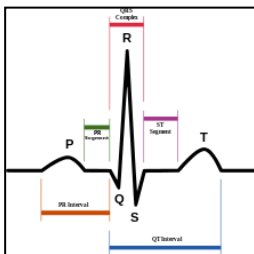


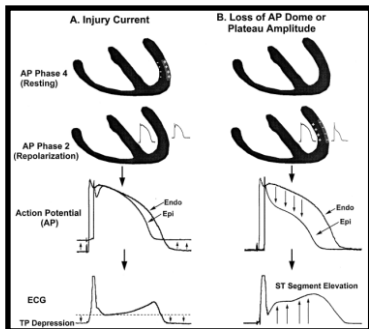
# ST-Segment Elevation on ECG

## Introduction

- Normally, the ST segment is a flat, isoelectric section between the end of the QRS complex (ie. J point) to the beginning of the T wave.
- Represents the transitional time during ventricular depo to repolarization.
- Measure in relation to the end of PR segment or T-P segment.
- 50-80% of patients with STE had diagnoses other than MI.
- 5-10% of patients underwent PCI or thrombolysis did not have MI. (NEJM 2003;349:2128-35).



## Mechanism Underlying ST segment elevation



A. "Injury current": The "injury zone" is in the epicardium, with a reduction in resting membrane potential, produces an injury current during resting phase → TQ depression (instead of ST-segment elevation)

B. "loss of AP dome or plateau amplitude": A difference in the AP plateau amplitude generates a transmural voltage gradient → ST-segment displacement. (True ST-segment elevation)

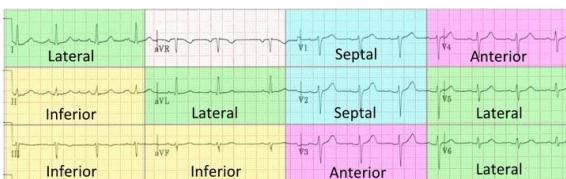
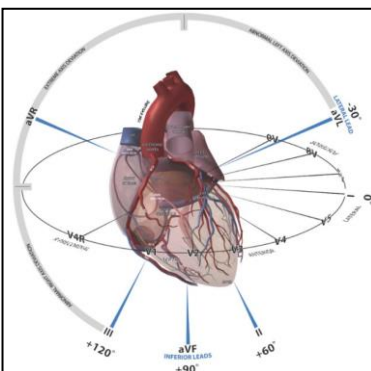
(JACC 2003;42:401-9)

## Clinical Note:

- Always start with H&P not the ECG.
- See table for common causes of STE on ECG.
- Other causes of STE may include myocarditis, post DC cardioversion (15%, last a few minutes, memory T wave?), ARVD, DCM, WPW (pseudo Q), pancreatitis, cholecystitis, external heart compression (tumor), too high chest lead, Tricyclic antidepressants or phenothiazines, scorpion bite.
- Recommend reading Wang K. NEJM 2003;35: 2128-35, 3<sup>rd</sup> universal definition of MI JACC 2012.

## Acute MI (STEMI)

- ECG diagnosis of STEMI (3<sup>rd</sup> universal definition of MI JACC 2012):
  - New ST elevation at the J point in 2 contiguous leads
  - In V2, V3 > 0.2 mV in men > 40 yo, > 0.25 mV in men < 40 yo, > 0.15 mV in women
  - In other leads; > 0.1 mV
- Other causes of STE may meet the criterion for STEMI according to guideline, and thrombolytic or PCI may be harmful.
- Understand axis (in vertical and horizontal plane is crucial for thinking of coronary representation on ECG.



Condition	Note	STE features	ECG Example
<b>STEMI</b> JACC2012;60:1581-98	<ul style="list-style-type: none"> <li>• Clinical + ECG + cardiac marker</li> <li>• usually described as plateau, shoulder, upsloping, tombstone</li> <li>• Dynamic changes</li> </ul>	Convex STE Q, Loss of R wave TWI Reciprocal changes	
<b>Normal (so-call male pattern)</b> JACC2002;40:1870-6	<ul style="list-style-type: none"> <li>• seen in healthy young men</li> <li>• ↓ Prev. with ↑ age. (90% in 20s, 30% in 70 yo men).</li> <li>• The deeper the S the greater the STE</li> </ul>	Concave, 1-3 mm STE Mostly in V2	
<b>Early repolarization</b>	<ul style="list-style-type: none"> <li>• Normal variant?</li> <li>• Young black athlete male.</li> <li>• Early repolarize = short QT, high QRS voltage, where as it is not in AMI or pericarditis.</li> </ul>	Concave STE Most in V4 notch J - "fish-hook" Large not inverted T May see PR depression	
<b>Acute Pericarditis</b> Circ2006;113:1622-1632	<ul style="list-style-type: none"> <li>• Sub-epicidal involvement causing STE</li> <li>• Elevation seldom &gt;5 mm</li> <li>• STE axis usually at 45 degree</li> </ul>	Diffused, concave STE Depressed PR Reciprocal ST/PR segment in aVR	
<b>Left ventricular hypertrophy</b>	<ul style="list-style-type: none"> <li>• Most common cause of STE in ED patient with chest pain</li> </ul>	Concave, V1-V3 See LVH criteria	
<b>Left bundle branch block</b>	<ul style="list-style-type: none"> <li>• abnormal depolarize sequence → abnormal repolarization</li> <li>• Sgarbossa criteria for dx MI in patient with baseline LBBB (NEJM 1996;334:481-7)</li> </ul>	Concave STE, V1-V3 ST-QRS discordant (the opposite direction between ST & QRS)	
<b>Pulmonary embolism</b>	<ul style="list-style-type: none"> <li>• RV pressure overload, dilate, and ischemia</li> <li>• Mild ↑ troponin</li> </ul>	STE in inf, ant'septal S1Q3T3 (~20%) Sinus tachycardia	
<b>Takotsubo</b>	<ul style="list-style-type: none"> <li>• Transient left ventricular apical ballooning, stress induced CM.</li> <li>• DDx: occlusion of wrapping LAD.</li> </ul>	ECG indistinguishable from STEMI	
<b>Brugada syndrome</b> JACC 1992;20:1391-6 Europace 2014;16:1257-1283	<ul style="list-style-type: none"> <li>• loss function of Na channel (SCN5A gene) → loss of AP dome in the RV epicardium (Circ 1999;100:1660-1666)</li> <li>• Unmasked by class 1C.</li> </ul>	rSR' in V1,V2 Downsloping STE Begin at top of R' wave TWI	
<b>Hyperkalemia</b>	<ul style="list-style-type: none"> <li>• Tall T/ P ทาย / wide QRS/ sine wave</li> <li>• DDX with hyperacute T in AMI</li> </ul>	Downsloping Bizarre- looking STE	
<b>Subarachnoid hemorrhage</b>	<ul style="list-style-type: none"> <li>• Catecholamine flooding</li> <li>• May have the same patho with Phaeochromocytoma</li> <li>• +/- RWMA</li> </ul>	Deep, symmetrically TWI QT prolong ↓HR (cushing reflex)	
<b>Hypothermia</b>	<ul style="list-style-type: none"> <li>• Osborn wave</li> <li>• DDx: HyperCa (short QT)</li> </ul>	Prominent J wave "slurred" downstroke QRS complex	
<b>Left ventricular aneurysm</b>	<ul style="list-style-type: none"> <li>• Same patient setting as STEMI w/o acute chest pain</li> <li>• the Taller T, smaller QRS amplitude, the more like AMI than aneurysm.</li> <li>• Tw/QRS ratio &gt; 0.36 = AMI (sens.90%)</li> </ul>	Concave/convex Well-form Q wave No reciprocal ST segment change	