Let's Talk About Reciprocal Changes Discussion

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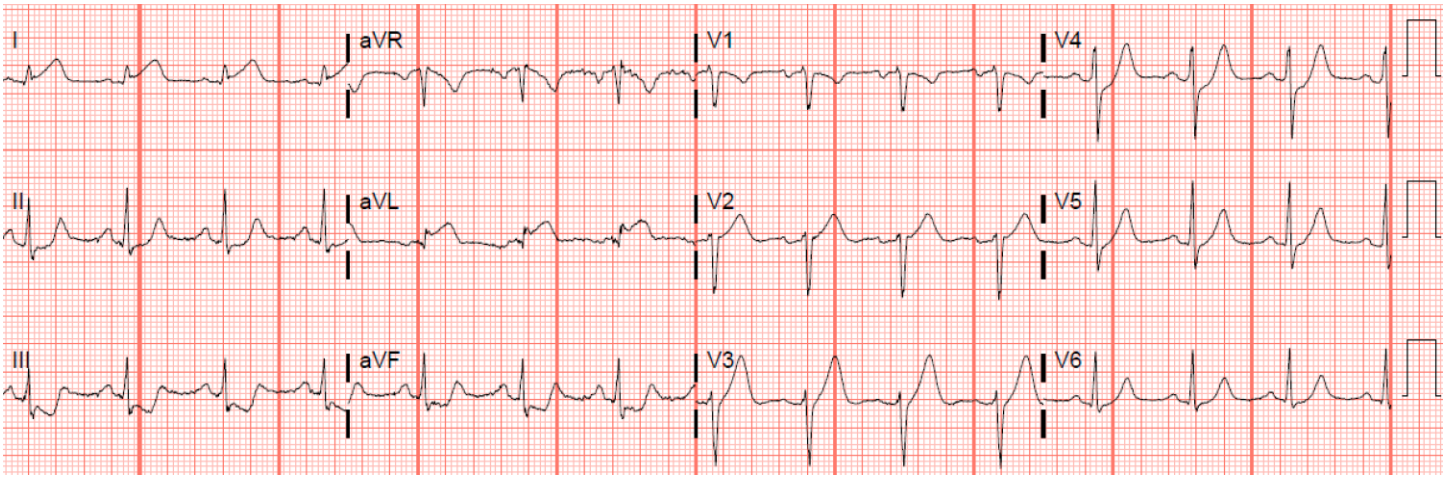

Figure 1

Do you know what **reciprocal changes** are? When there is ST *elevation* viewed by certain leads in a specific vascular territory, leads that are positioned opposite to those leads will view the same ischemia as ST *depression* – a mirror image. There is an *injury vector* that travels from normal tissue to ischemic tissue. That vector has a POSITIVE head and a NEGATIVE tail. The leads manifesting ST elevation see the positive head coming toward them and inscribe ST *elevation* while the opposing leads see the negative tail and record a negative deflection – in this case, ST *depression*.

On the ECG above (Figure 1) we see a *basolateral* (formerly *high-lateral*) ST elevation (Leads I and aVL) indicating a ***transmural ischemia***. There is **NO MYOCARDIAL INFARCTION** on this ECG! You should think of "MI" as **M**yocardial **I**schemia – not Myocardial Infarction – until the ST segments have come back to baseline and pathological Q waves have appeared in the leads that manifested ST elevation.

The inferior leads (II, III and aVF) have ST depression – not elevation. That's because they are located *somewhat* opposite Leads I and aVL. No leads in the frontal plane are truly 180° apart. If you move 180° from Lead I, you're right back on Lead I! The furthest two leads can be in the frontal plane is 150°.

Now think about this: leads that are 90° from each other are essentially *invisible to each other* and absolutely cannot act reciprocally. OK, so what is Lead I's relation to the inferior

leads? Well, Lead I and Lead aVF are 90° from each other so they are invisible to each other. Lead I isn't causing the ST depression in Lead aVF. Lead I is only 60° from Lead II, which places Lead II in its positive hemifield. Lead I certainly isn't causing any reciprocal change in Lead II. Lead III is 120° from Lead I so YES – Lead III can act reciprocally to Lead I.

Lead aVL is perpendicular to Lead II, so Lead II cannot act reciprocally to Lead aVL since they are invisible to each other. Lead II is 150° from the positive pole of Lead aVR – but there is no ST elevation in Lead aVR. Most likely Lead II is acting reciprocally to ischemic myocardium that is not covered by a recording electrode. Lead aVF is 120° away from Lead aVL so, yes, it can react reciprocally to the ST elevation In Lead aVL and Lead III is 150° so Leads III and aVL can certainly react reciprocally.

Now which coronary artery is occluded: the RCA, the LCx or the LAD? Let's use the **Jones Method of Diagnostic Elimination** and get to the diagnosis quickly.

An occlusion of the RCA *cannot* produce ST elevation in Leads I or aVL. Therefore, the RCA is ruled out.

Occlusions of either the LAD or the LCx *can* produce ST elevation in Leads I and aVL, but if you look in Leads V2 – V4 you will see the *hyperacute T waves* indicating the ischemic wave rapidly approaching the epicardium in those areas. The LCx *cannot* produce ischemic changes in Leads V2 and V3, so an occlusion of the LCx is ruled out.

The LAD is our culprit vessel.

Sometimes it is easier to decide what could NOT have happened than to calculate all the likely possibilities. That is the basis of the **Jones Method of Diagnostic Elimination**.

Want to learn more about ECG interpretation? Come join us in one of my Masterclasses! If this was too far over your head, then enroll in the ***Introduction to Electrocardiography for ABSOLUTE BEGINNERS!*** It lasts **2.5 days**, is completely **online**, and **interactive**. You can still ask questions at any time. Check my website for dates:

https://medicusofhouston.com

At the top of the home page is an announcement about the live webinar I am presenting on June 28th. It is **FREE** and discusses occlusions of the left circumflex artery (LCx) in depth. This is the artery in which up to 48% of its occlusions do not show up as ST elevation! ***It's really worth learning about!***