a good reason for increased parasympathetic influence on the rate. No apparent lethal dysrhythmias and no blocks are noted. Next, we turn our attention to repolarization (ST segments and T waves) …

When I am looking for abnormalities of the ST-T, more specifically, *subendocardial* or *subepicardial ischemia*, the first lead I specifically inspect is Lead aVL. Of course, just like you, I will immediately notice any *obvious* ST elevation or depression but Lead aVL has such an *infamous* reputation for being overlooked and/or misinterpreted that I always look there first! And what do I see? Obvious *ST depression* and *T wave inversion*. Do I see any obvious ST

elevation in the limb (*frontal plane*) leads? Maybe… or maybe not. The inferior leads look suspicious but I'm not so sure, given the amount of baseline wander. Do I see any other ST depression in the limb leads? Yes, the ST segment in Lead I is depressed and flat.

Now what about the precordial (*horizontal plane*) leads? Well, there is certainly very obvious ST depression in Leads V1 – V3. Leads V1 and V2 are definitely right-sided leads (remember: **V2** is also called **V1R** and **V1** is also called **V2R**). Lead V3 can still see the right ventricle from time to time, depending on the rotation of the heart in the horizontal plane.

So, what do we have here? "*Basolateral" subendocardial ischemia* (Leads I and aVL) and "*anteroseptal" subendocardial ischemia* (Leads V1 – V3)? Remember: ***subendocardial ischemia does not localize… but reciprocal changes do!*** When you see ST depression on the ECG of a patient who is having classic chest pain compatible with an acute coronary syndrome (ACS), ***do NOT think of subendocardial ischemia first! Think of reciprocal change!*** Remember **Jones's Rule**…

Jones's Rule

Any ST depression on the ECG of a patient having chest pain or discomfort compatible with an acute coronary syndrome is a RECIPROCAL CHANGE to an acute ST elevation occurring elsewhere until proved otherwise. Always remember that the reciprocal change of ST depression may appear BEFORE the primary change of ST elevation and – even when BOTH are present – the reciprocal change may be more impressive.

If the ST depressions in Leads I and (especially) aVL are reciprocal changes, they would have to be reciprocal to primary changes (STE) in the inferior leads (II, III and aVF). Let's look very closely at the leads – especially Lead III. If we move out 60 to 80 msec from the J point, we DO see STE – not much, but it's there. And, after having enlarged the ECG on my computer, I will say that the ST elevation at 60 msec in Lead III is greater than in Lead II at the same point. If that were all that we had as proof of an acute inferior MI, we might have a tough time selling that idea to a cardiologist. However, it won't be a problem in this case because of the ST depressions in V1 – V3. That is an obvious posterolateral (formerly *posterior* and soon-to-be *lateral*) MI. Actually, *there is no MI present on this ECG* – what we are seeing is an ***acute epicardial ischemia*** of the inferior and posterolateral walls of the left ventricle. *There is still time to save the myocardium!*

Let's go back to the frontal plane (limb) leads for a moment. The STE involving the inferior leads is very, very subtle. The reciprocal ST depression in Leads I and (especially) aVL is NOT subtle. It is easily seen. This is a case where *the reciprocal changes are much more visible and impressive than the primary change of ST elevation*. We might even be able to say that the *reciprocal*

changes of ST depression have appeared before the *primary* changes of ST elevation. ***Don't ever forget that!*** I have seen too many patients sent out of the ER with ECGs just like this one – diagnosed as subendocardial ischemia and given an Rx for oral nitrates.

In reference to something I mentioned earlier regarding the rate and rhythm – there can be a lot of parasympathetic input during an acute inferior epicardial ischemia causing a variable, non-respiratory related sinus arrhythmia.

Let's return now to the horizontal (precordial) leads.

The first thing that I want to discuss is the QRST complexes in Leads V1 – V3. This is very typical of the reciprocal change to a posterolateral *subepicardial ischemia* (formerly "posterior MI"). As I said before, *there is no MI present on this ECG* – we are dealing with *subepicardial ischemia only*. Look at those leads carefully once again.

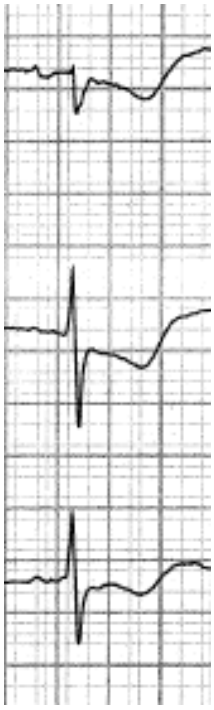

Figure 2

Figure 2 on the left is from the ECG posted at the beginning of this article and Figure 3 on the right is from another ECG in my collection. Notice anything different? In our 12-lead ECG (Figure 2), the T waves in V1 – V3 are ***inverted*** while the T waves in Figure 2 are ***upright***. The ST segments in the Figure 2 are mostly *downsloping*, while the more "classic" ST segments in Figure 3 are *flat*. Therein lies the problem. ***Remember: there is no T wave inversion during acute epicardial ischemia until the ischemic episode is over and reperfusion has begun***. The T waves on this ECG snippet (Figure 3) are *upright* – but remember everything is being *recorded in reverse.* These are changes reciprocal to the primary STE occurring on the opposite side of the heart. What you are *really* seeing in Figure 3 is *ST elevation with inverted T waves in the posterior (V7 – V9) leads*. ***This is an epicardial ischemia that is over and reperfusion changes are now taking place***. In other words, the snippet in Figure 3 – with its "classic" changes of an acute posterolateral MI is exactly *that* – ***a completed MI!*** The Q waves have already appeared, which in V1 – V3 will look like R waves.

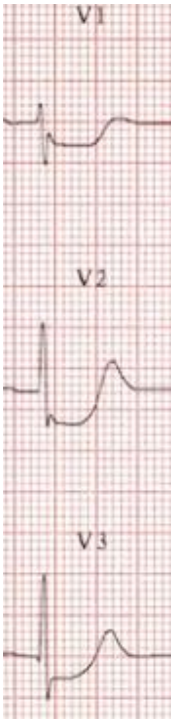

Figure 3

If we were viewing Leads V1 – V3 from our 12-lead ECG (Figure 2) in reverse, we would see ST elevation with an upright T wave. While the R waves would be seen as Q waves, Q waves can appear early in the course of an acute epicardial ischemia, caused by conduction delay through the ischemic area(s) and do not necessarily indicate dead tissue. The fact that the T waves have not begun to invert adds support to that concept here in Figure 2.

When Dr. Joseph K Perloff wrote his famous paper, *The Recognition of Strictly Posterior Myocardial Infarction by Conventional Scalar Electrocardiography* back in 1964, all his examples

of "posterior MI" were indeed *completed* myocardial infarctions. Because we use the term *myocardial infarction* so loosely nowadays in referring to ischemias that have not yet infarcted, we too often forget that a true posterolateral epicardial *ischemia* will be represented on the 12-lead ECG by *ST depression* and *T wave inversion* in Leads V1 – V3, NOT *upright T waves*.

Another issue I would like to discuss is the ectopic beat, best seen in the three rhythm strips at the bottom of the page. If we look in the Lead II and Lead V5 rhythm strips (Figure 1), the ectopic beat looks pretty narrow – perhaps a PAC or PJC with a small bit of aberrancy. The QRS appears minimally widened and repolarization appears appropriate with upright T waves. A glance at the Lead V1 rhythm strip, however, leads us to the correct diagnosis – a classic *left-sided* PVC! We know it originated in the left ventricle because it has a right bundle branch block-*"like"* morphology. And, because it does NOT have a *classic* RBBB morphology (rSr') and the left peak of the notch appears slightly taller than the right, we are sure this represents *ectopy* and not *aberrancy*.

But there are still a few more issues with this ventricular ectopic beat. The PVC in Lead V1 is pretty classic for a PVC – widened QRS with repolarization (ST segment and T wave) directed in the opposite direction. But look at the same PVC in Leads II and V5. We can understand that what appears to be a relatively narrow QRS is actually widened but where are the typical repolarization changes? We aren't seeing the expected repolarization changes *due to the ischemia in the left ventricle*. Remember when I said that this was a classic ***left-sided*** PVC? This PVC originated in the left ventricle and possibly in the ischemic area or in the margin between ischemic and non-ischemic myocardium. The reason you aren't seeing the classic repolarization changes in those leads is due to the effect of a *primary change of ischemia on the affected leads. That will cause repolarization to appear on the same side of the baseline as the QRS*.

There is still one more issue with the PVC: did it have an effect on the sinoatrial node? Did it

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PEARL!

Do NOT assume that a non-compensatory pause must be LESS than two normal P-P intervals. It can be MORE than two P-P intervals!

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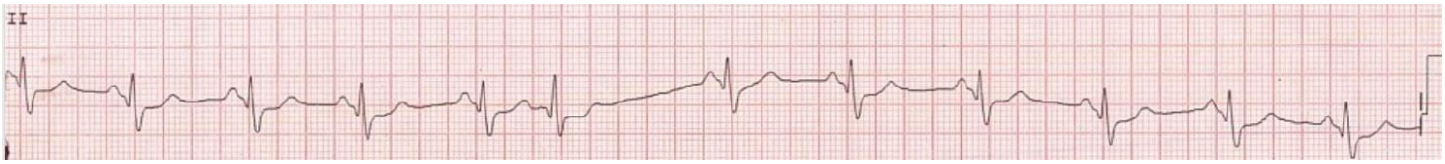
reach the SA node and reset it… or not? Let's measure the post-ectopic pause and see. If we find a *compensatory* pause, then we know that the SA node was never bothered by the PVC and continued to discharge as it had been. *A compensatory paused is recognized when the P-P interval surrounding the ectopic beat is equal to TWO normal P-P intervals*. [***Please note that I said P-P intervals and NOT R-R intervals! We do NOT use R-R intervals in measuring compensatory and non-compensatory post-ectopic pauses.***] But did the PVC manage to enter the SA node and reset it? If so, then our measurement of the P-P interval surrounding the ectopic beat will NOT be equal to two normal P-P intervals.

Using calipers, we find the P-P interval surrounding the ectopic beat to be *less than two normal P-P intervals*. So the PVC DID enter the sinoatrial node and reset it.

There is a continuing misperception about PACs, PVCs, compensatory pauses and non-compensatory pauses. I recently read an article posted online (though not on LinkedIn), that stated that PACs *always* resulted in non-compensatory pauses and PVCs *always* resulted in compensatory pauses. That concept was used as a "Pearl" to help you "distinguish" between a PAC conducted with aberrancy and a PVC. Be very careful! There is a lot of serious misinformation regarding ECGs floating around on the web.

Some of you may be wondering how a non-compensatory pause can be ***LONGER*** than two normal P-P intervals. Usually, when an ectopic beat enters the SA node and resets it, the pacemaking cells in the node are discharged and then immediately repolarize down to their normal resting potential (~ -60mV). On rare occasions, however, the pacemaking cells are ***hyperpolarized***, i.e., repolarized to a much more negative membrane potential than usual – perhaps to -80 or -90 mV (remember: their *normal* resting potential is around -60 mV). In such cases it will take them longer than usual to depolarize up to their threshold potential, so the first post-ectopic sinus P wave may appear well AFTER two normal P-P intervals. Here is such a case from my ECG collection involving a PAC…



I hope you learned something from this 12-lead ECG. At least, I hope you learned that there is seldom just a single issue on an ECG!

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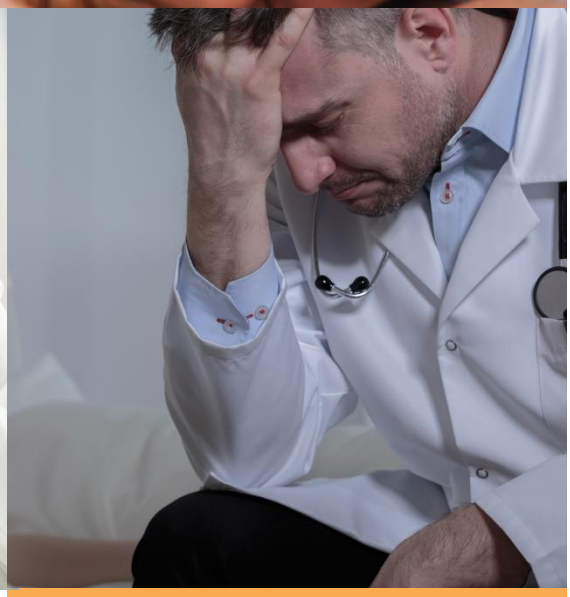
Is there something you're just not understanding?



Are you having difficulty reading and interpreting ECGs? Do you find yourself depending on the machine interpretations? Have you experienced a "bad outcome" because an ECG was misinterpreted? Have your ECG interpretation skills come to the attention of your hospital's peer review committee?

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