Origin of ‘ischemic' ECG changes’

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Many years ago I was asked to write a topic for “UpToDate” explaining the origin of ‘ischemic' ECG changes’ during exercise ECG testing. After reviewing the literature at the time (1990’s) I designed the two figures shown below using a simple 2 cell model, one from the endocardium and one from the epicardium, correlating the action potentials (APs) to a hypothetical ECG obtained from lead V5 on the body surface. Although the model is way oversimplified, I think it helps our understanding of ST segment depression (STD) and ST elevation (STE) seen during exercise testing and also during acute myocardial infarction. In the normal V5 ECG (Figure 1) the relatively flat ST segment on the baseline reflects the lack of a potential difference between the 2 cells during this phase of repolarization. The upright T wave is the result of the shorter AP duration in the epicardial cell.

In Figure 2, I consider the V5 ECG during subendocardial ischemia (injury) and during transmural ischemia (injury). In subendocardial ischemia 3 things happen to the subendocardial cell: 1) loss of resting membrane potential (diastolic injury) resulting in TQ segment elevation on the ECG; 2) reduced voltage during the plateau phase resulting in true ST segment depression (systolic injury), and shortening the AP duration resulting in T wave inversion. The TQ segment elevation is corrected by modern ECG electronics further depressing the ST (‘pseudo’ ST depression). In transmural ischemia (injury) the V5 ECG is changed in a reciprocal manner because the V5 ECG lead is now looking directly at injured cells behind which are normal non-ischemic cells. So, from the perspective of the 2-cell model true and ‘pseudo’ ST segment elevation and hyperacute T wave change are the reflection of systolic and diastolic currents of injury. (This simple model also explains the ST elevation seen in acute pericarditis where the epicardial cell is injured behind which are normal endocardial cells).

Therefore, with regards to acute STEMI myocardial infarctions, the finding of STD in a particular lead of the 12- or 15-lead ECG might truly be

a ‘reciprocal’ change 180 degrees away from the leads showing STE, or might reflect additional subendocardial ischemia in regions adjacent to the transmural injury.

In real life ECG interpretation of suspected acute ischemic events we assume 1) the ECG precordial electrodes are properly placed (often not so in a busy ER) and 2) the limb leads reflect an perfect equilateral triangle (clearly not so). But we clearly strive to do the best we can.

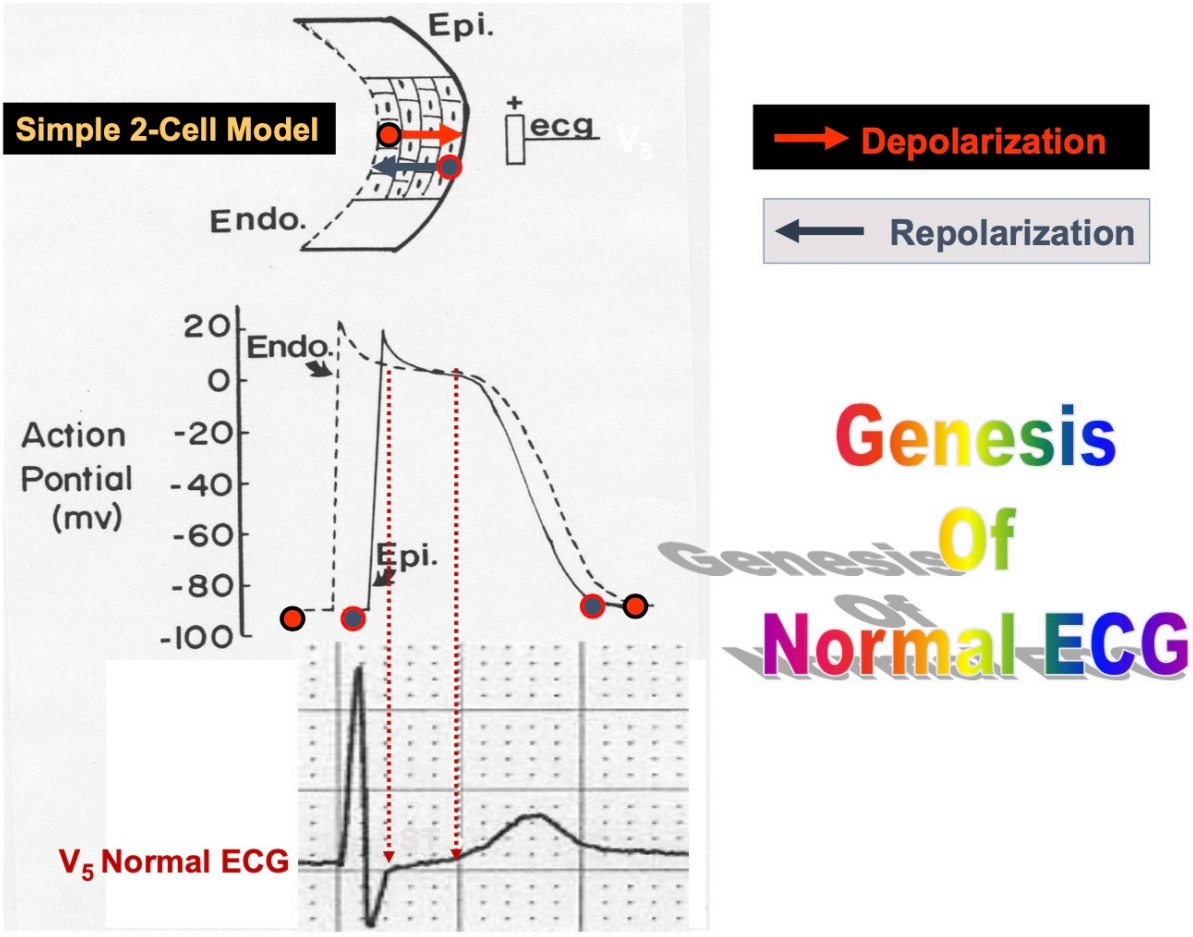

Figure 1

ECG in Injury/Ischemia

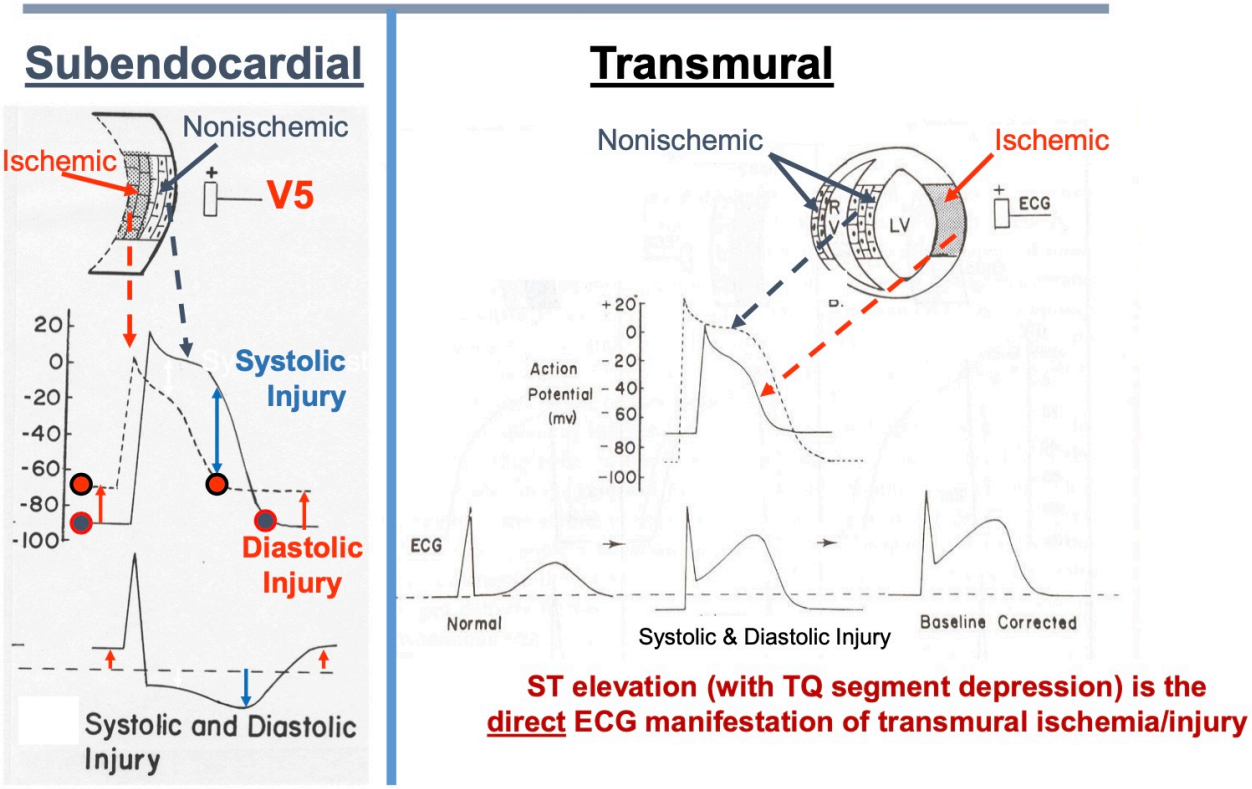

Figure 2