Signs of Occlusion Myocardial Infarction (OMI)

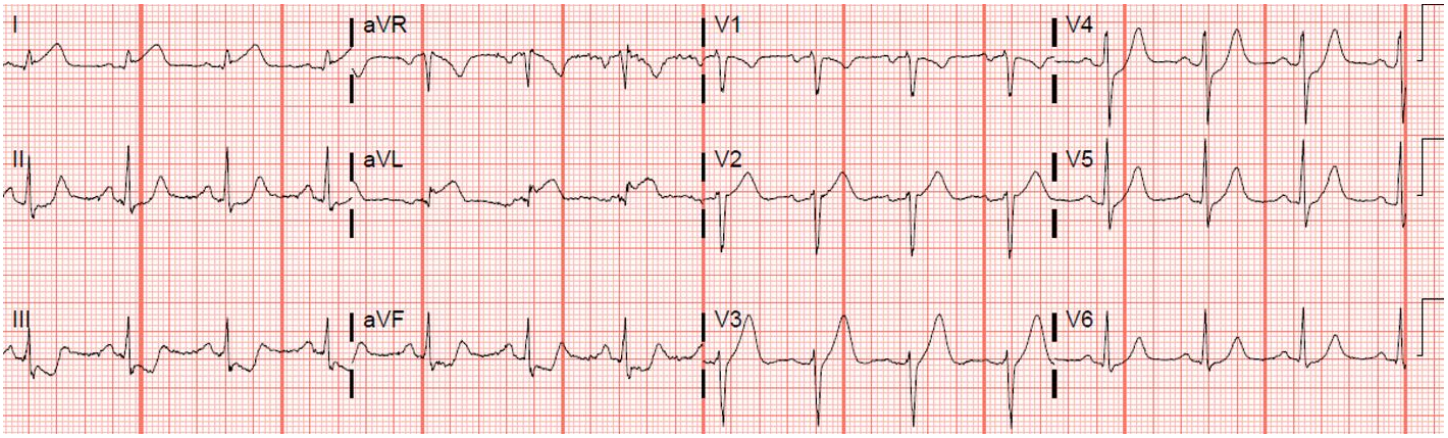


Figure 1

This is another ECG that was sent to me. It appears, on the surface, to be a fairly obvious diagnosis. And, on a superficial level, the main diagnosis is obvious: there is an OMI present. Most people still adhere to the STEMI concept – but there are problems with that. The difference between an OMI and a STEMI is this…

With an **OMI**, diagnosis is based more directly on diagnosing a coronary vascular *occlusion*.

With a **STEMI**, diagnosis is based on the 12-lead ECG appearance which requires a pre-determined amount of ST elevation in certain leads in order to make the diagnosis. This approach puts the "non-STEMI" label on a lot of *complete coronary occlusions* which would otherwise benefit from emergent revascularization.

If a patient presents with chest pain, very elevated troponin I and has *STE in Leads II, III and aVF*, then he or she has an *inferior STEMI* – case closed! It's off to the cath lab!

But if another patient presents with *exactly* the same history, *exactly* the same risk factors, *exactly* the same physical exam and *exactly* the same troponin I – *but no ST elevation on the 12-lead ECG* – then he or she has a *"non-STEMI"* – case closed! It's medical management (unless the pain *persists* in spite of such management)!

But many of those "non-STEMIs" are actually STEMIs in which the provider *failed to recognize ST elevation*, or *failed to record enough leads*, or *failed to repeat the ECG after 20 – 30 minutes*. Or, in which the *ECG machine failed* to record any qualifying ST elevation.

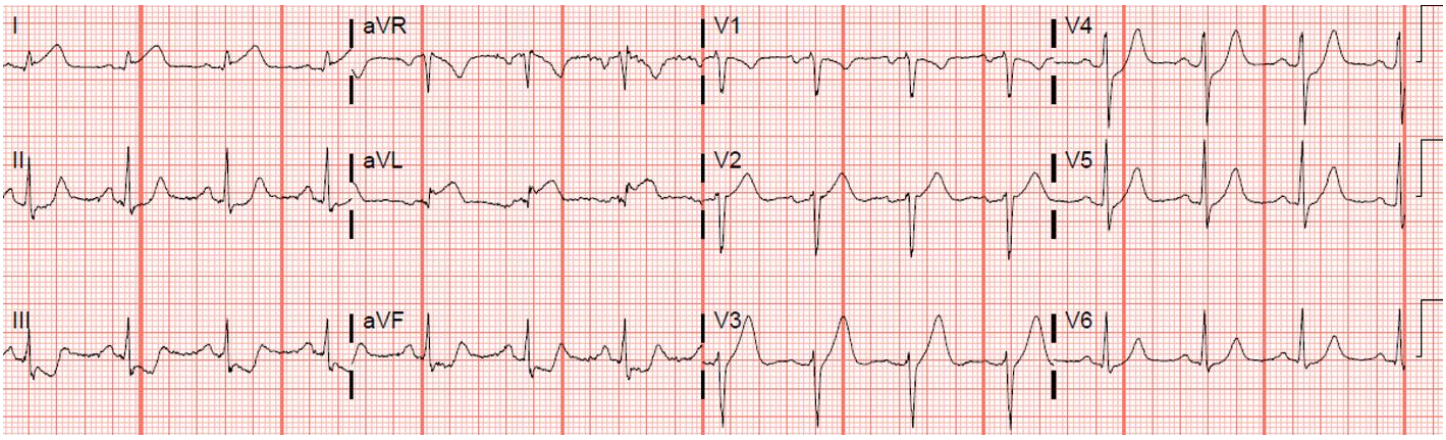


Figure 1 (repeated)

Before we get into any misunderstandings (by the "but that's not the way we've always done things!" crowd), please understand that ***all* true STEMIs are OMIs but *not* all OMIs are STEMIs**.

A **STEMI** is a *transmural ischemia* caused by the *occlusion of an epicardial coronary vessel*. It *will benefit from immediate revascularization*, whether by PCI (preferably) or by thrombolytics (when PCI is not an option).

An **OMI** is a *transmural ischemia* caused by the *occlusion of an epicardial coronary vessel*. It *will benefit from immediate revascularization*, whether by PCI or by thrombolytics. But it may not present with ST elevation. Remember: some "non-STEMIs" are actually real "STEMIs" that would also benefit from revascularization. Once a provider realizes this, he or she also immediately realizes that diagnosing an OMI is going to take a lot more skill than diagnosing an "obvious, textbook STEMI." There may indeed be ST deviations present, but they may be less than the standard 1 or 2 mm. The same applies to reciprocal changes which can also be very subtle. But let's keep the record straight: non-transmural ischemia/infarctions that are best managed medically do occur every day and the concept applies to both the STEMI and OMI paradigms. Today, we are concerned only with true transmural ischemias.

The first protest encountered usually comes from the cardiologists: "So, you want us to take *everybody* with chest pain to the cath lab?" I respond, "No, I want *all of us* to stop admitting patients with complete coronary occlusions for medical management."

There must be a way to reduce the number of patients with complete coronary occlusions and who present (presumably) as “non-STEMIs” that end up with medical management instead of emergent revascularization. There IS a way, but it will require much more than an introductory-level of skill in ECG interpretation. The physician or advanced practice provider who makes first contact with the patient is going to have a tremendous responsibility, and must be capable of advanced and sophisticated ECG analyses.

The most frequent mistake in misdiagnosing a “non-STEMI” typically involves lesions in the left circumflex artery (LCx). Occlusions of the RCA and LAD are generally easier to recognize, but the LCx can be very problematic.

The course of the LCx is furthest away from the anterior precordial recording electrodes which makes the 12-lead ECG less sensitive to LCx occlusions. And even when posterior leads (V7, V8 and V9) are placed for recording, they are still much further away from the *posteriorly-located* surface of the heart (which is actually the *lateral* surface) than precordial leads V1 – V4 are from the *anterior* surface of the heart. Recognizing acute epicardial ischemia in the LCx vascular areas is indeed very problematic.

To improve diagnostic accuracy and sensitivity, those interpreting 12-lead ECGs will have to develop greater levels of skill. I am disheartened by the number of medical providers who still rely on the machine interpretation and also by the number of providers who are still reading ECGs at an introductory level. I have people sign up for my courses (which I clearly state is for those with an intermediate skill level) who not only cannot recognize a bundle branch block – some have never even *heard* of a bundle branch block. And they are occasionally making life-or-death decisions based on their ECG interpretations!

And don’t feel you can wait for AI to take over the interpretations so you don’t have to interpret the ECGs yourself. AI will be a great adjunct, but the final diagnostic and management decisions will still be with the physician or advanced practice provider who has the responsibility for the patient. About six years ago, I was contacted by another fairly well-known ECG instructor who told me she was working with an AI company and that their ECG interpretation program based on AI would make ECG interpretation by a human obsolete in 2 years. Did I mention

that was six years ago? Besides, if the world turns ECG interpretation over to an AI program and it misdiagnoses an ECG with tragic results – you can't sue a machine – just the person who accepted the machine results *without question*.

Switching from the STEMI concept to the OMI concept will require more skillful ECG interpretation. But doing so is the only way we are going to improve our diagnostic accuracy. Now let's return to our ECG for this post…

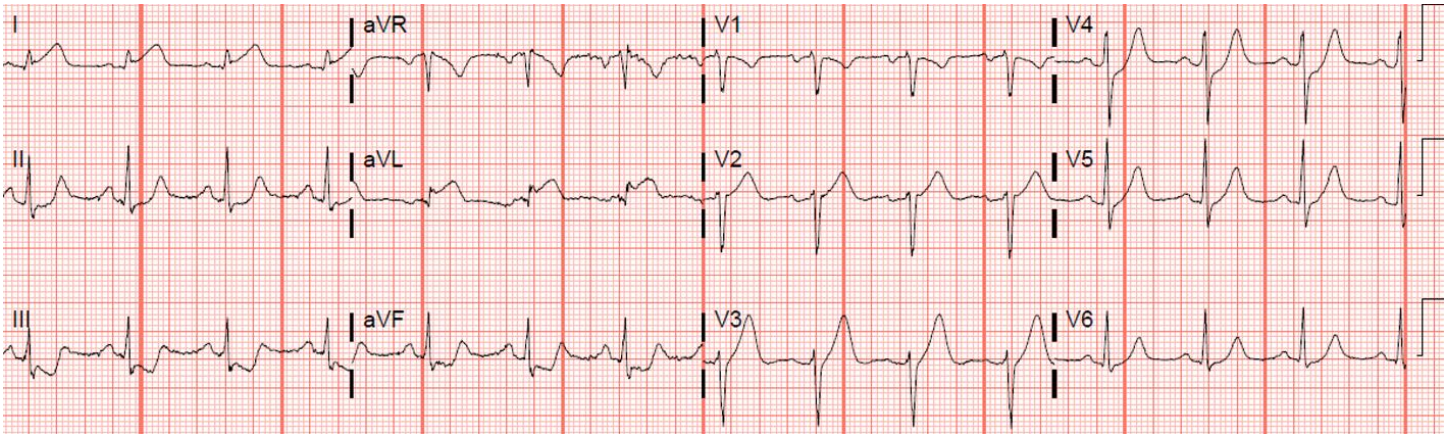


Figure 1 (repeated)

Looking at the limb leads (I, II, III, aVR, aVL, aVF), what did you notice first? I noticed the ST depressions in the inferior leads first (probably because the QRS complexes are larger); then I noted the STE in Leads I and aVL. But I did all that in less than one second. The instant I saw the ST depression I focused on Leads I and aVL, since the two sets of leads are reciprocal to each other.

OK, so there is an acute epicardial ischemia (a STEMI) involving the *basolateral* area of the left ventricle. This used to be called the "high lateral" area as opposed to the areas covered by Leads V4 and V5 and somewhat V6 (I say "somewhat" because Leads I and V6 cover the same area). This means that there is an occlusion of either the LCx or the LAD. I know the RCA is not involved because it doesn't cause basolateral ischemia (no STE in Lead aVL – ever!). So far, I have used only those skills one could acquire in most introductory ECG texts.

Now let's look at the precordial leads. Is there any acute epicardial ischemia present in V1 – V6?

NOTE: Some of you may wonder why I have not mentioned *transmural ischemia*. The reason is because using the term "epicardial ischemia" *implies that the ischemia is transmural*. Ischemia always begins in the *subendocardium*, and then

extends outward toward the epicardium. Ischemia does not begin in the epicardium.

Acute epicardial ischemia implies ST elevation and there is certainly NO ST elevation in the precordial leads. Is there any other indication of acute ischemia?

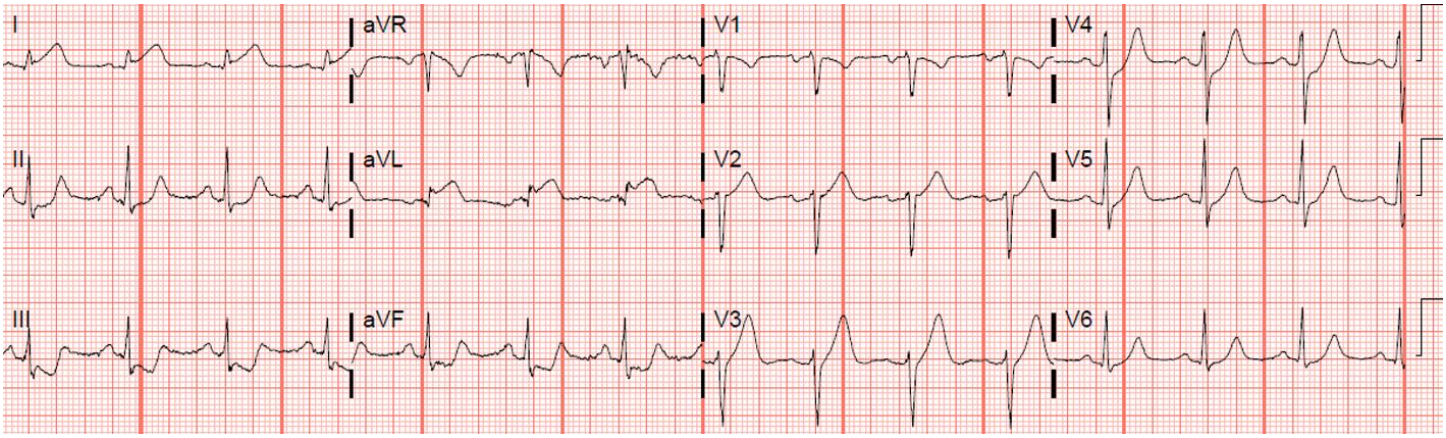


Figure 1 (repeated)

Yes there is!

First, I want you to look at the T waves in Leads II, III and aVF – the inferior leads in the frontal plane. What do you notice about those T waves *other* than the fact that they are all upright? (Note: we define a T wave's morphology as *upright* or *inverted*, depending on its *terminal portion*.) You notice that there is baseline (ST segment) between the end of the QRS (the J point) and the upslope of the T wave. What else do you notice about the T waves? Look at the width of the base of each of those T waves – they aren't very wide, are they? Good.

Now look at the T waves in the precordial leads. I want you to notice two things…

First, look at the bases of the T waves in V2 and V3. Notice the width and compare it to the T waves in the inferior leads. The difference is very obvious… right? Compare the width of the T wave in Lead V3 with the T wave in Lead aVF. A very obvious difference in width!

Second, look at where the upslope of the T waves in V2 and V3 begins… and then look at where the upslope of the T waves in the inferior leads begins. In Leads V2 and V3 there is essentially no separation between the end of the QRS complex and the upslope of the T wave. Those are **classic hyperacute T waves** in Leads V2 and V3!

Do ALL hyperacute T waves begin at the end of the QRS? No… we're going to look at some that don't in a moment. However, *hypercalcemia* can do the same thing. So, if you have a patient with severe chest pain and diagnostic findings of an acute STEMI with hyperacute T waves that begin at the end of the QRS, but… he is taking a lot of calcium supplements – you should check for hypercalcemia, also. But you'll find that it is generally not necessary.

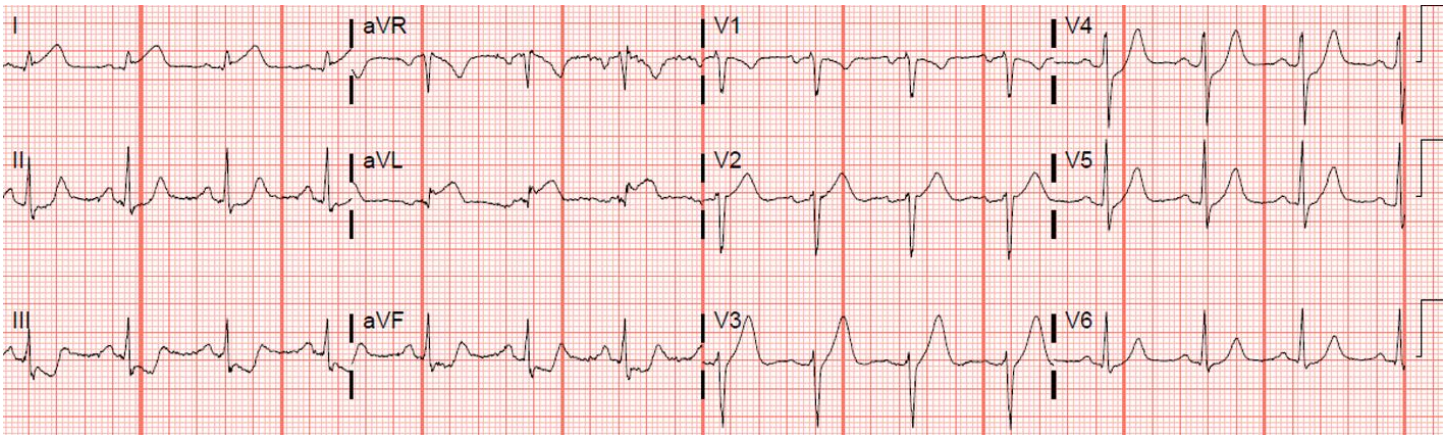


Figure 1 (repeated)

Now let's look at leads V4 – V6. All the T waves appear a bit too large for normality. Here are some normal T waves in Leads V4 – V6…

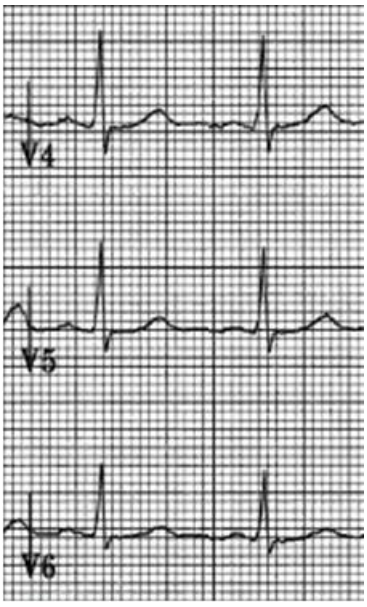


Figure 2

These T waves are very normal in Figure 2. Look at the difference between them and the T waves in the corresponding leads in Figure 1.

The T waves in Leads V4 – V6 in Figure 1 are also hyperacute T waves – *but with a slight difference*. They begin below the baseline and after the ST segment. Are these de Winter T waves?

While Leads V4 – V6 bear some resemblance to de Winter T waves, I don't think they are for TWO reasons…

Reason #1 – They just don't LOOK enough like de Winter T waves.

Reason #2 – I'm going to show you some examples of de Winter T waves (from the *Life in the Fast Lane* website) and let's see if you can determine the second reason that Figure 1 is not likely to represent de Winter T waves…

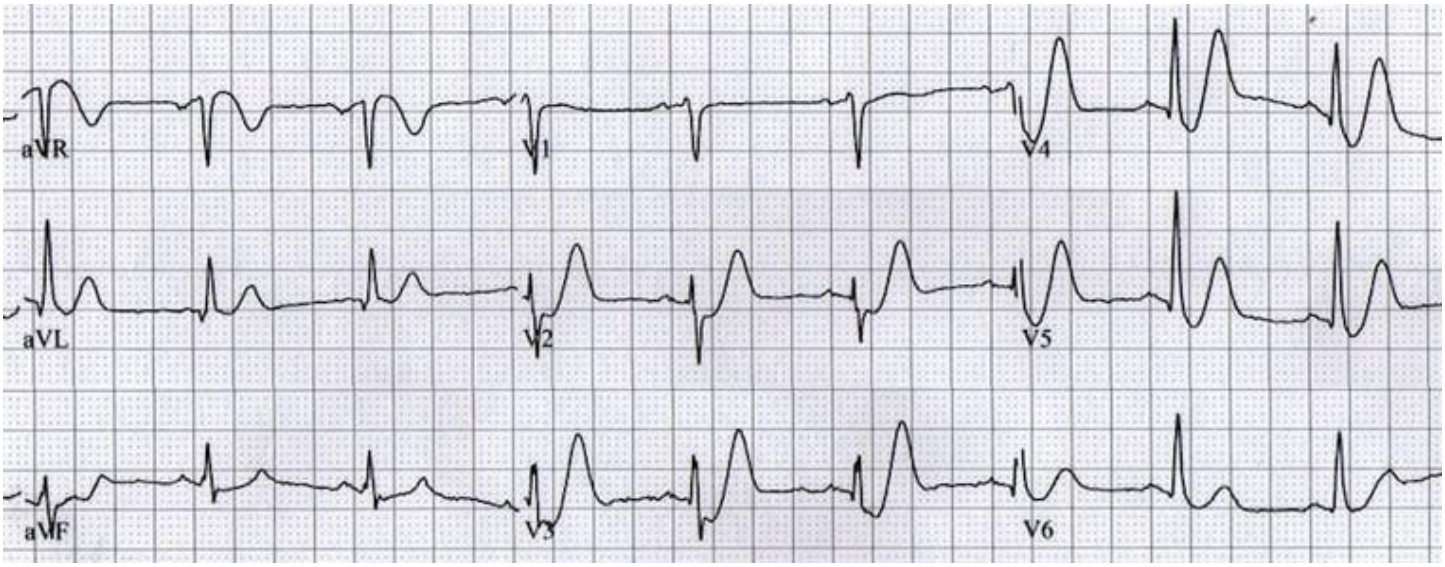


Figure 3

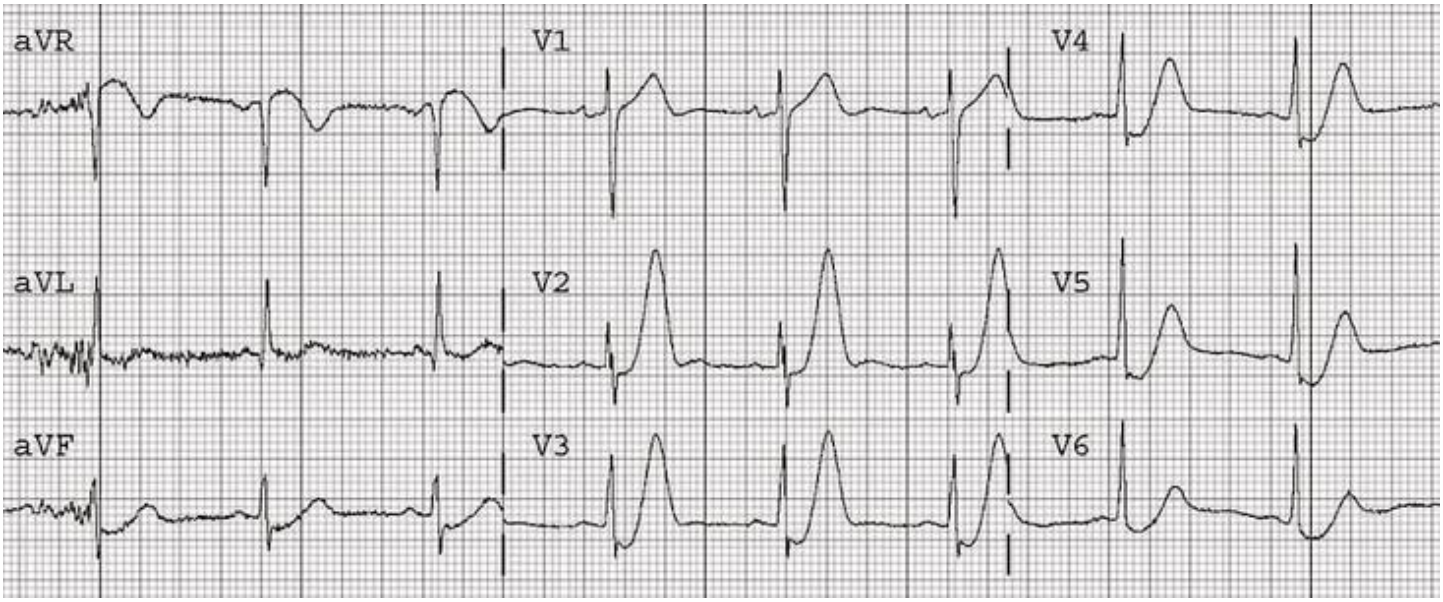


Figure 4

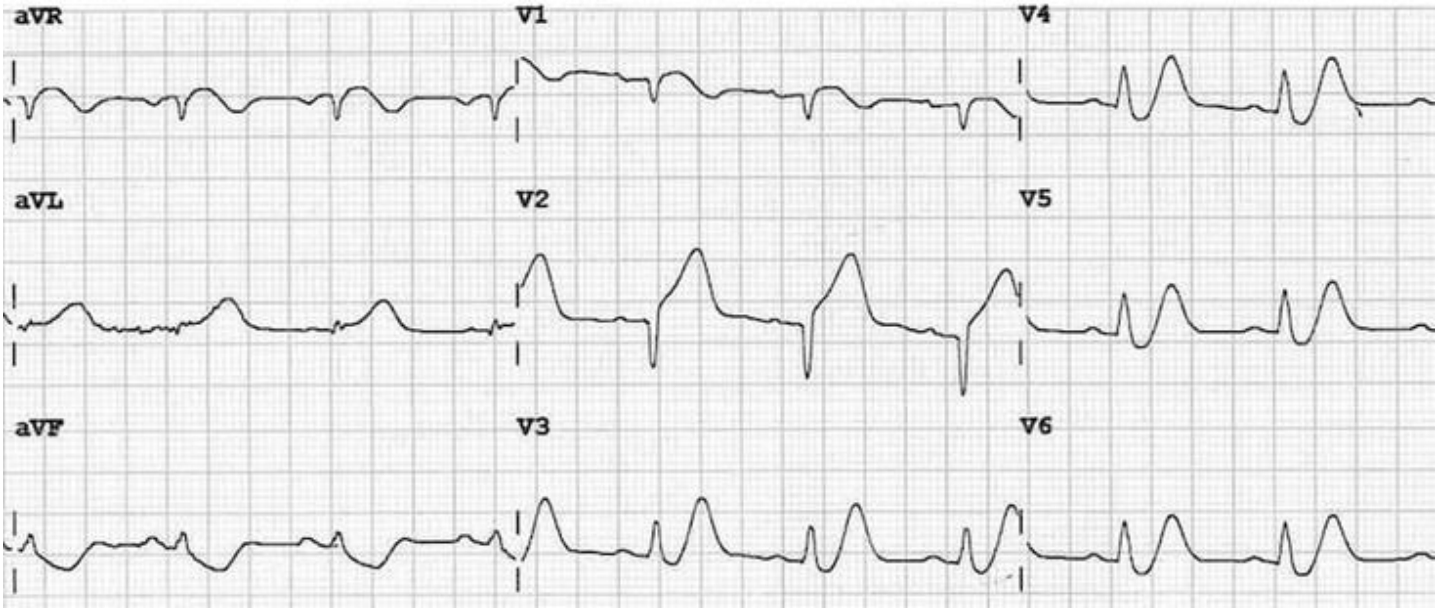


Figure 5

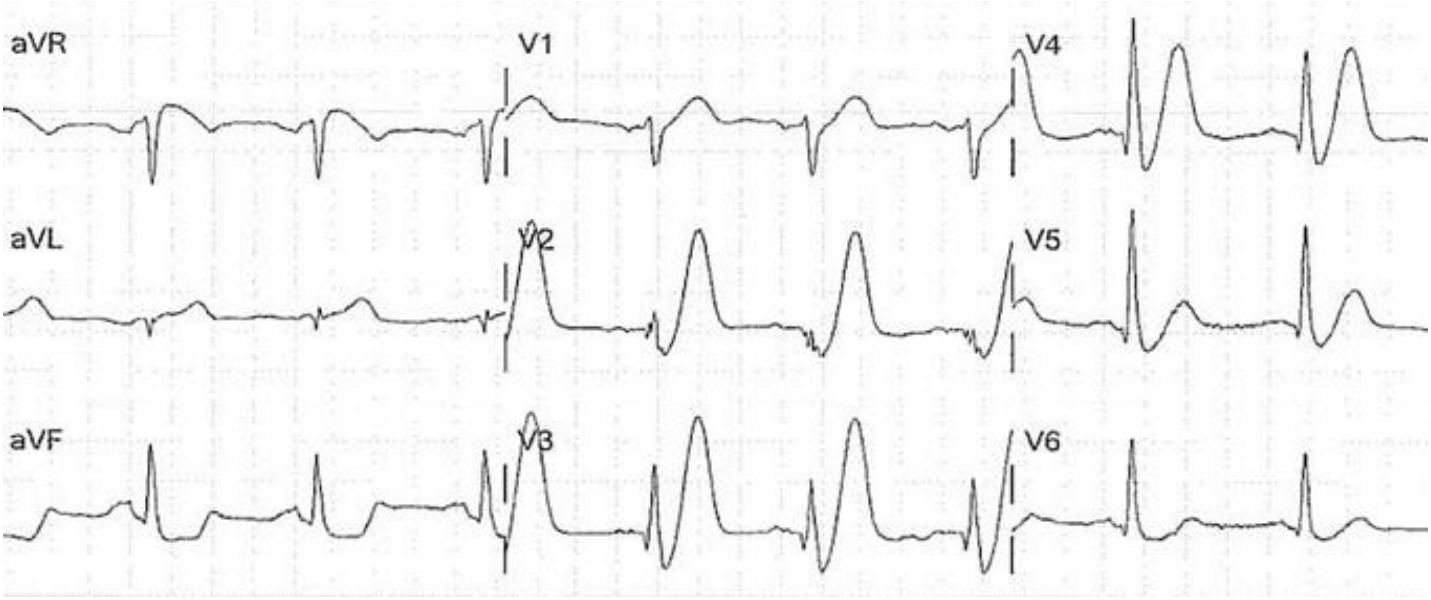
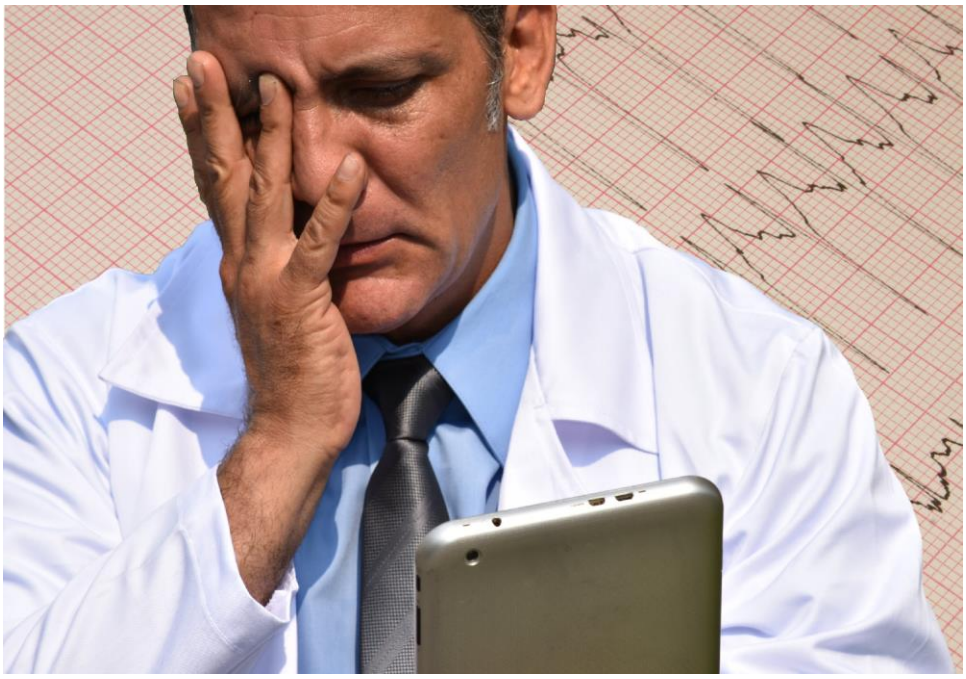


Figure 6

Do you see the second reason why I think that Leads V4 – V6 in Figure 1 do NOT represent de Winter T waves? If you still don't, then strongly consider enrolling in the ***Masterclass in Advanced Electrocardiography***. In the meantime, I'll give you a hint: focus on Lead aVR in all the ECGs. You'll notice that with every manifestation of de Winter T waves, there is *very obvious ST elevation in Lead aVR but not in the ECG in Figure 1* (however, you can *still* enroll in ***The Masterclass in Advanced Electrocardiography***).

I hope you enjoyed this short introduction to **Occlusion Myocardial Infarction** (**OMI**) and why it is going to lead to a greater need for very skilled ECG interpreters in the near future. For more on this topic, visit Stephen Smith's website, "Dr. Smith's ECG Blog." (https://hqmeded-ecg.blogspot.com/). Dr. Ken Grauer is also a regular contributor to the blog. I recently taught one of the ER physicians who works with Steve Smith at Hennepin County Hospital in the ***Masterclass in Advanced Dysrhythmias***. He had recommended the course to her. He later wrote to me that she had *loved* the course!



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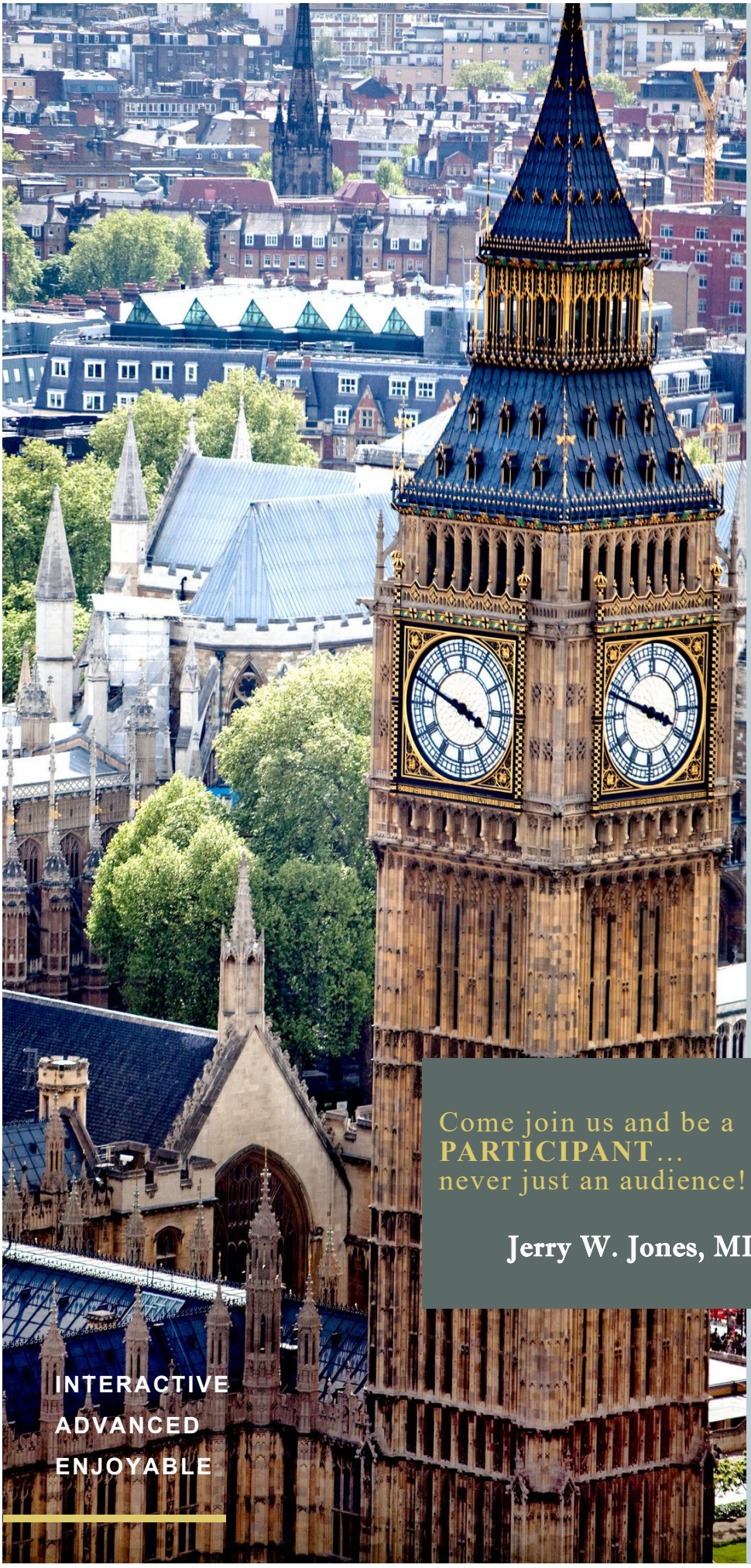

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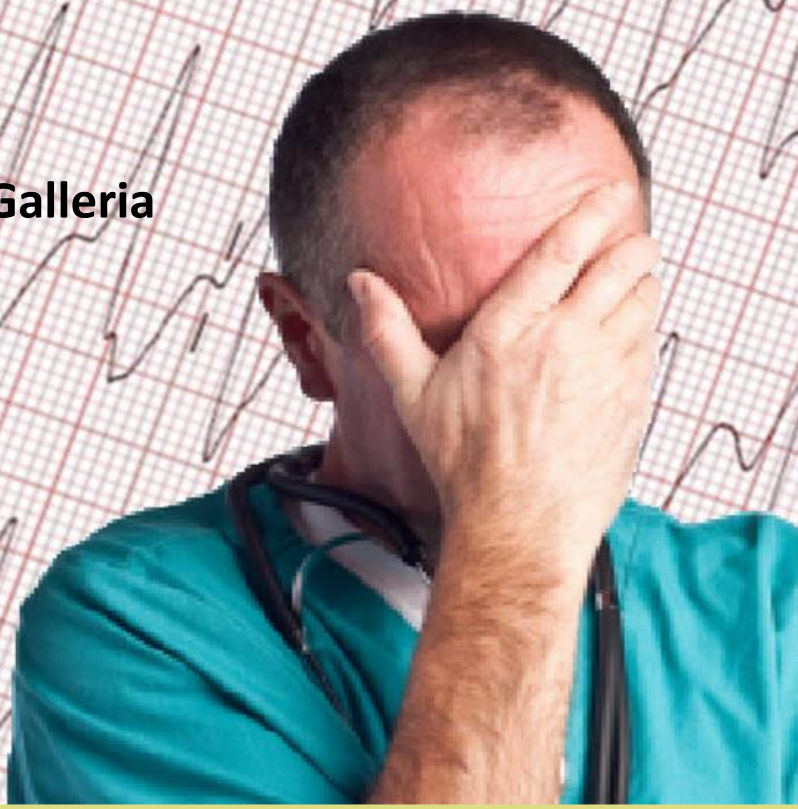

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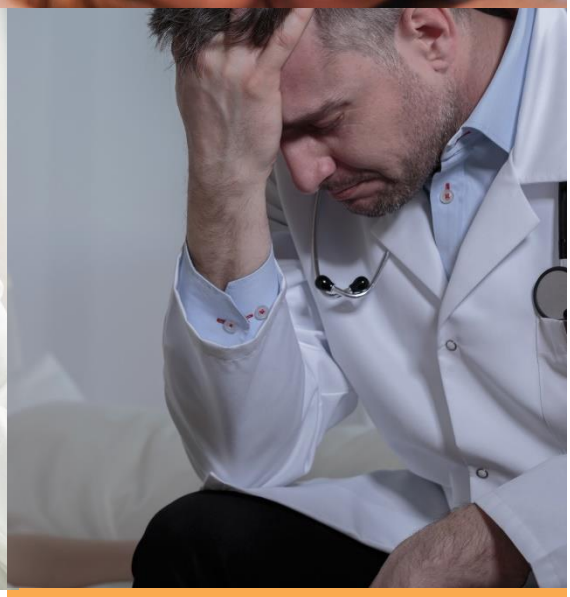

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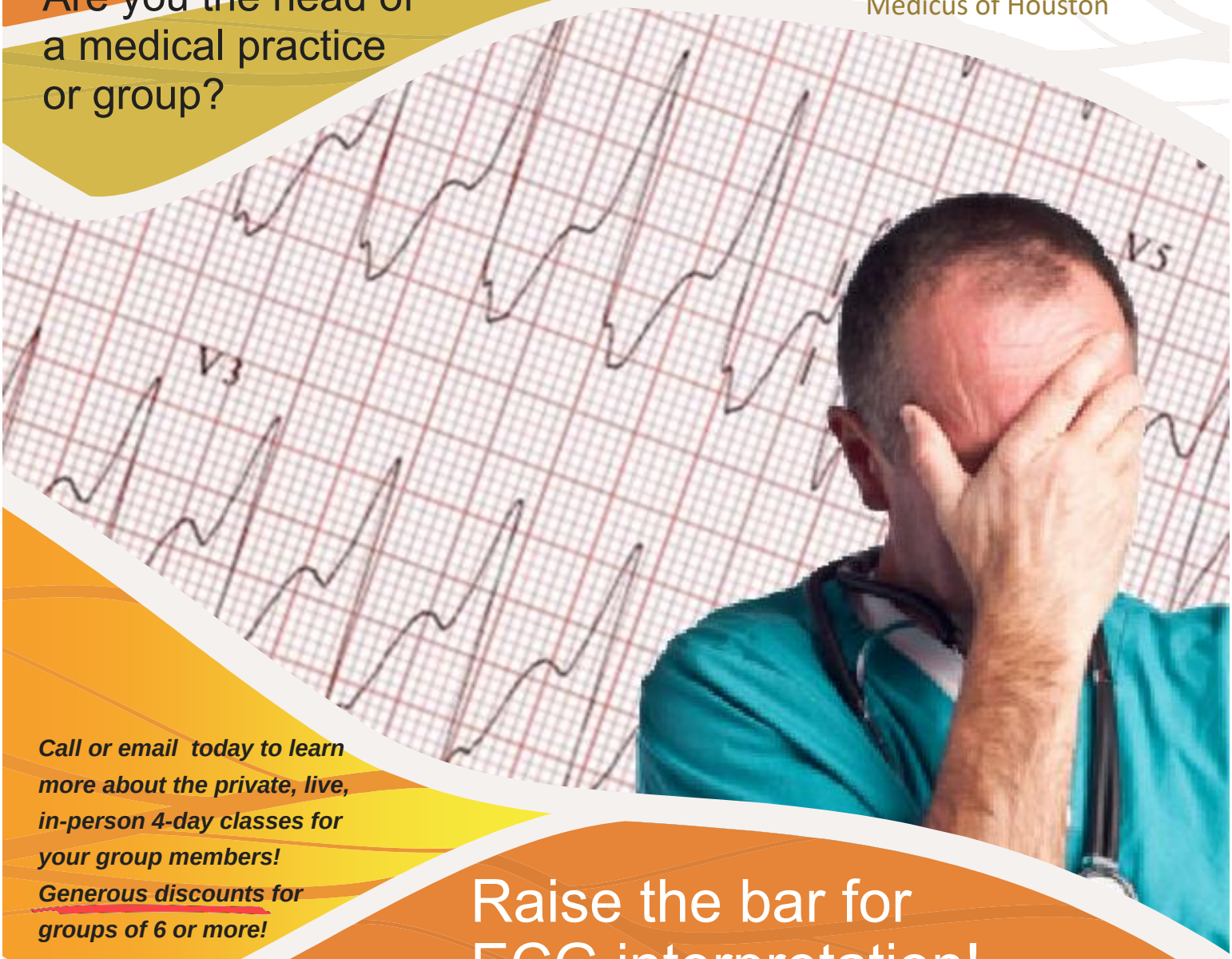

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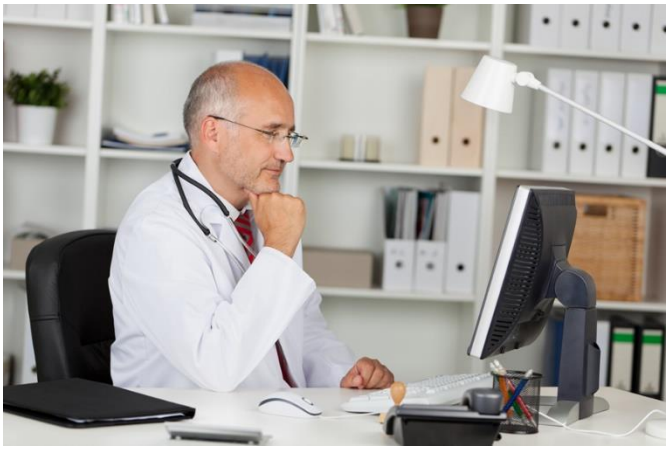

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If so, are any of your physicians or advanced practice providers struggling with 12-lead ECG interpretation?

Are you a physician, nurse practitioner or physician assistant who is facing peer-review, license suspension or suspension of hospital privileges due to poor ECG interpretation skills?

Has an incorrect interpretation of a 12-lead ECG resulted in litigation?

I can't change what's happened in the past, but I can certainly help you with your ECG interpretations in the future. I am Jerry W. Jones MD, the CEO and chief instructor of *advanced electrocardiography* for **Medicus of Houston**. I have taught ECG interpretation informally for over 30 years but in 2011 I formed my *own* CME company – **Medicus of Houston** – and have devoted myself to teaching full time. Just ***advanced electrocardiography*** – nothing else.

Based in Houston, Texas, I have presented the ***Masterclass in Advanced Electrocardiography*** in Toronto, Vancouver, Budapest, London, Sydney and, of course, in the United States (Houston, San Antonio, New Orleans, Savannah). I have also lectured on ECG interpretation at scientific assemblies for the **European Society of Emergency Medicine** (Vienna, Austria), the **Dutch North Sea Emergency Medicine Conference** (Egmond aan Zee, Netherlands), **American Academy of Emergency Medicine** (San Diego, CA and Las Vegas, NV), the **American College of Emergency Medicine** (Las Vegas, NV) and the **Urgent Care Association of America** (Las Vegas, NV).

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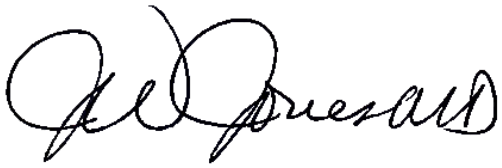

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