Here Is Where Mistakes Happen…

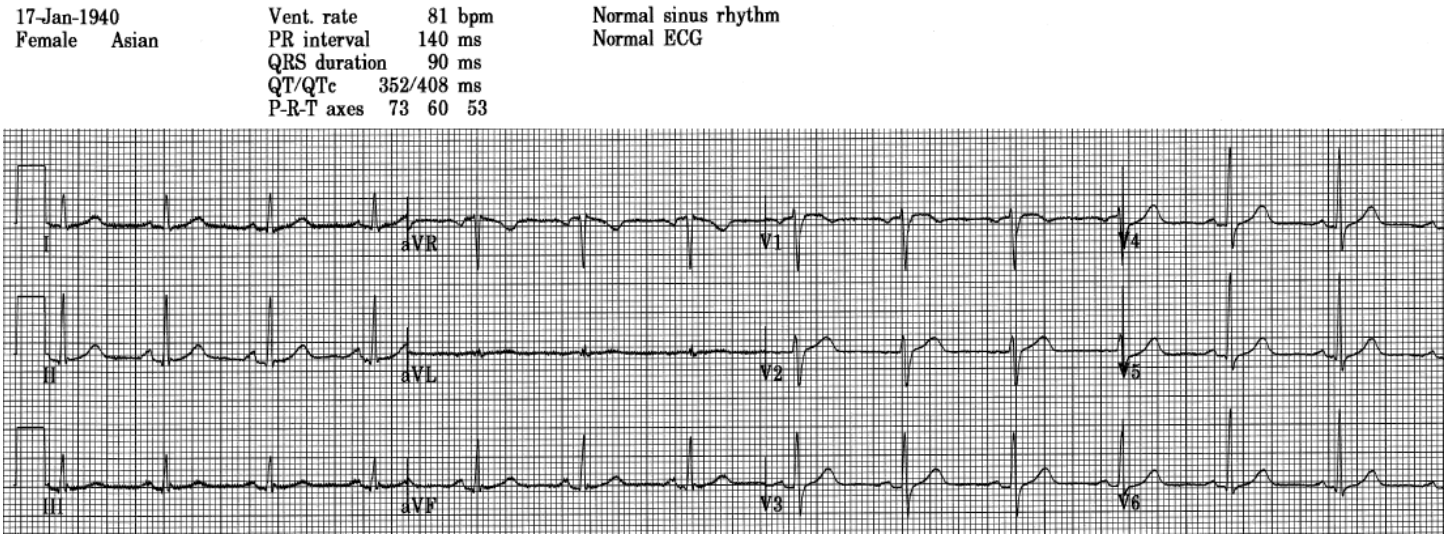

Figure 1

Today, we are going to look at a seemingly normal ECG (actually, it really *is* normal) so that I can point out the areas where you must be especially careful with your interpretation. These are the places where – if you are going to miss something – it will most likely happen.

The ECG in Figure 1 was recorded on a 71 year old female of Asian ethnicity in an urgent care center. I do not know why she was being seen, but let's consider chest pain as her chief complaint. She is in sinus rhythm (upright P waves in Leads I and II with Lead II having the largest P wave). The mean QRS vector in the frontal plane (ÂQRS) is +60° according to the ECG machine. Does that seem appropriate? The QRS in Lead II has the *tallest* R wave and the Lead perpendicular to it – Lead aVL – has the *smallest* QRS complex, so yes, it is appropriate. This leads us to our first problem…

…the *size* of the QRS interval in Lead aVL. What can you say about Lead aVL? Not much because it is *so small!* Therein lies the problem… its size makes it very difficult to interpret it. What if there were ST elevation or depression in that lead? How much ST deviation would be required for us to say that ischemia – epicardial or subendocardial – is present? Current guidelines say 1 mm of ST elevation must be present to be of significance. But the entire complex has hardly 1 mm of magnitude… *if that!* It is in situations like this that the whole STEMI/NSTEMI paradigm breaks down. The next thing that we would do is look in the inferior leads for any sign of a reciprocal change and we see none. Does that rule out an acute coronary syndrome? Not at all, because if you learned **Jones's Rule** you would know that the ST depression of a reciprocal change is not always coordinated with the appearance of the ST elevation. It may appear *before* or *after* the appearance of any STE. Now let's be clear: I'm speaking *theoretically* at the moment – personally, I do NOT see any STE in Lead aVL, but at that size, it is certainly difficult to be sure.

That is why many of us are trending away from the STEMI/NSTEMI method of identifying acute epicardial ischemias. Too many acute coronary occlusions – *complete* occlusions – are being missed and written off as non-STEMIs receiving medical therapy initially rather than more emergent reperfusion interventions. Worse – some of these patients are getting discharged from hospital ERs inappropriately rather than being observed for changes that would indicate the correct diagnosis.

If you are going to miss an MI – *THIS is where it is likely to happen!* Be *extremely* careful with Lead aVL. One of my cardiology attendings when I was a resident suggested that its name should be changed from aVL to eVL (the "evil" lead!).

Let's look at Lead V1 now. Looking at the P wave, we see that it is monophasic and negative. A quick glance reveals an isoelectric P wave in Lead V2. These findings suggest that perhaps the adhesive electrodes for those leads were placed too high on the chest wall. However, I don't see any major consequences in this particular instance that would lead me to a misdiagnosis. Now let's focus on the ST segment in Lead V1. The amount of ST elevation at the J-point is just under 1 mm, but it's very close. Actually, it appears under 1 mm by the width of the inked line. Let's look at an enlargement…

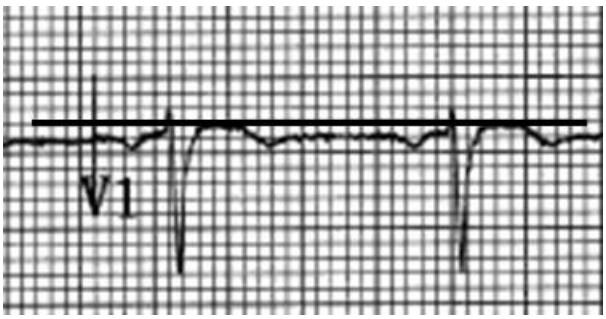

Figure 2

I've drawn a 4 pixel line 1 mm above the TP segment (baseline). "But," you say, "there can be up to 3 mm of STE in Lead V1 normally." I would say you are correct… except that the allowance of up to 3 mm STE in Lead V1 applies *only to men under the age of 40 years*. Our patient is a 71 year old female. For females, only 1 mm of "normal" STE is allowed in Lead V1 and that applies to women of ALL ages. Still, the STE in Lead V1 on this ECG is under 1 mm (barely). There really is no suggestion of STE in Leads V2 or V3, so we needn't be concerned about a "suspected" STE in Lead V1… right? There are two things to remember here that *might* suggest the need for extra leads:

1) about 15% of hearts will have a left-dominant system, and
2) 5% of ALL acute MIs are *isolated* to the RIGHT ventricle.

When a left-dominant system is present, the RCA typically ends after the acute marginal artery. A proximal occlusion of a non-dominant RCA will knock out the conus artery which perfuses the right ventricular outflow tract and the right basilar septum and which also acts as a major collateral to the left-sided circulation. In 55% of cases, it will also result in occlusion of the artery to the sinus node (be aware that "artery to the sinus node" is really a euphemism because that artery supplies just about ALL the circulation to the right atrium!). In these cases, a right

ventricular infarction may occur *without* the usual accompanying STE in the inferior leads. Again, that occurs in up to 5% of *all* acute epicardial ischemias!

Now, many of you may say, "Why should I look for an isolated right ventricular infarction when I know the chance of finding one is *low*?" Please allow me to rephrase exactly what you are saying: "Why should I record a few extra leads – *at essentially no extra cost to the patient* – when nothing else has shown up on this patient with chest pain in order to see if she might have an MI that we *know* occurs in up to 5% of cases?" When you say that a test's yield is *low*, you are also saying that the test's yield is NOT ZERO! In every differential diagnosis, we arrange potential diagnoses in order of likelihood – and that's very appropriate. In this patient, let's say we have ten conditions in our differential diagnosis and isolated right ventricular infarction is number 10. All the probabilities will add up to 100%. With the elimination of each condition beginning with the first and most likely diagnosis, the probabilities change and that "zebra" at number 10 becomes more and more likely. When I was practicing emergency medicine, I worked in the hospital associated with the Texas Heart Institute here in Houston. Can you guess how long it would take me to diagnose 100 acute myocardial infarctions? It could take me just *a few weeks* – not even *months*. And 5% of those could easily have been isolated right ventricular infarctions. Certainly, I am *not* advocating recording right-sided leads on *every* chest pain patient – especially as a first step in your work-up – but sometimes it *is* very appropriate to record them after you have ruled out everything else in your differential diagnosis – *even when there is no inferior STE present*. ***I can think of no worse reason to justify NOT doing a test than "the yield is low."*** Remember: a test whose yield of a positive result is low, is a test whose yield of a positive result is NOT ZERO! If you want to talk about ZERO results, do a malaria screen as part of your ACS work-up… *then* you *would* have more reason to complain about a "low yield test." (FYI: in *this* particular patient, if I *really* suspected an acute coronary occlusion, I would record posterior leads *before* right-sided leads.) So, if you decide NOT to do a test because the "yield is *low*," you'd better think of several *better* reasons for not doing the test because I assure you that a plaintiff attorney will be *very* aggressive in pursuing a response that will make you appear uncaring and/or incompetent to a jury.

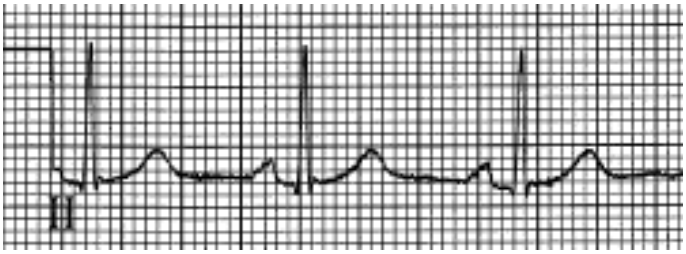

Figure 3

One last issue where a mistake can be made: look at Lead II. Is there ST depression present?

The answer is "No." You may get the impression that ST depression is present (especially following the 3rd QRS complex), but there is none. Remember that the P wave is the atrial equivalent of a QRS complex and, just like the QRS, it also has a T wave representing repolarization. Usually, the atrial T wave (Ta wave) is

hidden by the QRS complex; but sometimes, when the QRS is narrow, it is more visible. Always look for the PR and ST segments to form the bottom of a hyperbolic curve.

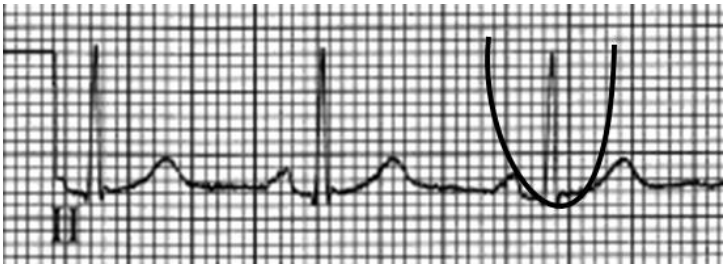

Figure 4

I hope you have learned some practical information from this essentially normal ECG and from my short discourse on doing tests that have a low yield for a positive result. During my years as an attending physician, I found that many residents develop the mistaken idea that a low-yield test should *never* be done. Yes… they ARE indicated *at times*.

Don't forget that my next live, in-person class will be ***The Masterclass in Advanced Electrocardiography*** July 24 – 27, 2023 in exciting San Antonio, Texas. It lasts four days and is an intense study of ECG *interpretation*. Twenty-seven (28) AMA PRA Category 1 Credits™ will be awarded for completing the course. Learning to *read* an ECG is like learning the multiplication tables: every mathematician has to do it, but they really serve no usefulness until used to solve a mathematical problem. Reading an ECG has little usefulness until you use those findings to decide not only what *happened* to the patient, but also what *is happening* and what *may happen*.

I still have some time available this summer for private tutoring sessions via ZOOM if you need help with a specific area of ECG interpretation. Though I teach advanced ECG interpretation, the tutoring sessions – which last one hour – can cover introductory concepts as well.

To learn more about ***The Masterclass in Advanced Electrocardiography***, please visit:
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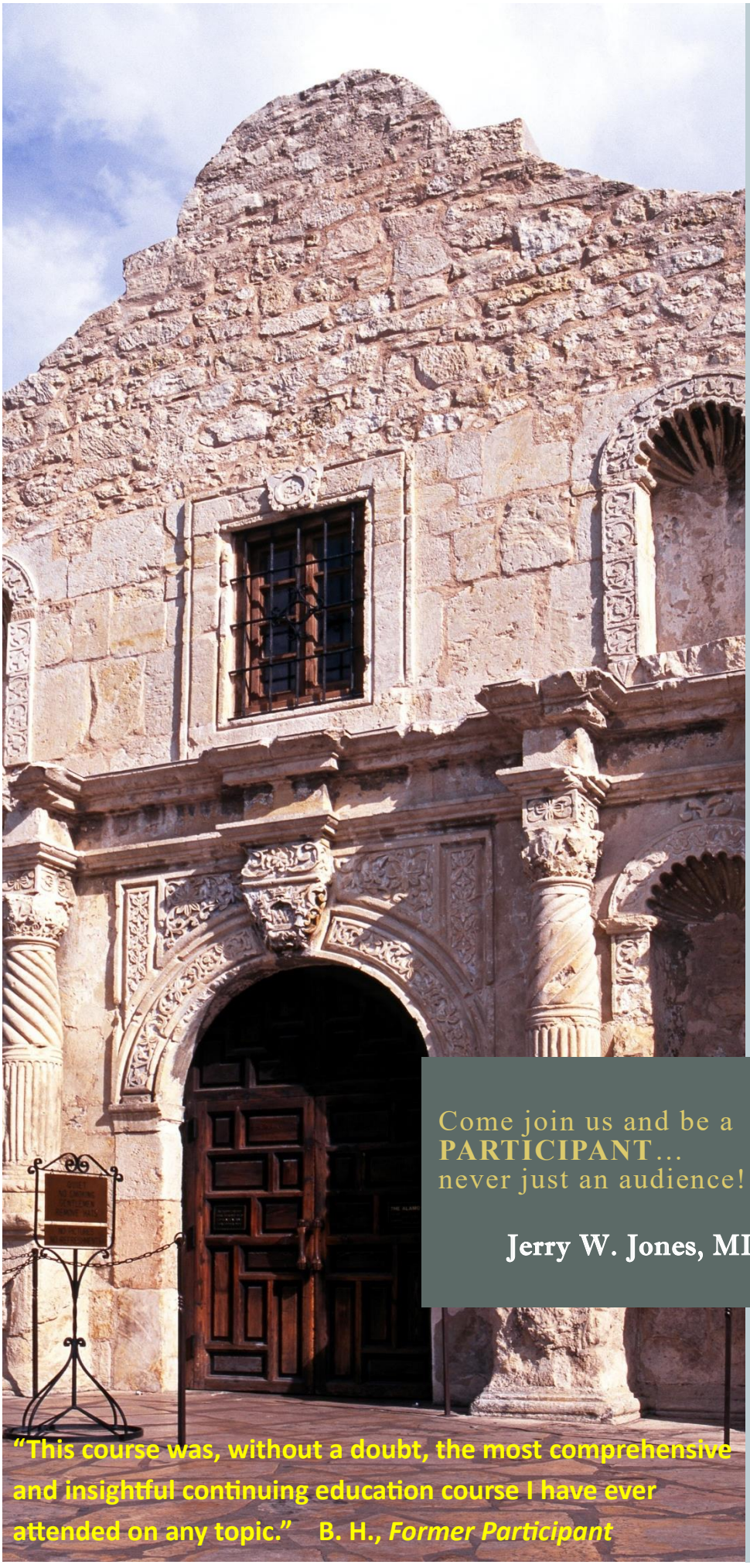

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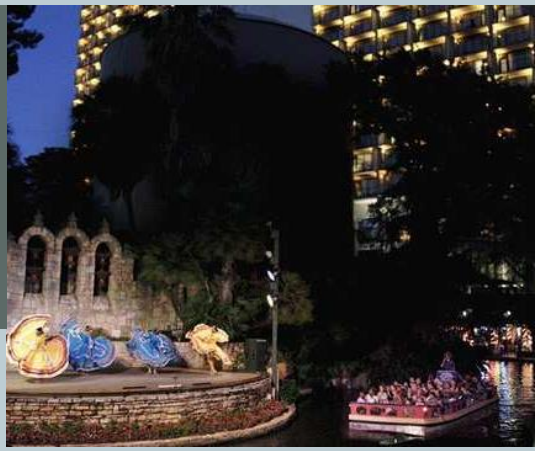

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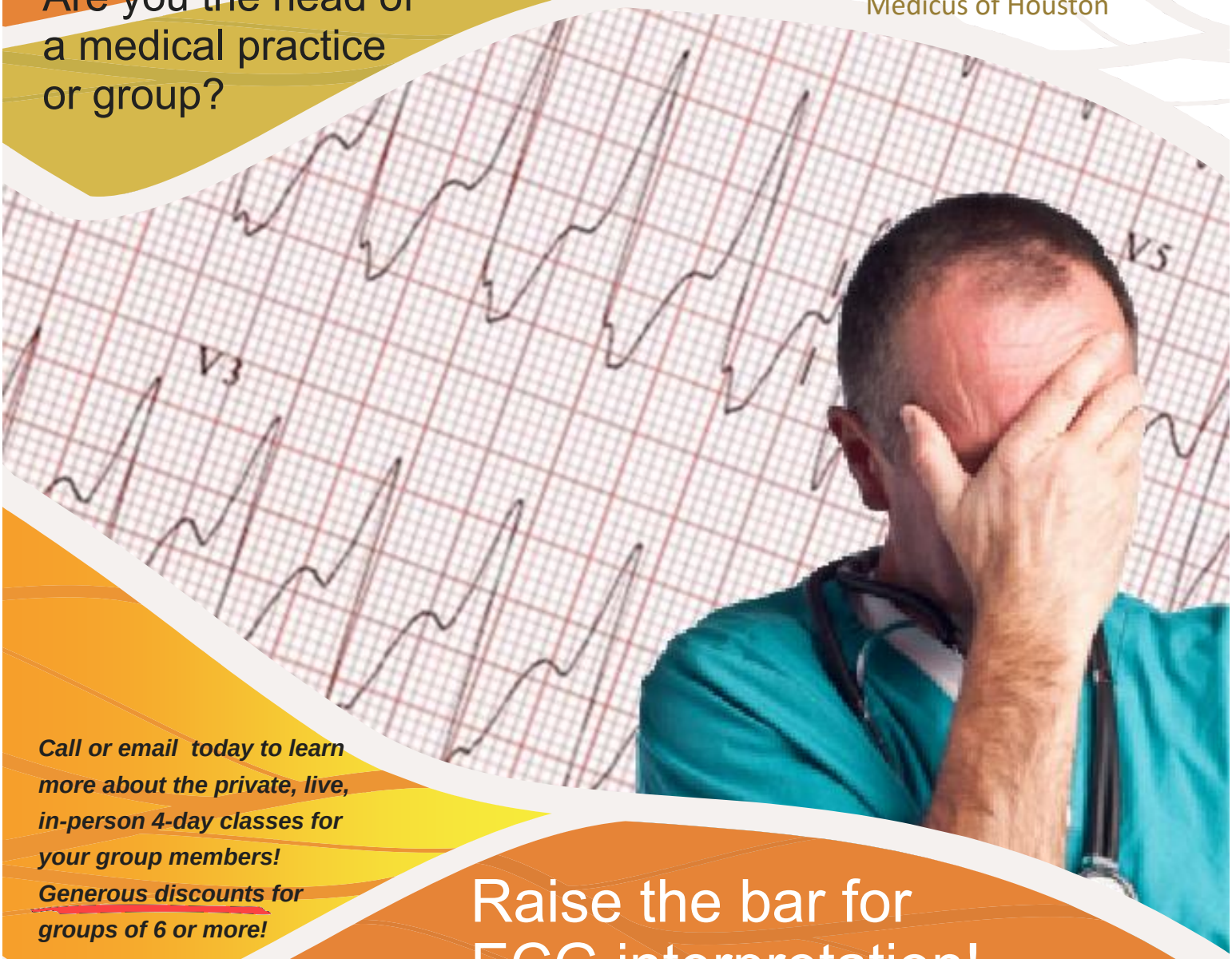

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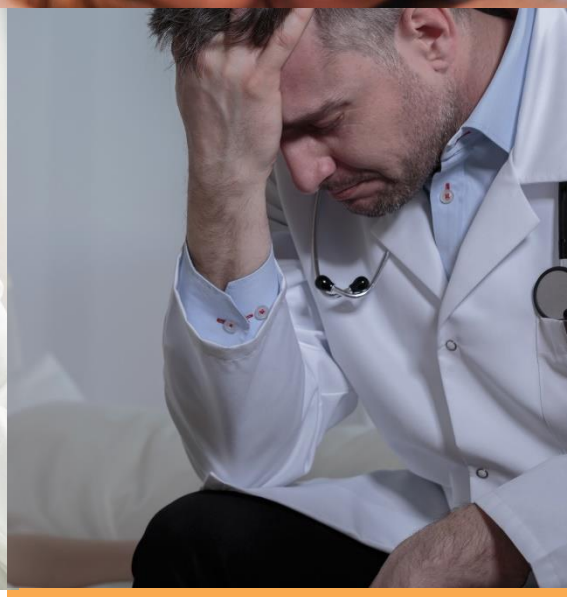
Is there something you're just not understanding?



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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