What Are Reciprocal Changes?

A reciprocal change usually refers to repolarization changes in acute ischemia being viewed from opposite perspectives. The pitcher in a baseball game sees the ball travelling away from him while the catcher sees the same ball – the same pitch – traveling toward him. Although those are opposite directions – both players are correct in their own perspective.

When the left arm electrode sees an impulse traveling toward it, the right arm electrode sees the same impulse traveling away from it. So when there is ST elevation for instance in Lead V8 or V9, there will be ST depression in Lead V2. Because these poles are opposite each other, they see the same repolarization abnormality from opposite perspectives. A reciprocal ST depression does not mean there is any ischemia in that area.

The world of electrocardiography is divided into two camps: those that believe reciprocal changes must occur within the same plane and those that feel that a plane that is perpendicular to the affected lead can display reciprocal changes. I am a firm believer that reciprocal changes must occur within the same plane. In other words ST elevation in the horizontal plane leads (V6R – V9) does not create ST depression in the frontal leads (I, II, III, aVR, aVL, aVF). Only leads that are directly (or almost directly) opposite each other in the same plane can manifest reciprocal changes. The axes of the various leads only detect those vectors that are traveling in straight lines – they do not make right-angled turns!

“But wait!” you exclaim. “I have seen acute anterior MIs with reciprocal ST depression in the inferior leads!” I’m sure you have. So have I! But those reciprocal changes in leads II, III and aVF were not reciprocal to elevations in V2, V3, V4, V5 and V6. They were reciprocal to the ST elevations in Leads I and aVL. Leads I and aVL have a good view of much of the area supplied by the first diagonal branch of the left anterior descending artery and the obtuse marginal branch of the left circumflex artery. Both leads are well-positioned to detect the acute epicardial ischemia that occurs in those areas. Thus, they will manifest ST elevation. But those leads are actually in the frontal plane, so when they manifest acute ST elevation, the inferior leads – and especially Lead III – will display reciprocal changes in the opposite direction, i.e., ST depression. Leads aVL and III are the leads in which we most commonly see reciprocal changes. In fact, in computing Lead III, the electrocardiograph basically uses the calculated voltage of the left arm electrode (which is the positive pole for Lead aVL) as the negative pole for Lead III.

“Well then,” you say. “How about acute inferior ischemia with ST elevation in Leads II, III and aVF and reciprocal ST depression in Leads V1 through V3?” OK, OK. You’ve got me there. (Just kidding!) That is not an unusual situation. There can indeed be ST elevation in Leads II, III and aVF indicating acute ischemia of the inferior wall of the left ventricle with reciprocal changes in precordial leads V1 – V3 (and sometimes even V4). But that ST depression isn’t reciprocal to the elevation in the inferior leads – it’s reciprocal to the acute ischemia that is occurring beneath the posterior leads V7 – V9 – if any were done!

And here’s a bit more information. In the case of acute high anterolateral ischemia with ST elevation, the reciprocal changes in the inferior leads – II, III and aVF – may be evident well before the ST elevation in Leads I and aVL. If you happen to see it at that time, it can look like the inferior leads are responding reciprocally to ST deviations in Leads V2 – V6. They aren’t. The same thing can happen the other way around. Acute epicardial ischemia in the inferior leads may manifest first by the reciprocal changes in Leads I and aVL – especially aVL. If a patient presents with credible* chest pain and shows only some ST depression in perhaps Leads III and aVF – and nothing else – you had better be very suspicious of an occlusion in the LCx or proximal LAD. And likewise, a patient with credible chest pain and just a little bit of ST depression in Lead aVL has just profoundly increased his/her likelihood of having an acute inferior wall ischemia.

There is another area where reciprocal changes occur. This happens when there is ST elevation in V1. Leads V5 and V6 are opposite enough to display reciprocal changes at times (and they are all in the same plane!). This is not seen as frequently as the other cases, but it certainly does occur.
An Asterisk and an Afterthought...

credible chest pain – This has nothing to do with believing the patient. A credible chest pain is one that is characteristic of pain caused by myocardial ischemia (centrally located, pressure-like or squeezing in quality, often precipitated by exertion or sudden exposure to cold temperatures and lasting at least a minute and often longer than that if no nitroglycerin is taken). A non-credible chest pain would be one that is located eccentrically in the chest with a sharp, stabbing or needle-like quality and lasting just a few seconds. It usually occurs with either no provocation or during emotional upset and can often be precipitated by local pressure on the chest wall.

Here’s a history-taking pearl for you to remember when seeing a patient complaining of chest pain: for ischemic chest pain (angina pectoris) to occur, something must happen to suddenly restrict the supply of oxygenated blood to an area of the heart, that decrease or loss of oxygenated blood must have a few moments to have its effect, the problem causing the angina must then be resolved and then the coronary vasculature must have time to restore the circulation to that area and then the heart must have time to react to the restoration of the circulation. I don’t care how credible the description of chest pain sounds; if it lasts only a few seconds there isn’t enough time for all that to occur! Here’s another pearl to help you have more confidence in determining the credibility of a patient’s chest pain. Whenever you see a patient who suffers from angina from time to time – ask him/her how long the pain lasts from start to finish. And don’t ask just one patient – ask every time you see such a patient! It won’t be long before you will have a very confident “feel” for when a patient is describing real angina pectoris.