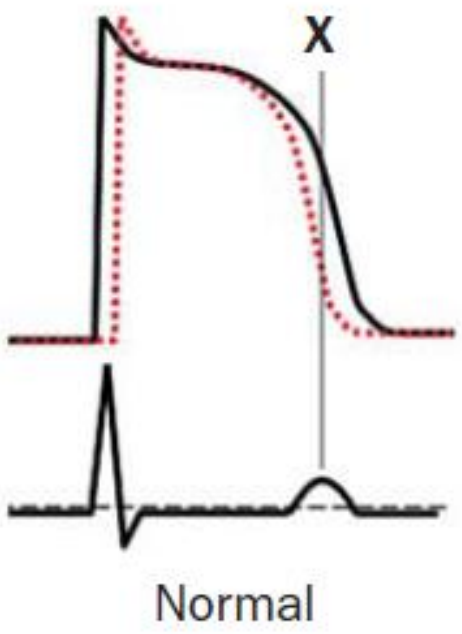
Ischemic Pseudonormalization of T Waves



LEFT: *Normal action potentials of the subendocardial (solid line) and subepicardial (dotted line) layers of a working myocyte.*

Here, we are only concerned with repolarization. Under normal circumstances, repolarization begins in the subepicardium and travels to the subendocardium.

As we can see on the diagram on the left, subepicardial depolarization actually begins after subendocardial depolarization. Therefore, Phase 0 of the subepicardium occurs slightly later.

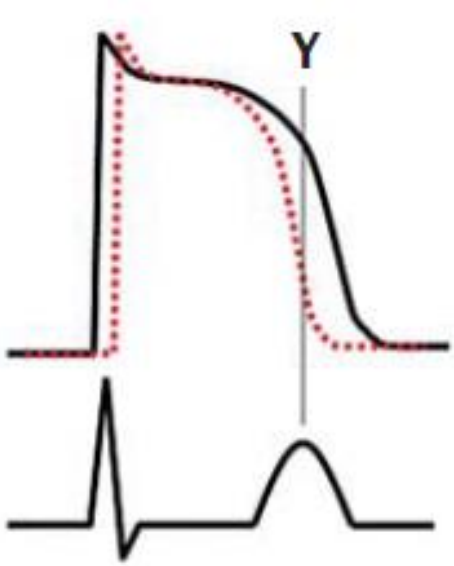
Because there is a delay in the onset of repolarization of the subendocardium, the subepicardium begins repolarization first. Therefore, Phase 3 begins earlier in the subepicardium. Although the action potential (and consequently the QT interval) is shortened in the subepicardium, it is not shortened over all. The action potential duration (and consequently the QT interval) is determined by the portion of the myocardium that finishes repolarization last.

When repolarization occurs, the interior of the cell becomes – once again - negatively charged. That means the exterior of the cell becomes positively charged. So here is the situation at point "X" in our diagram: at the same moment in time (point "X"), the subendocardium designated by the solid line is still depolarized, with a positive intracellular charge and a negative extracellular charge. However, the epicardium is already repolarized with a negative intracellular charge and a positive extracellular charge. The difference in charges of the contiguous tissue causes a current to flow. **When we discuss current flowing from one area to another in living tissue, we are discussing the charge on the outside of the cell membrane – not the interior charge. By an arbitrary definition, current flows from areas that are negatively charged (externally) to areas that are positively charged (externally).**

Because the subendocardium is still depolarized, that means the external charge is *negative*, so the current will flow from the subendocardium to the already-repolarized subepicardium where the external charge is now *positive*. A recording positive electrode will see a current traveling toward it – from subendocardium to subepicardium – and will inscribe a positive (upright) T wave in that lead.

CAUTION!

Don't confuse CURRENT with VECTOR. **The current appears during Phase 4 while there is essentially no vectors due to no net ion exchange.** Vectors are ***based on the interior charge*** and indicate the *direction* of depolarization or repolarization. The direction of the current flow is ***based on the exterior charge***. ***Because current generated in living tissue is defined as flowing from negative to positive, current flowing toward a positive electrode will cause a positive deflection to be inscribed in that lead***. We generally only concern ourselves with currents during **Phase 4** (electrical diastole, T-P segment) – not during depolarization or repolarization. The "deflection" that a current (typically an *injury* current) produces is the ST segment, a "positive" deflection being ST elevation and a "negative" deflection being ST depression.



LEFT: *Hyperacute epicardial ischemic action potential (subendocardium - solid line and subepicardium - dotted line) of a working myocyte.*

(Hyper)Acute subepicardial ischemia causes a shortening of the action potential of the cells in the ischemic area. This means that the subepicardial cells repolarize sooner, i.e., their interior becomes negatively charged and their exterior becomes positively charged. The subendocardial cells at the same point in time are still negatively charged externally. Thus, a current will begin to flow from the negative exterior charges on the subendocardial cells to the positive external charges on the subepicardial cells. A positive electrode over this area will record a repolarizing current traveling toward it and will thus inscribe a positive – or upright – T wave.

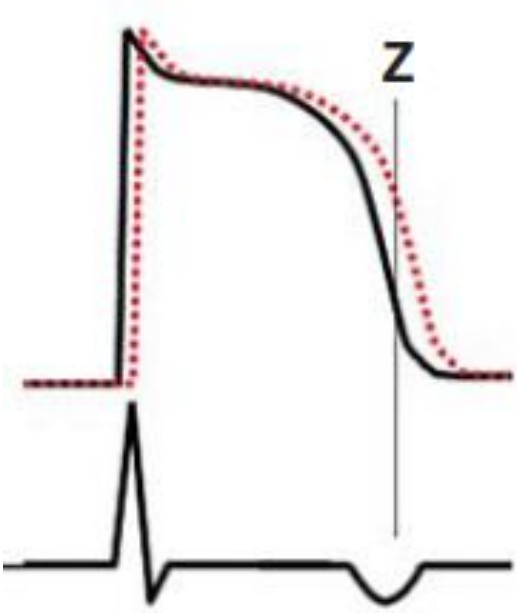
CAUTION!

Once again, we must avoid a misunderstanding regarding ischemic tissue and its effect on the QT (QTc) interval. When myocardium becomes acutely ischemic, the action potentials of the ischemic cells shorten, as seen in the diagram on the left. This is fact and has been documented many, many times. However, there are a number of scholarly articles – as well as the direct observations of thousands of very competent electrocardiographers – that state the QT interval is prolonged during acute ischemia, including STEMI and NSTEMI. This, too, is fact and has been documented many, many times. There seems to be a conflict here – but there really isn't.

The issue arises in that the QT interval is the full duration of the action potential. But if you look at the diagram, you can clearly see that while the duration of the ischemic action potential has decreased, the duration of the total action potential has not. The ECG machine will record the longest action potential of all regions of the heart. The action potential duration in the ischemic area will indeed shorten, but it isn't going to be discernable on the 12-lead ECG. Other areas of the myocardium will prevail. And the shortened action potential duration of the ischemic area does not necessarily remain shortened.

If you look at the above diagram, you will see that there is ***a considerable voltage difference between the subendocardial cells and the subepicardial cells*** and that ***this difference lasts a longer period of time***. Thus, the T waves at this point are going to be ***larger and wider – hyperacute T waves***. And since repolarization will still be travelling from subepicardium to subendocardium, they will also be upright.

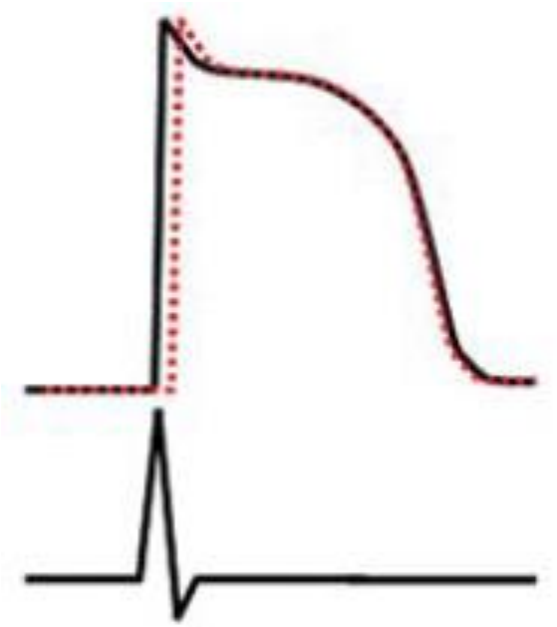
[**Erratum:** In an earlier document, I had inadvertently switched "subepicardium" and "subendocardium" in the last sentence of the paragraph above. After noting the error, I have now changed it. The sentence is now correct and the statement reads as I originally intended. – JWJMD]



LEFT: *Chronic (post-reperfusion) epicardial ischemic action potential (subendocardium - solid line and subepicardium - dotted line) of a working myocyte.*

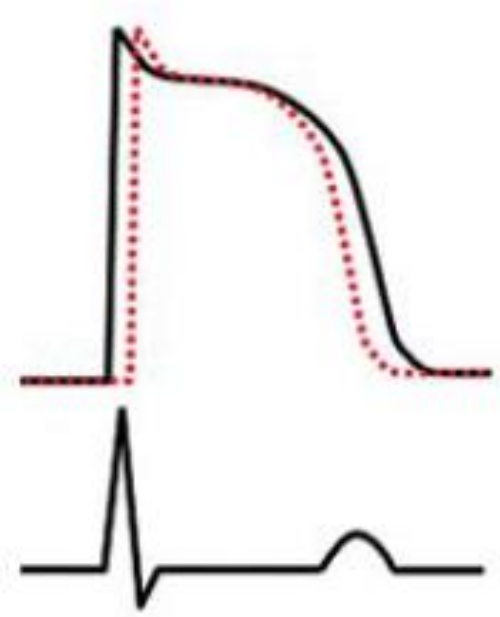
After reperfusion, T waves will invert. As can be seen in the diagram on the left, the ischemic subepicardium is now slower to repolarize. Thus, there is a negative charge on the exterior of the ischemic subepicardial cells after the subendocardial cells have begun to repolarize. A current is consequently established that runs from the subepicardium to the subendocardium. Because this current is traveling away from the positive electrode overlying this area, a negative deflection (inverted T wave) is inscribed. This is exactly what we would *expect* to happen following an episode of acute epicardial/subepicardial ischemia. This is how a reperfusion T wave is formed.

But sometimes, following reperfusion or a partial reperfusion, the patient again has another acute ischemic episode. What will happen to the action potential in the subepicardial cells during the acute ischemia? The action potential duration will *shorten*! Let's see what happens when the action potentials in that area shorten after the reperfusion T waves have appeared:



OK. The action potential of the cells in the subepicardial layer have shortened. Remember: they were *prolonged* following reperfusion. Now they have shortened to the point that the action potential duration in the ischemic subepicardium and in the subendocardium is the same. Also, we can see that Phase 3 in both action potentials overlie each other so that there is no electrical potential difference between them. Therefore we have an isoelectric T wave (and ST segment). An isoelectric T wave following reperfusion is *not* normal!

But let's say that these two action potentials were drawn while the ischemia was relatively mild and that now it has increased significantly:



Now we can see that the action potential duration in the ischemic subepicardium has decreased even more. Phase 3 in the two different action potentials (subepicardium and subendocardium) are no longer overlying each other but now demonstrate a significant electrical potential difference. Repolarization has begun in the subepicardium *before* the subendocardium. Once again, the cells of the subepicardium are *positively*-charged externally and the subendocardial cells – at the same moment in time – have *negative* external charges. Thus a current begins to flow from the subendocardium to the subepicardium and this is manifested on the surface 12-lead ECG as a positive (upright) T wave.

That is how Pseudonormalization of the T wave occurs.

Remember: Following successful reperfusion, the T waves should remain inverted from 1 – 3 weeks. Any T wave normalization before then should be carefully considered and ***any T wave normalization before 7 days should be considered evidence of new ischemia until proved otherwise***.

The action potential duration (and, by inference, the QT interval) of the cells in an acutely ischemic area of myocardium will always shorten. However, this is not evident on the surface 12-lead ECG because of the longer duration of action potentials in other areas and layers of the myocardium. Though it doesn't affect the QT (or QTc) interval, this shortening of the action potential will, however, affect the shape of repolarization on the ECG.