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Five tips to help keep you from making a big mistake!

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This is the second installment in the electrocardiogram (ECG) interpretation series by Jerry W. Jones MD FACEP FAAEM for this journal. In the previous installment, he discussed simple atrioventricular (AV) dissociation versus AV dissociation caused by third degree AV block (1); in this issue, he also shares some very important pearls regarding ECG interpretation.

Introduction

There are five things you should check before considering your interpretation of an electrocardiogram (ECG) complete (2). In my many years as an emergency department (ED) attending physician, I've been called in to assist with some of these unfortunate mistakes or else consulted to meet afterwards with physicians who made other unfortunate mistakes.

1. ST Elevation in Leads II, III and aVF, Elevated Troponin – It's an MI… Right?

It's always very gratifying to diagnose someone with an acute myocardial infarction (MI) quickly and then begin therapy early enough to save all or most of their myocardium. But don't let your good intentions and hard work lead you down the road to a disaster. The diagnosis of acute MI is arrived at by considering the patient's history, physical exam, ECG and cardiac enzymes. Positive responses or findings in all these processes leads to a quick and definitive diagnosis of acute MI. There's just one problem: similar findings may be present in patients who are suffering from two other conditions that may be even more lethal and for which the management is quite different for each.

When we make a diagnosis, we shouldn't stop there and consider our work finished. We should ask, "Why did this happen?" If you found that your hypertensive, diabetic, four pack a day smoker with chest pain has ST elevation in Leads II, III and aVF and a Troponin I of 26.4, you should not immediately conclude that he or she has an acute inferior MI (or only an acute inferior MI). You should ask yourself, "What else could cause this?" Too many physicians immediately conclude that the patient is experiencing a thrombotic occlusion of the right coronary artery (RCA) or left circumflex (LCx) artery and initiate a ST elevation myocardial infarction (STEMI) protocol. But before you do that, consider that there are two other conditions that can cause the same findings in patients with the same characteristics: aortic dissection and acute pulmonary embolus.

An ascending aortic dissection can present with an acute epicardial ischemia in the inferior leads due to occlusion of the RCA by an intimal flap rather than a ruptured plaque or thrombus. Cardiac markers may also be elevated. Before giving heparin, antiplatelet meds or thrombolytic, be certain that you are not dealing with an acute aortic dissection. In these cases, the dissection isn't just mimicking an infarction, it is causing an infarction in addition to its own damage.

An acute pulmonary embolus can also present with the same symptoms such as chest pain, dyspnea, weakness, and also demonstrate an elevation of the cardiac enzymes. The ECG may show ST elevation in Leads II, III and aVF, and sometimes in the right precordial leads (V1 – V3) as well.

Remember that ST elevation in leads associated with a vascular territory, a compatible history and elevated cardiac enzymes doesn't limit your differential diagnosis to just one condition.

2. Check Lead V1 for extra P waves

You have a 60-year-old female patient that appears to be experiencing a sinus tachycardia. You look at the Lead II rhythm strip and there is a P wave in front of every QRS and the rate appears to be around 110/minute and regular (Figure 1). The QRS complexes appear narrow and normal. The

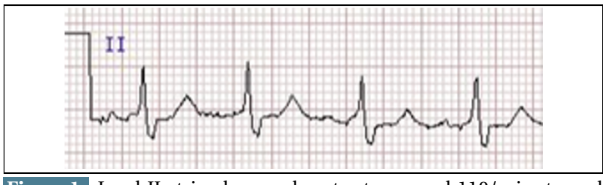

Figure 1 Lead II strip shows a heart rate around 110/minute and regular

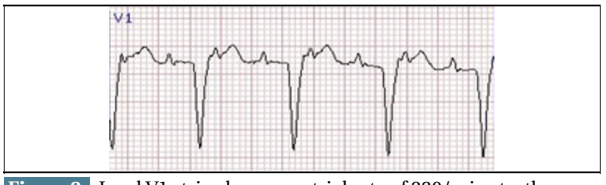

Figure 2 Lead V1 strip shows an atrial rate of 220/minute, the same ECG as in Figure 1

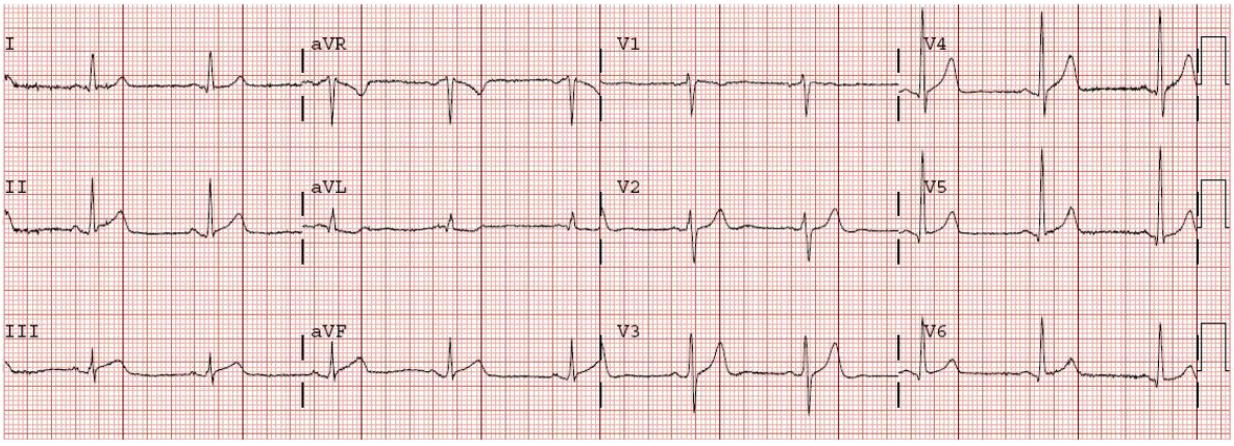

Figure 3 An ECG of a 56 year old male patient presenting to the ED with chest pain

patient has no fever and no other symptoms. Other than the increased heart rate, she feels relatively normal, though she does mention that it has felt faster at times causing her to feel light-headed. What is wrong here?

Well, what is "wrong" is that you may be looking at the "wrong" rhythm strip for a diagnosis. If you want to check for atrial enlargement, especially right atrial enlargement, Lead II is definitely the best lead to analyze. But if you are trying to diagnose a tachycardia, you should be looking in Lead V1. A look at Lead V1 may quickly reveal to you that the atrial rate is actually 220/minute and not 110 (Figure 2). This isn't sinus tachycardia! This is atrial tachycardia that probably conducts 1:1 occasionally, causing her palpitations and light-headedness.

Lead V1 in figure 2 is from the same ECG as in figure 1. As you can see, there are twice as many P waves. In any ECG with rhythm problems, always look in Lead V1 to assess the P waves and P-P interval. Don't ever try to diagnose a dysrhythmia (tachycardia, block, whatever) from just a single rhythm strip.

3. Is there ST depression in Leads III or aVL?

This is an introduction to Jones's Rule, which states:

Any ST depression on the 12-lead ECG of a patient complaining of chest pain consistent with an acute coronary syndrome is a reciprocal change to an acute MI until proved otherwise.

The question in the topical line ("Is there ST depression in Leads III or aVL?") leads us to the concept that ST depression due to subendocardial ischemia does not localize, while ST depression as a reciprocal change to an acute epicardial ischemia localizes quite well.

I have seen records of patients with acute onset chest pain who responded to nitroglycerine and the proverbial gastrointestinal (GI) cocktail sent home with a diagnosis of "mild, chronic subendocardial ischemia" or "angina pectoris"; and several of these cases had very bad outcomes later. This was because the ECG "just showed some ST depression in Lead aVL" and a troponin I, drawn less than four hours after the onset of the pain, was normal.

ST depression on a 12-lead ECG may be chronic… or it may be acute! Granted, some may be seen on serial ECGs, but one should never assume that ST depression is "chronic" in a patient with chest discomfort without the benefit of seeing one or more previous ECGs.

Figure 3 shows an ECG of a 56 year old male patient who presented to the ED with chest pain. This patient is having an acute inferior MI. There is ST depression in Lead aVL. Don't let the small size of the complexes in Lead aVL fool or distract you. They are small only because they are at 90° to the large deflections in Lead II. For the size of that QRS, that is a very significant ST depression. But, had the physician that discharged this patient looked a bit more closely, he or she may have noticed that the T waves in Leads II, III and aVF have very wide bases. Those are hyperacute T waves. No, they aren't tall at all (most are not tall), but all hyperacute T waves have very wide bases… all of them! Usually, hyperacute T waves are symmetrical, but they don't have to be symmetrical. As you can see, these hyperacute T waves are quite asymmetrical. Another thing to notice is that you don't see any ST segments in those leads anywhere on the baseline. Always be very suspicious when the bases of the T waves are so wide that there is no longer any baseline ST segment distinguishable. Think about the possibility of hyperacute T waves.

Another issue that may have led to the physician's misinterpretation of this ECG could have been due to the fact that the reciprocal change to an acute MI may appear *before* the ST elevation (or the classic form of ST elevation)! The basolateral leads (I, aVL) and the inferior leads (II, III, aVF) are the usual leads involved in this phenomenon. Since Leads III and aVL are the most opposing and reciprocal of the frontal plane leads, those are the leads you should scrutinize most carefully.

4. Looks like atrioventricular nodal reentry tachycardia (AVNRT). Give some adenosine and if that doesn't work, we'll give some verapamil.

A 45-year-old female patient has arrived with a rapid heart rate. Blood pressure is normal and she is alert. The nurse shows you the rhythm strip (Figure 4). Had a 12-lead ECG been recorded first, one would have seen this in Lead V1 (Figure 5).

That is a wide QRS tachycardia with a right bundle branch morphology. It is not a classic right bundle branch block morphology since it is a QR complex. Had this been a ventricular tachycardia (VT), adenosine would likely not have helped (although some rare VTs do respond to adenosine) and verapamil would have led to disastrous results. The point I want to make is that occasionally a portion of a QRS complex will have a vector that is perpendicular to a particular lead. The result is that part of the QRS complex will appear isoelectric making the QRS appear narrower than it actually is. This phenomenon is not unusual and you will encounter it from time to time (usually during normal rhythms, fortunately!). Mistakes can be avoided if you always remember to diagnose tachycardias and other dysrhythmias from a 12-lead ECG first.

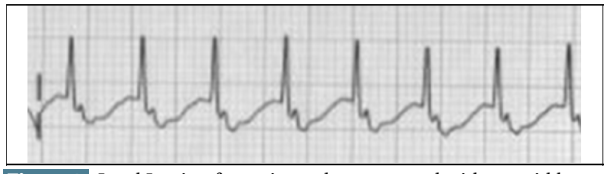

Figure 4 Lead I strip of a patient who presented with a rapid heart rate

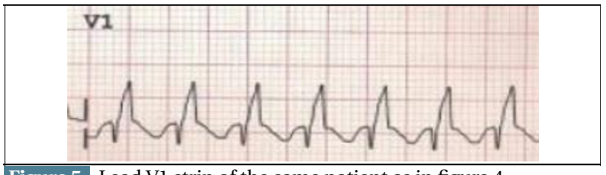

Figure 5 Lead V1 strip of the same patient as in figure 4

5. Check Lead aVL for subtle changes

Lead aVL is a very problematic lead and here's why: the positive pole for Lead aVL is located at -30° on the Hexaxial Reference Grid. That puts it exactly 90° from the Lead axis for Lead II, which has its positive pole located at +60°. Remember that when two lead axes are almost perpendicular to each other, their deflections will act inversely; that is, as a deflection gets larger in one lead, it will get proportionally smaller in the other lead. Here's the problem with Lead aVL: mean QRS axes (ÂQRS) in the frontal plane tend to cluster around +60°, which is the positive pole of Lead II. Therefore, since the QRS in Lead II tends to be relatively large most of the time, the QRS in Lead aVL will be small. And sometimes very small! That often makes it quite difficult to interpret – and especially at those times that are most critical. ST elevation and contour changes are often missed. Add to that the fact that ST depression in Lead aVL may appear before the ST elevation in Leads II, III and aVF (as happened in the ECG above). Or, ST elevation in Lead aVL may appear after the ST segment depression in the inferior leads. The ST depression of subendocardial ischemia usually involves a large number of leads that include more than one vascular territory. The ST depression of a reciprocal change to an acute MI typically involves only the leads that are found in a single vascular area, such as ST depression in Leads II, III and aVF as a reciprocal change to an acute basolateral MI, or ST depression in Leads I and aVL reciprocal to an acute inferior MI. Remembering Jones's Rule should help keep you out of trouble here.

6. Always compare Leads I and V6 before setting the ECG aside

Although the title of this article states "five tips," I'm going to give you a sixth. In New Orleans, that's called a "lagniappe" (pronounced "lan-yap" – a little something "extra"). The QRS complexes in Leads I and V6 should look very similar and should especially have the same polarity. If not, and this is an *unexpected* finding, then you have missed something! The most common cause is a lead wire switch, usually a left/right arm exchange. An anterior fascicular block may also be responsible, as well as advanced chronic obstructive pulmonary disease (COPD), remote anterior MI, left axis deviation, or a number of other causes.

References

1. Jones JW. Simple atrioventricular (AV) dissociation or AV dissociation caused by third degree AV block?. Front Emerg Med. 2022;6(1):e13.
2. Strauss, David G.; Schocken, Douglas, editors. Marriott's Practical Electrocardiography, 13th Edition. Lippincott Williams & Wilkins. 2020.