The Non-Dominant Right Coronary Artery Paradox

Jerry W. Jones, MD FACEP FAAEM

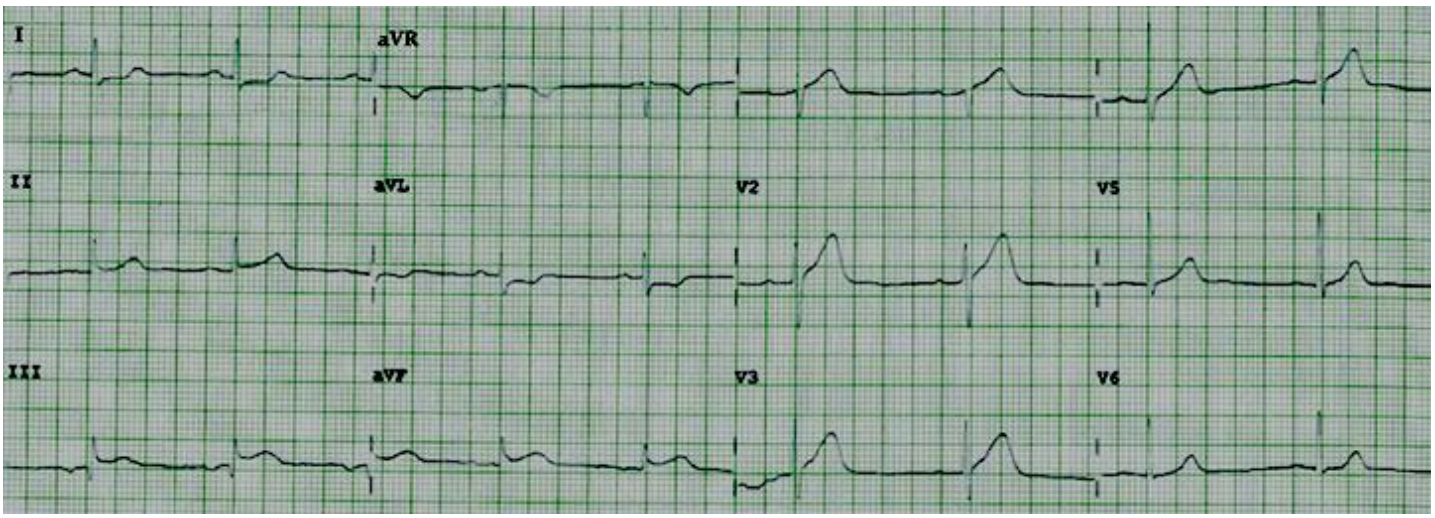


Figure 1

I ended my previous post with this ECG asking you if you see anything that shouldn't be there. It is a cath lab proven occlusion of a non-dominant RCA that resulted in an acute ischemia of the right ventricle. Proximal occlusions of the RCA are defined as occlusions occurring between the beginning of the artery and its marginal branch, inclusive. Since a non-dominant RCA ends with the marginal branch, all occlusions of a non-dominant RCA are – by definition – *proximal*.

In this ECG (Figure 1), we see very large hyperacute T waves from Lead V1 to Lead V4. The T wave in Lead V4 is visibly smaller than the T waves in Leads V1 – V3. The T waves in Leads V5 and V6 are essentially normal. This is certainly compatible with a right ventricular pattern and we even have ST elevation in Leads II, III and aVF with reciprocal changes in Leads I and aVL to support that pattern.

Wait a minute! **ST elevation in Leads II, III and aVF!?** How is that possible if the RCA is not providing the posterior descending artery? Does this mean there is a simultaneous occlusion in the LCx? That is the **PARADOX** of the non-dominant RCA!

There are many journal articles about occlusions of non-dominant right ventricular arteries available online. The interesting thing about these articles is that – while indicating that *all were diagnosed by catheterization* and even *displaying photos of the angiograms* – almost *all* cases demonstrate ST elevation in the inferior leads!

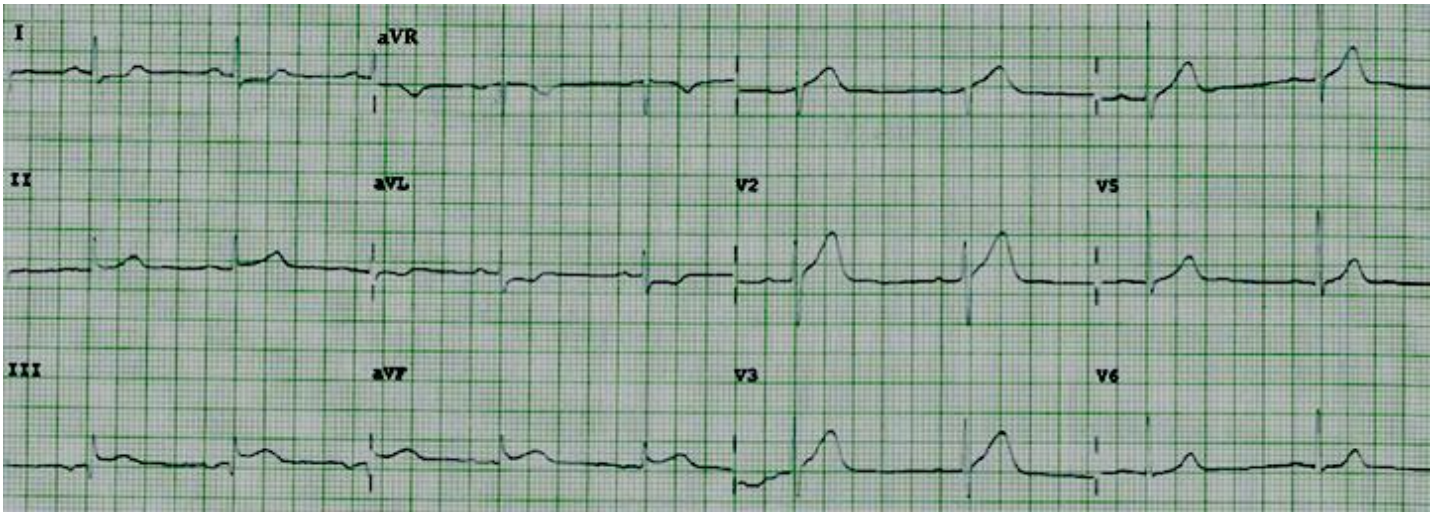


Figure 1 (repeated)

Yet, while some of the authors actually comment on the fact that STE is indeed *present* in the inferior leads, NONE suggest why! How is that possible?

Well, it IS possible and it has everything to do with the anatomy of the right coronary artery as well as the anatomy of the heart itself! Let's back up a bit and put this in historical perspective…

It has been shown only in the last few years that we have been using an *incorrect concept of the anatomy of the heart* in diagnosing acute myocardial infarctions. Of course, anatomists, pathologists and radiologists were well aware of the *true anatomy*, but we clinicians had simplified things so much we *began to believe our own simplifications*.

For many years we diagnosed ST depression in Leads V1 – V3 or V4 as a "posterior" MI when the acute ischemia was actually located on the inferolateral wall of the heart – that's *lateral*, not posterior.

So how does an RCA – *without a posterior descending artery* – result in an inferior wall STEMI? Understanding how that can happen requires you to understand a few things about the general anatomy of the heart – things that are very obvious but which we have given little or no thought while formulating our diagnoses. It also requires you to study accurate, anatomically-correct photos or illustrations of the heart and its coronary arteries. There are so many illustrations that are completely incorrect regarding the position of the heart in the chest as well as the locations of the coronary arteries in relation to the landmarks on the heart itself.

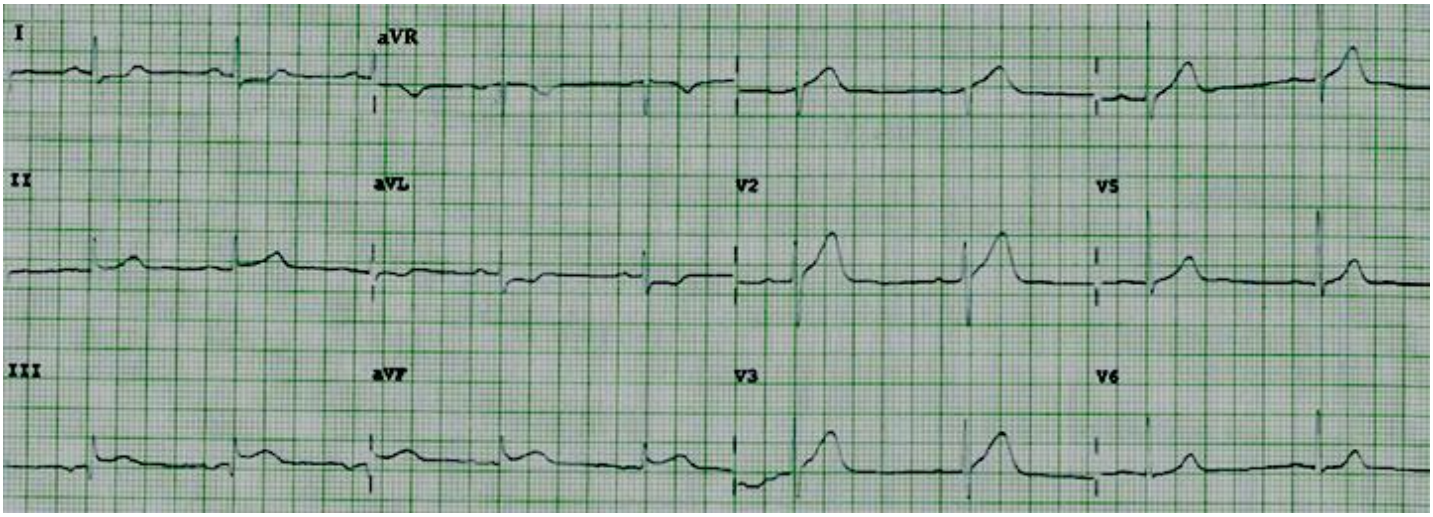


Figure 1 (repeated)

First, we really don't have a RIGHT ventricle or a LEFT ventricle. We have an ANTERIOR ventricle and a POSTERIOR ventricle. The two chambers are arranged more in an anterior/posterior alignment with only *slight* right/left orientation.

Second, the RIGHT ventricle can have its *own* anterior MI and inferior MI – unrelated to events in the left ventricle. And, just to make matters even more confusing, they can look very similar on the 12-lead ECG!

Let me give you a quick review of the branches of the RCA that are present when it is non-dominant…

The first branch off the RCA as it comes out of the right coronary cusp is the **conus branch**. This is a very important branch because it supplies the *right ventricular outflow tract* and it sometimes provides *collateral circulation to the proximal left anterior descending (LAD) artery*. This function is *extremely* important. If you review some 12-lead ECGs with acute myocardial infarctions that are due to *proximal* LAD occlusions, you will notice that some manifest ST elevation in Lead V1 and others do not. This is often due to whether a "long conus branch" is present or not – suggesting that the MIs with no S1 involvement occur in those hearts that are receiving collateral circulation from a "long" conus branch. Now, there are other reasons there may be no ST elevation in Lead V1, but the presence of a long conus branch can be life-saving. The conus branch at times may arise *directly from the aorta* making it a third coronary artery (if you consider the LAD and LCx as branches of the LMCA).

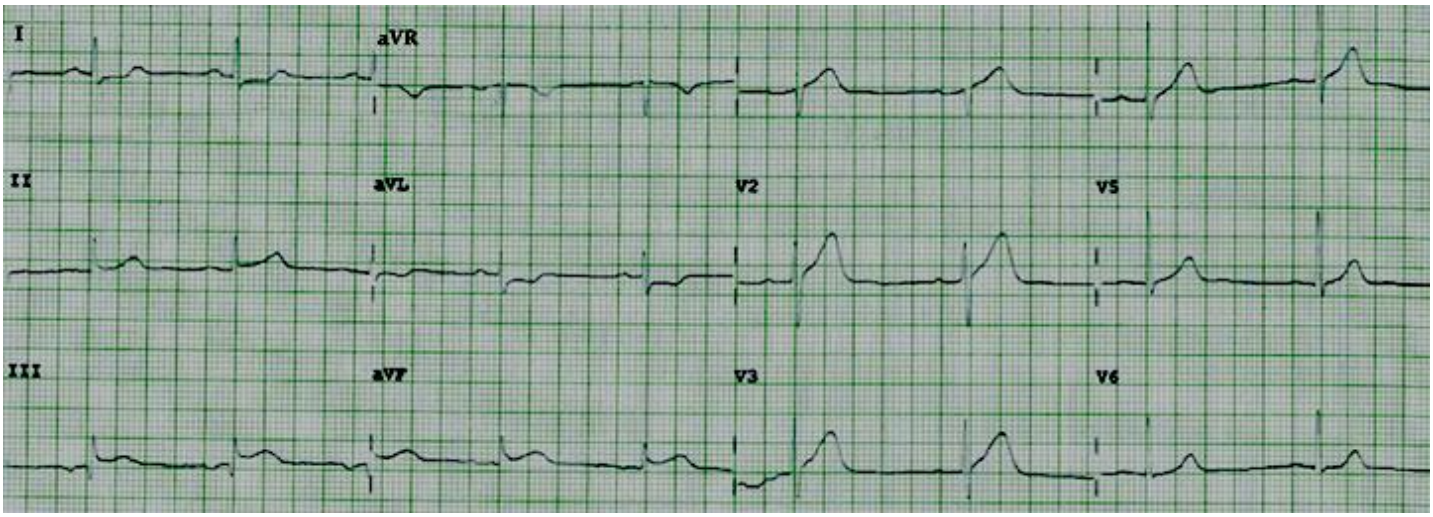
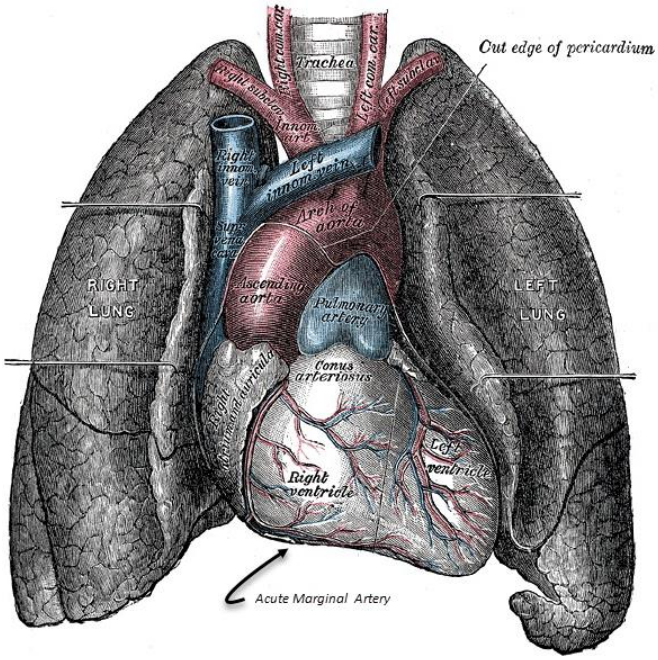


Figure 1 (repeated)

The next branch is the **artery to the right atrium**. The **artery to the SA node** branches off this artery. Obviously, it's very important. If an occlusion proximal to this branch occurs, the patient may suddenly experience *atrial fibrillation* (a *disaster* in the presence of a right ventricular infarction) or *SA blocks*, *pauses* or *arrest* (also *disastrous* during a right ventricular infarction), all of which contribute significantly to cardiovascular collapse.

And finally we have the branches to the right ventricle of which there are several. The **acute marginal artery** is the *largest* and usually *the last branch*. Now let's look at a very *accurate*, *anatomically-correct* diagram of the heart (Figure 2) as it is actually positioned in the chest and I think you will understand how the ST elevation in the inferior leads caused by occlusion of a non-dominant RCA occurs…



As you can see, the acute marginal artery runs along the *acute margin of the heart* which is located at the *junction of the anterior and diaphragmatic surfaces of the right ventricle*. This artery runs along the bottom of the free wall of the right ventricle. In this very anatomically-accurate illustration, you are looking directly at the free wall of the right ventricle.

Figure 2

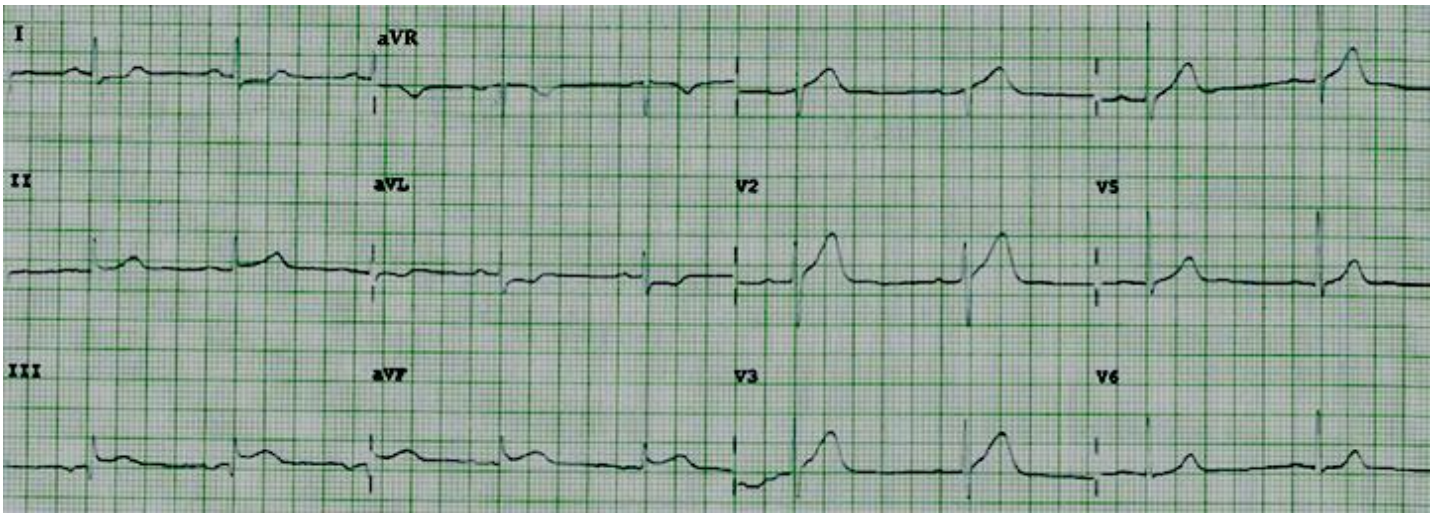


Figure 1 (repeated)

Many of you may have been laboring under the misconception that the free wall of the right ventricle is its lateral wall, just as the free wall of the left ventricle is mostly *its* lateral wall. But look at the illustration once more: the *right atrium is located lateral to the right ventricle*. When blood enters the right ventricle, it is traveling from right-to-left – *not* from top-to-bottom.

It is very important to have a realistic concept of the anatomy of the heart.

OK… I'll bet most of you have already begun to see how an occlusion of a non-dominant RCA can result in STE in Leads II, III and aVF.

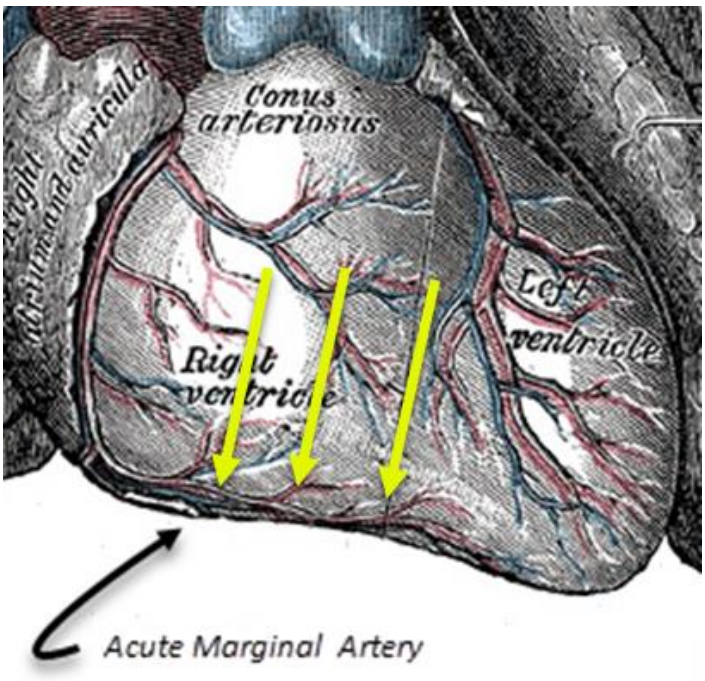


Figure 3

An occlusion of the non-dominant RCA will cut off circulation to the area perfused by the acute marginal artery. This, in turn, will result in ischemia of the antero*inferior* (*inferior* being the key word here) wall of the right ventricle. The ischemic vectors (yellow arrows) will point downward and are seen by Leads II, III and aVF as traveling *toward* them; thus, they will inscribe *ST elevation*. But this is ***ischemia of the bottom of the RIGHT ventricle*** – *not* the LEFT ventricle.

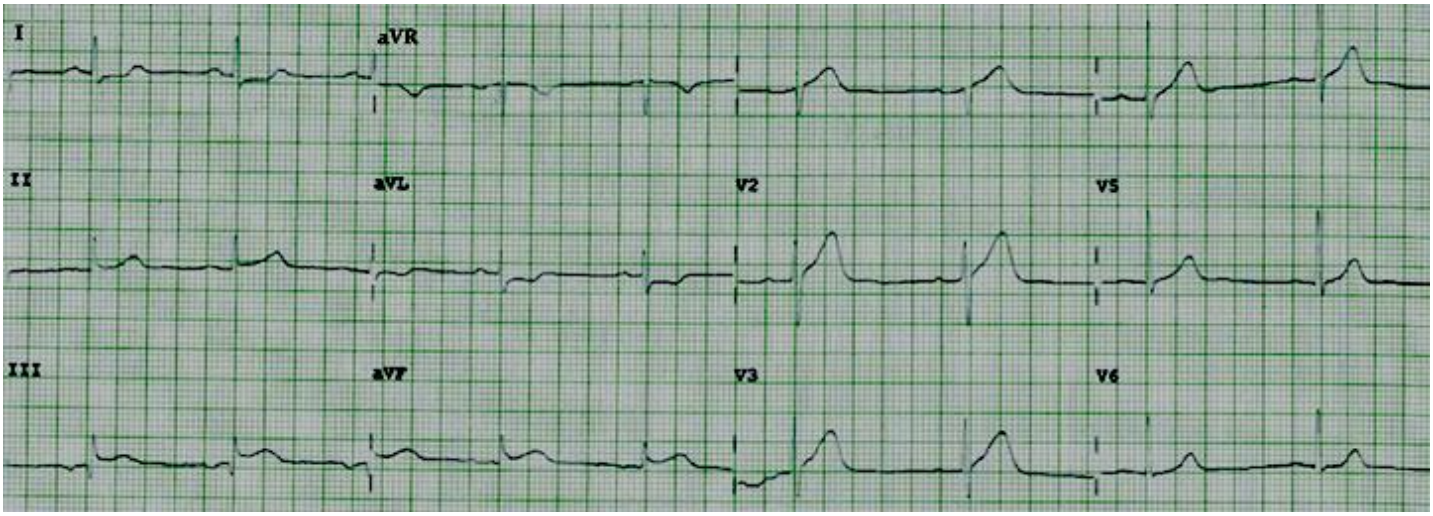


Figure 1 (repeated)

Leads I and aVL also inscribe *reciprocal changes* to the ST elevation in Leads II, III and aVF. Remember: the electrodes for each lead have no way of recognizing which chamber is producing the electrical voltage that it is recording. The electrode only knows the extent to which an impulse is traveling toward or away from it: either *directly* toward or away from it or *at an angle* toward or away from it.

OK… that explains why we may see STE in the inferior leads following the occlusion of a non-dominant RCA. But what about those occlusions of non-dominant RCAs that show NO inferior wall ischemia (i.e., ST elevation) on the ECG? Like this one…

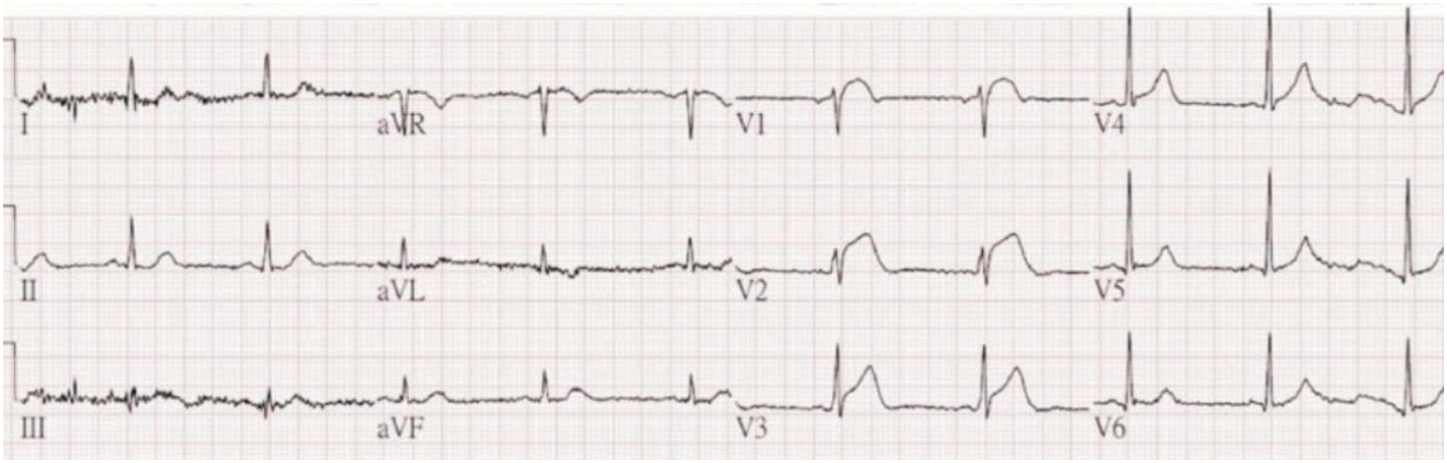


Figure 4

In the precordial leads, we see a classic example of a right ventricular infarction: STE begins in Lead V1 and becomes maximal in Lead V2. Then becomes minimal in Lead V4 and disappears in Lead V5. Again, a *classic pattern* for right ventricular infarction.

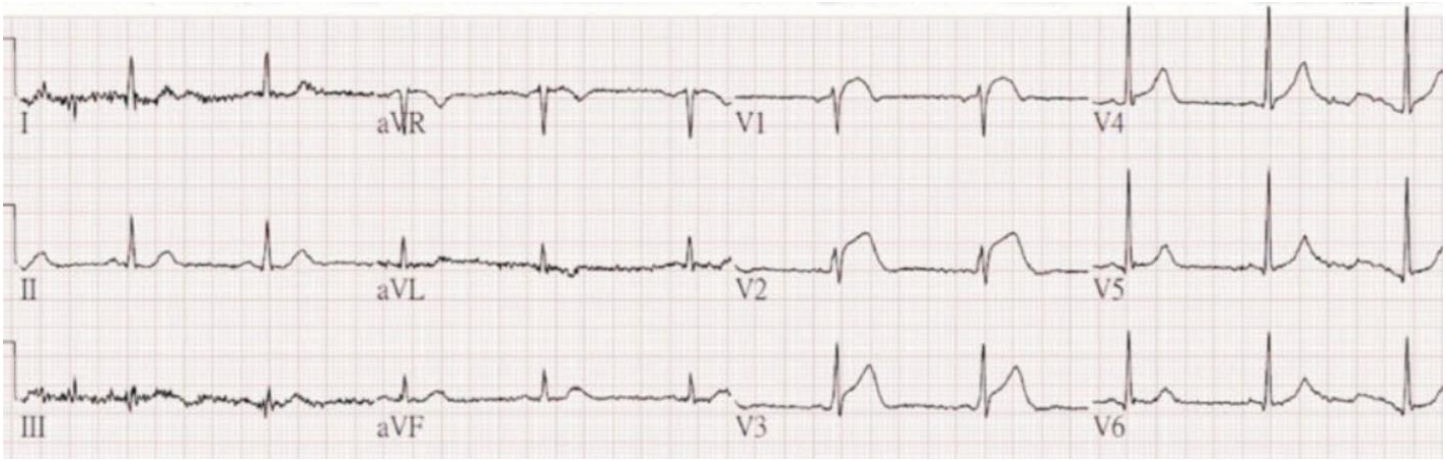


Figure 4 (repeated)

So why didn't the occlusion proximal to the acute marginal artery result in ST elevation in the inferior leads in *this* case? There are several possible reasons…

First, there may have been sufficient *collateral circulation* to that area to prevent anything more than minimal ischemia from developing. *Never forget or underestimate the presence or value of collateral circulation*.

Second, if the patient had been experiencing repeated episodes of angina, he or she may have developed sufficient *ischemic preconditioning* that helped prevent the development of acute ischemia when the occlusion finally occurred.

Third, there is a variation of the acute marginal artery in which it branches much more proximally from the RCA and then crosses the anterior wall of the right ventricle, traveling toward the apex of the heart. This anterior surface is perfused by other right ventricular branches from the RCA and also from the right diagonals from the left anterior descending artery which could greatly reduce the influence of an acutely occluded non-dominant RCA. If the occlusion is distal to this early take-off of the acute marginal artery, then there is not likely to be any acute anteroinferior ischemia.

As a reminder…

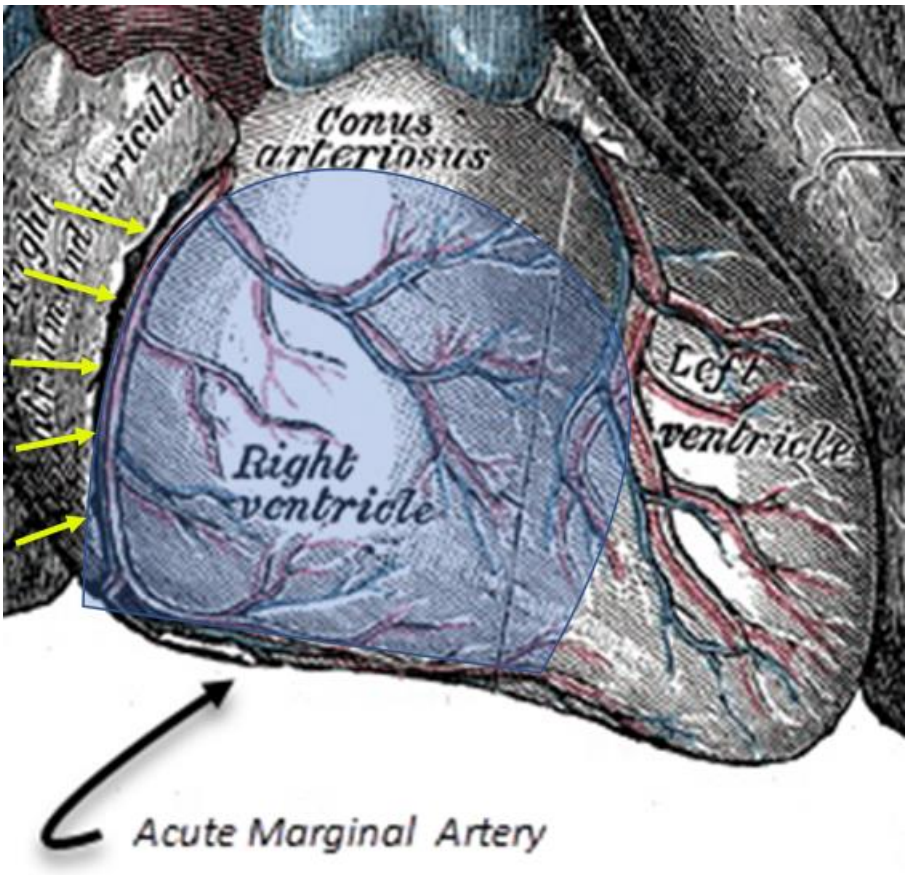
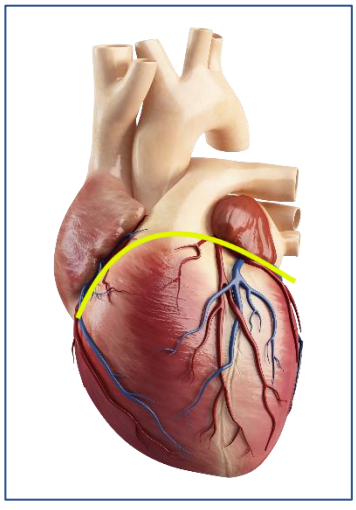


Figure 5

The blue shaded area approximates the free wall of the right ventricle. The curved black arrow indicates the *acute margin of the heart* being perfused by the *acute marginal artery of the RCA* which runs along its lower border. The small, yellow arrows indicate the ***atrioventricular groove*** – the route that the RCA takes from its origin in the right coronary cusp of the aorta. Most people think of the atrioventricular groove as curving around the heart, like the yellow line in Figure 6…



And because an occlusion involving the acute marginal artery will result in ischemia on the bottom of the free wall (Figure 5), ***the injury vectors will be pointing downwards toward the positive poles of Leads II, III and aVF***. Therefore, an occlusion of a non-dominant RCA *can* still result in ST *elevation* in the inferior leads ***even though it does not provide a posterior descending artery!***

Figure 6

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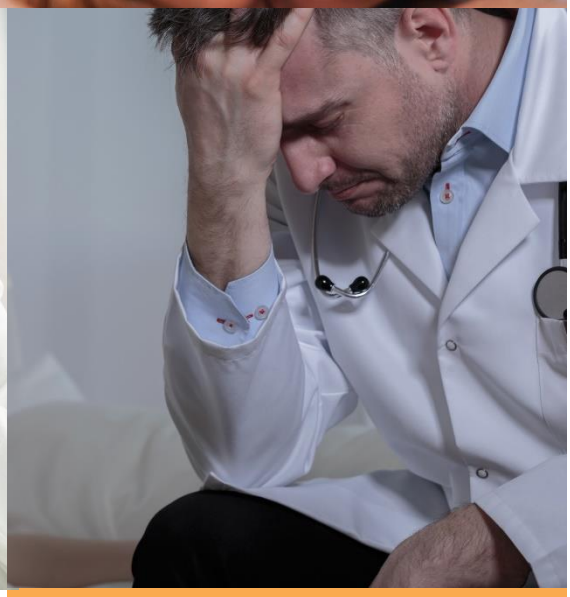

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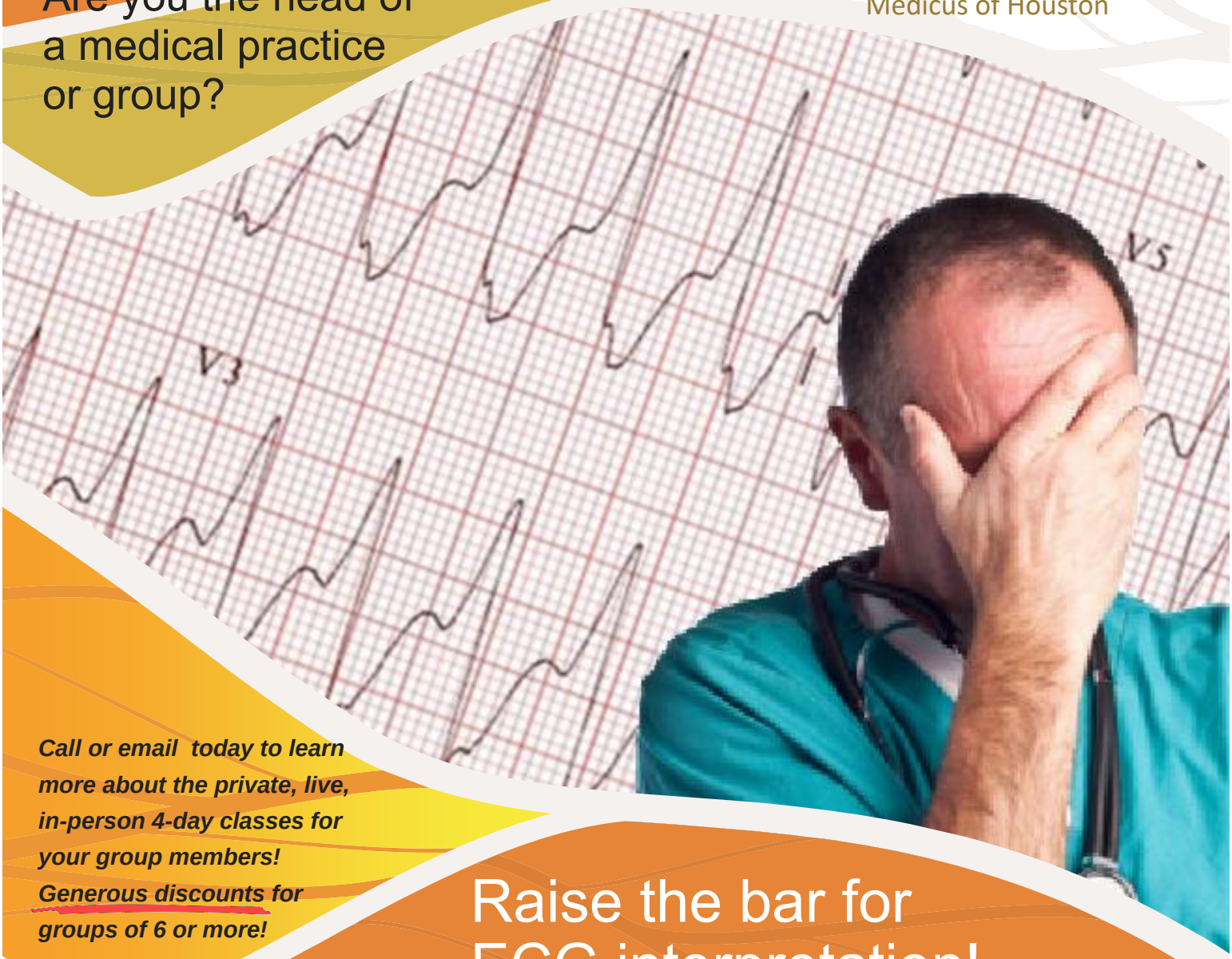

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