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.

Acute MI (STEMI)
- ECG diagnosis of STEMI (3rd 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
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STEMI | • Clinical + ECG + cardiac marker | Convex STE, Q, Loss of R wave, TWI | [Image]
| | • usually described as plateau, shoulder, upsloping, tombstone | Reciprocal changes | [Image]
Normal | (so-called male pattern) | • seen in healthy young men | Concave, 1-3 mm STE
| | • J prev. with ↑ age: (90% in 20s, 30% in 70 yo men), The deeper the S the greater the STE | Mostly in V2
Early | repolarization | • Normal variant? | Concave STE
| | • Young black athlete male | Most in V4
| | • Early repolarize = short QT, high QRS voltage, where as it is not in AMI or pericarditis | Large not inverted T
Acute | Pericarditis | • Sub-epicardial involvement causing STE | Diffused, concave STE, Depressed PR
| | • Elevation seldom >5 mm | Reciprocal ST/PR segment in aVR
Left ventricular | hyper trophy | • Most common cause of STE in ED patient with chest pain | Concave, V1-V3
| | | | See LVH criteria
Left bundle | branch block | • abnormal depolarization sequence | Concave STE, V1-V3
| | | | ST-QRS discordant (the opposite direction between ST & QRS)
Pulmonary | embolism | • RV pressure overload, dilate, and ischemia | STE in inf, ant septal 1S1Q3T3 (~20%)
| | | | Sinus tachycardia
Takotsubo | | • Transient left ventricular apical ballooning, stress induced CM | ECG indistinguishable from STEMI
| | | | - DDx: occlusion of wrapping LAD.
Brugada | syndrome | • loss function of Na channel (SCN5A gene) | rsR’ in V1,V2
| | | | - Loss of AP dome in the RV epicardium (Circ 1999;100:1660-1666)
| | | | - Unmasked by class IC.
Hyperkalemia | | • Tall T/ P sine wave | Down sloping STE
| | | | - DDx with hyperacutE T in AMI
Subarachnoid | hemorrhage | • Catecholamine flooding | Deep, symmetrically TWI
| | | | - May have the same patho with Phaeochromocytoma
| | | | - +/- RWMA
| | | | - Prominent J wave "slurred" downstroke QRS complex
Hypothermia | | • Osborn wave | V2
| | | | - DDx: HyperCa (short QT)
Left ventricular | aneurysm | • Same patient setting as STEMI w/o acute chest pain | Concave/convex
| | | | - the Taller T, smaller QRS amplitude, the more like AMI than aneurysm.
| | | | - Tw/QRS ratio > 0.36 = AMI (sens.90%)