Systolic and Diastolic Currents of Injury

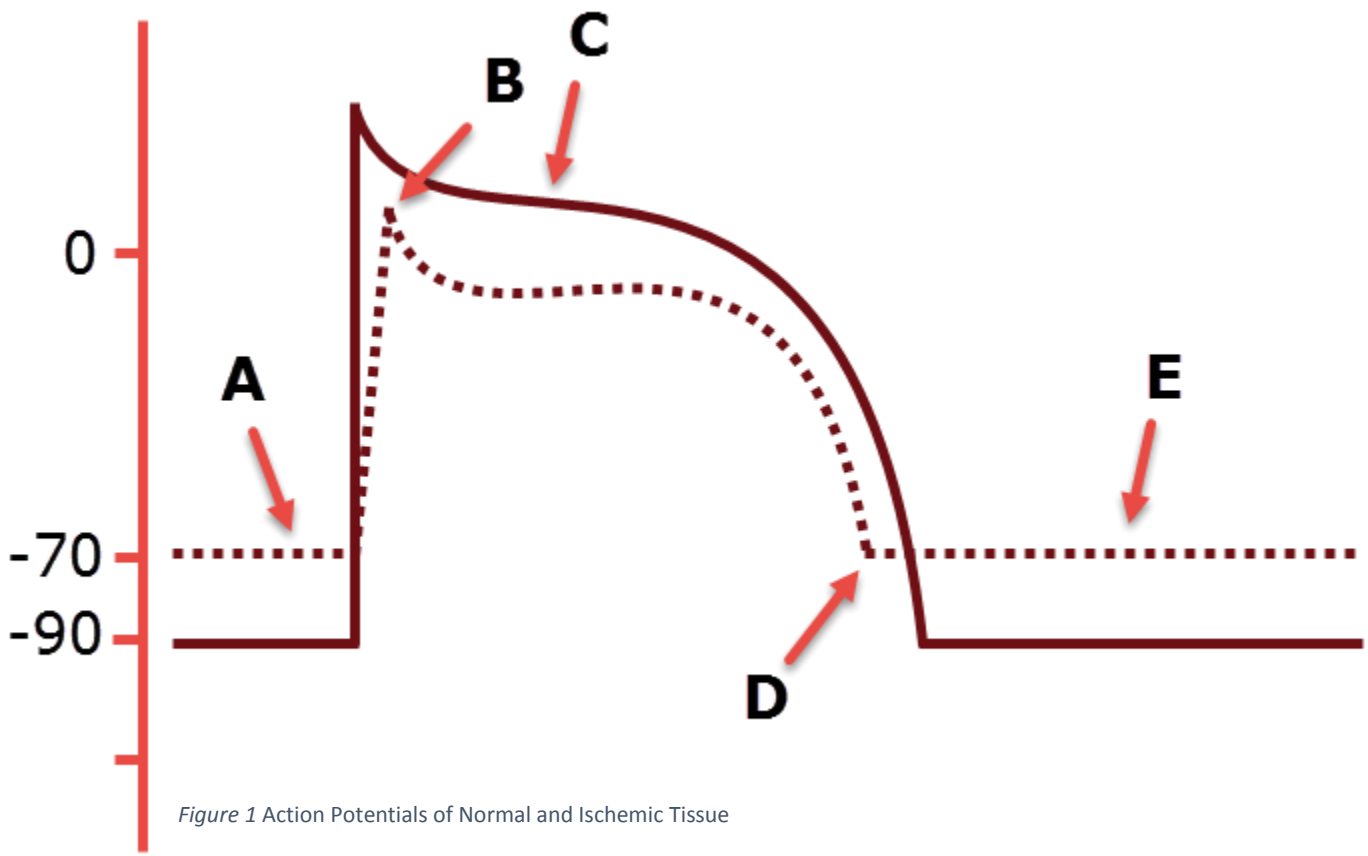


Figure 1 Action Potentials of Normal and Ischemic Tissue

In Figure 1 above, the action potential of *normal* myocardium is represented by the solid lines and the action potential of the *ischemic* myocardial area is represented by the dotted lines.

There are some basic concepts that must be remembered at all times during the discussion of the theories of systolic and diastolic currents of injury:

1. Localized ischemia always damages some cells. Loss of circulation to an area of the myocardium results in cellular hypoxia. The hypoxia leads to diminished levels of ATP in the affected cells. There are ionic transport systems in the cell membrane that depend on ATP for energy and the **Na^+ / K^+ pump** is one of them. Loss of this pump prevents K^+ from being pumped back into the cell during diastole, so the concentration of K+ builds up outside the cell while the intracellular concentration of K+ falls. The concentrations of K+ inside and outside the cell are less different, so the resting transmembrane potential is reduced.

CAUTION!

Everyone is familiar with the vertical scale that runs from -90 mV through 0 to around +30 mV. We have a tendency to think that a value increases as it moves up the scale and decreases as it moves down the scale – ***but it's just the opposite!*** -70 mV is less than -90 mV. The transmembrane potential increases when the difference between the internal and external charges increases. So the closer the resting membrane potential is to 0, the less it is.

2. The damage caused by ischemia is localized – it is not reflected in the organism as a whole, but just in the area affected by the ischemia.

3. The collection of K^+ in the extracellular space locally causes the extracellular concentration of K^+ to rise resulting in less difference between the intracellular and extracellular concentrations. A reduction in the membrane potential difference is the same as depolarization (the interior of the cell is becoming *less negative*), so *localized myocardial ischemia* results in *localized partial depolarization*.

4. Thus, the resting membrane potential of ischemic myocardium is less negative than the resting membrane potential of normal myocardium. (**Arrow "A"**) (***Resting membrane potential = Phase 4***)

5. The partial depolarization caused by ischemia is not rapid enough to trigger an actual depolarization wave. It does, however, cause many Na^+ channels to open momentarily and then close. So, when the depolarization wave eventually arrives, it finds fewer Na^+ channels available to open. Consequently, the action potential is both slowed (decreased slope) and has less amplitude (Phase 0 does not rise as far). (**Arrow "B"**)

6. Because the amplitude of Phase 0 is reduced in cells affected by ischemia, Phase 2 must therefore occur at a lower voltage (Phase 2 can never be higher than the peak of Phase 0). The Phase 2 maximum voltage in ischemic myocardium is significantly less than that of Phase 2 in the normal tissue. And notice that Phase 2 of the *normal* action potential is POSITIVE (above "0") during most of systole. (**Arrow "C"**)

7. When we speak of vectors, we are talking about the direction (as well as magnitude) of depolarization or repolarization. We use the internal charge as our reference point. However, when we are speaking of an injury current, we are referring to the charge on the exterior of the cell. If a cell is depolarized, the interior will be positively charged and the exterior will carry a negative charge; if the cell has repolarized, the interior of the cell will be negatively-charged and the exterior of the cell will carry a positive charge.

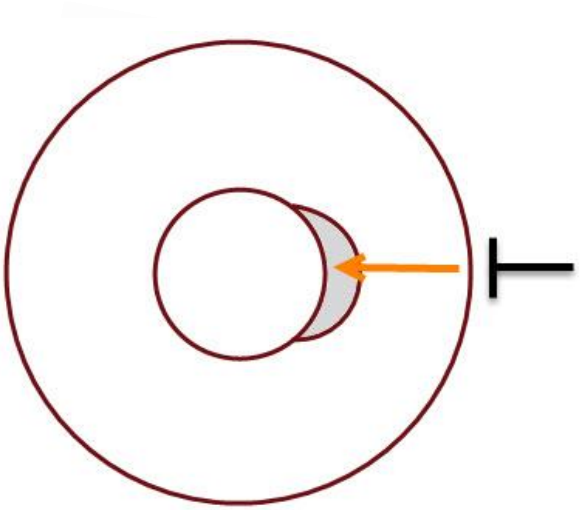
8. Current of injury in biological systems always flows from negative to positive, or negative to "less negative."

Now let's talk about the *theories of systolic and diastolic currents of injury*. Bear in mind – these are still *theories* and neither has been completely validated. During my reading, I have been impressed that more and more physiologists and electrophysiologists feel that there is truth in both theories.

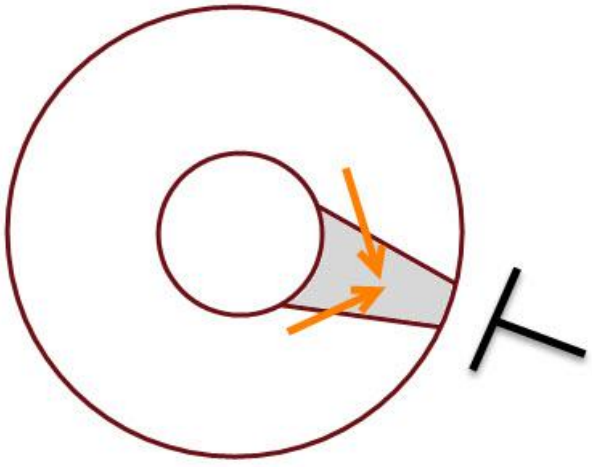
Systolic Current of Injury Theory

PERSPECTIVE: Bear in mind that the **Systolic Current of Injury Theory** attempts to explain **ST elevation or depression** by looking at the *ST segment itself* on the *action potential* – **Phase 2**.

During Phase 2 (the ST segment), the normal cells are more *positively-charged internally* than the ischemic cells (Figure 1 above where the solid line is above the dotted line) and thus have a *negative external charge*. On the other hand, the ischemic cells are unable to depolarize as completely as the normal cells, so their *internal charge is negative* relative to the normal myocardium and the *external charge is positive*. Since current travels from "negative" to "positive" or "*negative* to *less negative*," an *injury current* appears that runs from the normal tissue to the ischemic tissue *during systole*. Because Phase 2 is represented on the ECG tracing as the ST segment, whether the ST segment is elevated or depressed depends on the location and thickness (or depth) of the ischemic area.



Just picture this in your mind: there is an area of subendocardial ischemia. What will the positive electrode overlying this area see? Remember: ***during systole the normal myocardium is more positive internally and more negative externally than the ischemic myocardium and current will flow from negative to positive (or "less negative")***. So, the **systolic current of injury** will run from the normal tissue to the ischemic tissue. In our particular example, the ischemic tissue is subendocardium and the normal tissue is subepicardium. Therefore, the positive electrode overlying the area will see a current during Phase 2 (the ST segment) *traveling away from the body surface* (epicardium to endocardium) and will therefore register ST *depression*.

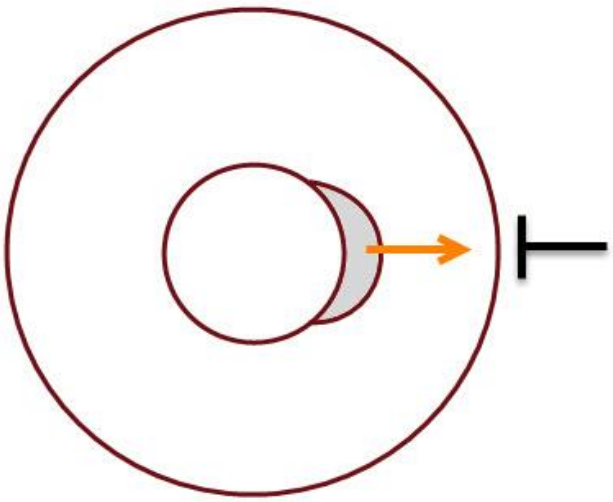


But what happens when the ischemia is *transmural*? Again, the current will travel from negative to positive (or *less negative*). Since – *during systole* – the normal tissue is more positively-charged internally and negatively-charged on the exterior surface than the ischemic tissue, the current will travel from normal myocardium (with the *negative external charge*) to ischemic myocardium (with a *positive external charge*). The resultant vector will be outward and the ECG will record ST *elevation*.

Diastolic Current of Injury Theory

PERSPECTIVE: The **Diastolic Current of Injury Theory** attempts to explain **ST elevation or depression** not by examining the ST segments themselves but by looking at the *baseline of the ECG – the T-P segment* (represented on the action potential by **Phase 4**).

Diastole is Phase 4 of the action potential. In this case, the Phase 4 of the ischemic cells have more positive internal charge (and thus a negatively-charged exterior) than the normal cells during Phase 4 (**Arrow E**). Therefore, the current in this case will travel from the ischemic myocardium to the normal myocardium. A very important point here is that we are talking about the effect that ischemia will have on **Phase 4**. Remember: Phase 4 represents the T-P segment. ***No one ever looks at the T-P segment when they are looking for ischemia or infarction! They always look at the ST segment!*** And for good reason: the ECG machine is programmed to keep the T-P segment at 0 mV with everything else on the strip moved up or down relative to that. If the T-P segment is depressed (below 0 mV) the ECG machine will raise it back up to 0 mV and concomitantly raise the rest of the tracing an equal amount. Thus instead of seeing actual T-P depression, you will think you are seeing ST elevation.



Let's look at the example of subendocardial ischemia being recorded during Phase 4 diastole (the T-P segment). Because Phase 4 of the ischemic myocardium is partially depolarized and therefore more positive interiorly and negative externally than the normal myocardium, the current will travel from ischemic myocardium to normal myocardium. In the case of subendocardial ischemia, this results in an injury current traveling toward the recording electrode during diastole resulting in an *elevation* of the T-P segment. However, the ECG machine brings the baseline T-P segment back down to 0 mV and so the ST segment is now even lower and appears *depressed*.

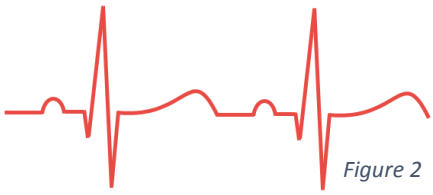


Figure 2 Normal ECG Tracing

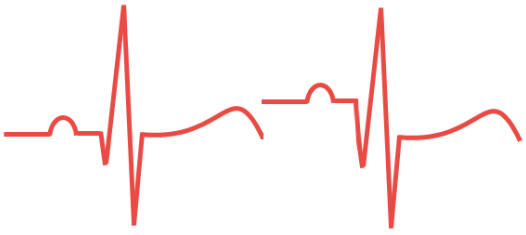


Figure 4 T-P Elevation Due To Ischemia

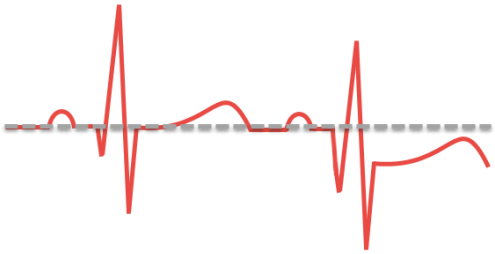
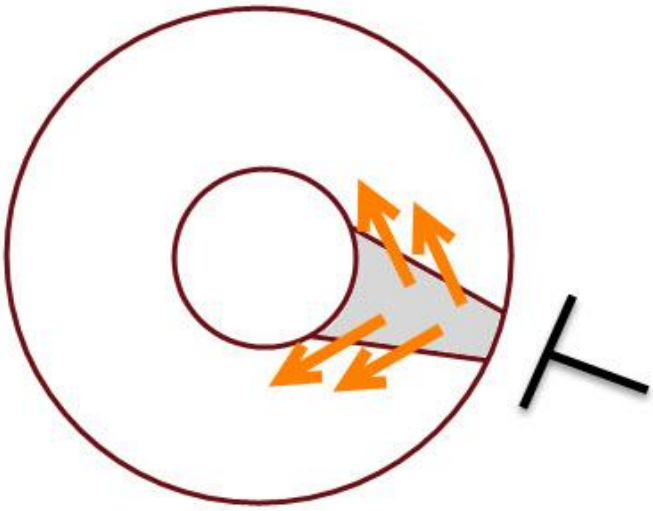


Figure 3 "Illusion" of ST Depression After the Baseline Is "Corrected" to 0 mV By the ECG Machine



Let's now look at an example of a *transmural infarction* or an infarction that has managed to extend from the epicardial layer at least to the *electrical subendocardium*. Again, we are speaking only of events that are occurring during diastole – Phase 4, the T-P segment. Because of the difference in membrane potential between the ischemic and normal tissues – ischemic, depolarized with a *positively-charged interior* and a *negatively-charged exterior* – a current is generated that runs from the ischemic tissue to the normal myocardium. The recording electrode will see only vectors traveling away from it, so it will inscribe a negative T-P segment. Again, the electrocardiograph will adjust the baseline T-P segment to 0 mV which raises everything on the ECG. Because we assume the baseline is always at 0 mV, what we thus see on the tracing is ST elevation.

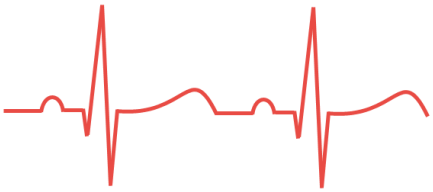


Figure 6 Normal ECG Tracing

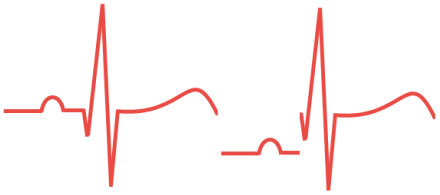


Figure 5 T-P Depression Due To Ischemia

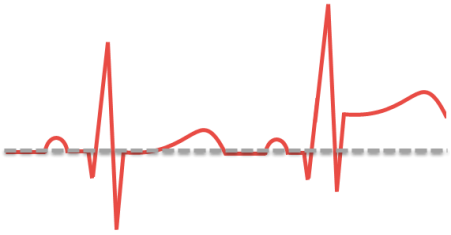


Figure 7 "Illusion" of ST Elevation After the Baseline Is "Corrected" to 0 mV By the ECG Machine

Some Points to Remember:

1. What we are trying to explain with these two theories is the *cause of ST elevation or depression with acute myocardial ischemia*. Remember: with the **Diastolic Current of Injury Theory**, even though we are examining and measuring the *T-P segment*, we are really trying to explain the actions of the *ST segment!*

2. The **Systolic Current of Injury Theory** looks at the relative voltages of the normal and ischemic myocardium during systole – which occurs primarily during repolarization. It is actually looking at the ST segment and the effects of the currents of injury on it.

3. The **Diastolic Current of Injury Theory** looks at the relative voltages of the normal and ischemic myocardium during Phase 4 diastole – which is represented by the T-P segment. By convention, the T-P segment is considered the baseline and the baseline is assumed to be at 0 mV. Because any deviation of the T-P segment (baseline) is automatically adjusted back to 0 mV by the ECG machine, *we can't see any effects on the T-P segment on the ECG. Whether it is elevated or depressed, the machine will always adjust it up or down to 0 mV*. But what we *can* see are the changes in the ST segment. The ST segment is not really involved in the **Diastolic Current of Injury Theory** except that if the T-P segment is depressed, its adjustment back to 0 mV will cause the ST segment to be raised up – giving the *illusion* of ST elevation. Likewise, if the T-P segment is elevated, its adjustment by the ECG machine back down to 0 mV will give the *impression* that the ST segment is depressed.