EKG in STEMI

Consideration of typical EKG patterns in STEMI and STEMI mimickers
EKG Waveform

Letters are used to indicate important points on a typical waveform.
The PR Interval indicates atrioventricular conduction time. The interval is measured from where the P wave begins until the beginning of the QRS complex.
The QRS complex indicates ventricular depolarization. The QRS interval is measured from the end of the PR interval to the end of the S wave.
The QT interval indicates ventricular activity, both depolarization and repolarization. Measure the QT interval from the beginning of the QRS complex to the end of the T wave.
The ST segment traces the early part of ventricular repolarization. The ST segment begins at the end of the QRS complex and continues to beginning of the T wave.
Normal blood supply of the heart
Onset of NSTE-ACS
- Initial recognition and management in the ED by first responders or ED personnel
- Risk stratification
- Immediate management

Hospital Management
- Medication
- Conservative versus ischemia-guided strategy
- Special groups
- Preparation for discharge

Management Prior to NSTE-ACS

Secondary Prevention/Long-Term Management
Pathogenesis of acute coronary syndrome

Hallmark of ACS is sudden imbalance between myocardial oxygen consumption and demand

- Usually result of coronary artery obstruction, MI
- Excessive O2 demand (SVT) in setting of noncritical CAD/tortuosity
- Vasospastic (Prinzmetal) angina
- Coronary embolism
- Coronary arteritis
Pathophysiology of Stable Angina and ACS

Pathophysiology

Decreased $O_2$ Supply
- Flow-limiting stenosis
- Anemia
- Plaque rupture/clot

Increased $O_2$ Demand

$O_2$ supply/demand mismatch $\rightarrow$ Ischemia

Myocardial ischemia $\rightarrow$ necrosis

ACS

Asymptomatic

Angina

Myocardial Infarction
Pathogenesis of acute coronary syndrome

Noncoronary causes of myocardial oxygen supply demand mismatch:

- Hypotension
- Severe anemia
- Severe hypertension
- Tachycardia
- Hypertrophic cardiomyopathy
- Severe aortic stenosis
Pathogenesis of acute coronary syndrome

Non-ischemic/multifactorial etiologies

- Myocarditis
- Cardiac contusion
- Cardiotoxic drugs
- Takotsubo cardiomyopathy
- Pulmonary embolism
- Severe heart failure
- Sepsis
Prevalence of Total Coronary Occlusion during the Early Hours of Transmural Myocardial Infarction


Abstract
To define the prevalence of total coronary occlusion in the hours after transmural myocardial infarction, we used coronary arteriography to study the degree of coronary obstruction in 322 patients admitted within 24 hours of infarction. Total coronary occlusion was observed in 110 of 126 patients (87 per cent) who were evaluated within four hours of the onset of symptoms; this proportion decreased significantly, to 37 of 57 (65 per cent), when patients were studied 12 to 24 hours after the onset of symptoms. Among 59 patients with angiographic features of coronary thrombosis, the thrombus was retrieved by Fogarty catheter in 52 (88 per cent) but was absent in seven (12 per cent false positive). Among an additional 20 patients without angiographic features of thrombosis, a thrombus was discovered in five (25 per cent false negative). Thus, total coronary occlusion is frequent during the early hours of transmural infarction and decreases in frequency during the initial 24 hours, suggesting that coronary spasm or thrombus formation with subsequent recanalization or both may be important in the evolution of infarction. (N Engl J Med. 1980; 303:897-902.)

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CLASS I

1. In patients with chest pain or other symptoms suggestive of ACS, a 12-lead ECG should be performed and evaluated for ischemic changes within 10 minutes of the patient’s arrival at an emergency facility (21). (Level of Evidence: C)

2. If the initial ECG is not diagnostic but the patient remains symptomatic and there is a high clinical suspicion for ACS, serial ECGs (e.g., 15- to 30-minute intervals during the first hour) should be performed to detect ischemic changes. (Level of Evidence: C)
3.3.2.4. Electrocardiogram

The 12-lead ECG is pivotal in the decision pathway for the evaluation and management of patients presenting with symptoms suggestive of ACS (58,59,85). Transient ST changes ($\geq 0.5$ mm [0.05 mV]) during symptoms at rest strongly suggest ischemia and underlying severe CAD.
Differential diagnosis of NSTE-ACS includes (41):

- Nonischemic cardiovascular causes of chest pain (e.g., aortic dissection, expanding aortic aneurysm, pericarditis, pulmonary embolism)
- Noncardiovascular causes of chest, back, or upper abdominal discomfort include:
  - Pulmonary causes (e.g., pneumonia, pleuritis, pneumothorax)
  - Gastrointestinal causes (e.g., gastroesophageal reflux, esophageal spasm, peptic ulcer, pancreatitis, biliary disease)
  - Musculoskeletal causes (e.g., costochondritis, cervical radiculopathy)
  - Psychiatric disorders
  - Other etiologies (e.g., sickle cell crisis, herpes zoster)

In addition, the clinician should differentiate NSTE-ACS from acute coronary insufficiency due to a nonatherosclerotic cause and noncoronary causes of myocardial oxygen supply-demand mismatch (41) (Section 2.2.2).
Alternative causes of ST-T changes include LV aneurysm, pericarditis, myocarditis, bundle-branch block, LV hypertrophy, hyperkalemia, Prinzmetal angina, early repolarization, apical LV ballooning syndrome (Takotsubo cardiomyopathy, Section 7.13), and Wolff-Parkinson-White conduction. Central nervous system events and therapy with tricyclic antidepressants or phenothiazines can cause deep T-wave inversion.
The evolution of an infarct on the ECG. ST elevation, Q wave formation, T wave inversion, normalisation with a persistent Q wave.
How to measure ST elevation?

ST elevation is measured at the junctional or J-point.
Risk Stratification by ECG

- Simple, quick, noninvasive tool
- Universally available, cheap
- Correlates with risk and prognosis
- Guides treatment decisions
- Can identify alternative causes
Risk Stratification by ECG

- ECG Findings and Associated LR for AMI
  - New ST-E $\geq$ 1mm
  - New Q waves
  - Any ST-E
  - New Conduction Defect
  - New ST-D

- NORMAL ECG

LR 5.7-53.9
LR 5.3-24.8
LR 11.2 (7.1-17.8)
LR 6.3 (2.5-15.7)
LR 3.0-5.2
LR 0.1-0.4

Risk Stratification by ECG

CAVEATS

- 1-8% AMI have a normal ECG

- Only Approx 50% of AMI patients have diagnostic changes on their *initial* ECG

Risk Stratification by ECG

CAVEATS cont.

- 1 ECG cannot exclude AMI
  - Brief sample of a dynamic process
- Small regions of ischemia or infarction may be missed

How Sensitive is the ECG Alone?

**Likelihood of MI**

- **WNL**
- **ST ↓**
- **ST ↑**
- **Q**
- **ST ↑ Reciprocal Δ’s or ST ↑ Q Waves**
- **90% - 95%**

The diagram shows the likelihood of myocardial infarction (MI) based on various ECG findings.
STEMI – EKG CRITERIA

- Diagnostic elevation (in absence of LVH and LBBB) defined as:
  
  - New ST elevation at J point in at least 2 contiguous leads

  - in leads V2-V3, men >2mm, women > 1.5mm

  - in other chest leads or limb leads, > 1mm
Normal blood supply of the heart
Wellens’ Syndrome.

Essentially it’s an ECG abnormality strongly associated with significant left anterior descending coronary artery stenosis.
**Hyperacute Anteroseptal STEMI**

- ST elevation is maximal in the anteroseptal leads (V1-4).
- Q waves are present in the septal leads (V1-2).
- There is also some subtle STE in I, aVL and V5, with reciprocal ST depression in lead III.
- There are hyperacute (peaked) T waves in V2-4.
- These features indicate a hyperacute anteroseptal STEMI
Anteroseptal MI: Fully Evolved-KH

Frank G. Yanowitz, M.D.

The QS complexes, resolving ST segment elevation and T wave inversions in V1-2 are evidence for a fully evolved anteroseptal MI. The inverted T waves in V3-5, I, aVL are also probably related to the MI.
Extensive Anterior/Anterolateral MI: Recent-KH

Frank G. Yanowitz, M.D.

Significant pathologic Q-waves (V2-6, I, aVL) plus marked ST segment elevation are evidence for this large anterior/anterolateral MI. The exact age of the infarction cannot be determined without clinical correlation and previous ECGs, but this is likely a recent MI.
Normal blood supply of the heart
Algorithm for Electrocardiographic Identification of the Infarct-Related Artery in Anterior Myocardial Infarction.

ST-segment elevation in V₁, V₂, and V₃

- ST-segment elevation in V₁ (>2.5 mm) or right bundle-branch block with Q wave or both
  - Proximal left anterior descending artery
    - Sensitivity 12%
    - Specificity 100%
    - Positive predictive value 100%
    - Negative predictive value 61%

- ST-segment depression (>1 mm) in II, III, and aVF
  - Proximal left anterior descending artery
    - Sensitivity 34%
    - Specificity 98%
    - Positive predictive value 93%
    - Negative predictive value 68%

- ST-segment depression (≤1 mm) or ST-segment elevation in II, III, and aVF
  - Distal left anterior descending artery
    - Sensitivity 66%
    - Specificity 73%
    - Positive predictive value 78%
    - Negative predictive value 62%

Proximal Culprit Lesion in LAD:

Short

Conventional

Wrap Around
Right Coronary Artery

100% blockage!!

The same artery after being treated with a stent procedure.
A 54 year old man presents with one hour of severe central chest pain.

His observations are:

<table>
<thead>
<tr>
<th>BP</th>
<th>80/60 mmHg supine</th>
</tr>
</thead>
<tbody>
<tr>
<td>O2 saturation</td>
<td>97% room air</td>
</tr>
</tbody>
</table>
Electrocardiogram Showing Inferior Myocardial Infarction Associated with Complete Heart Block with a Narrow Escape Rhythm.

Fig. 2

Example ECG of patient with subtle inferior STEMI, but evidence of ST depression in aVL which could help make this difficult diagnosis. This patient had acute 99% mid-RCA occlusion.
Algorithm for Electrocardiographic Identification of the Infarct-Related Artery in Inferior Myocardial Infarction.

ST-segment elevation in III > ST elevation in II and ST-segment depression in I, aVL, or both (>1 mm)

Yes

Right coronary artery
Sensitivity 90%
Specificity 71%
Positive predictive value 94%
Negative predictive value 70%

In addition, ST-segment elevation in V₁, V₂, or both

Proximal right coronary artery with right ventricular infarction
Sensitivity 79%
Specificity 100%
Positive predictive value 100%
Negative predictive value 88%

No

ST-segment elevation in I, aVL, V₅, and V₆ and ST-segment depression in V₁, V₂, and V₃

Left circumflex coronary artery
Sensitivity 83%
Specificity 96%
Positive predictive value 91%
Negative predictive value 93%

High Lateral STEMI:
Inferolateral STEMI:
Inferoposterolateral STEMI:
Alternative causes of ST–T changes

- LV aneurysm
- Pericarditis
- Myocarditis
- Bundle-branch block
- Left ventricular hypertrophy
- Hyperkalemia
- Prinzmetal angina
- Early repolarization
- Apical ballooning (Takotsubo cardiomyopathy)
- Wolff-Parkinson-White
- CNS events
- Tricyclic antidepressants
- There is generalised concave ST elevation in the precordial (V2-6) and limb leads (I, II, III, aVF).
- J-point notching is evident in the inferior leads (II, III and aVF).
- There are prominent, slightly asymmetrical T waves that are concordant with the main vector of the QRS complexes.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (so-called male pattern)</td>
<td>Seen in approximately 90 percent of healthy young men; therefore, normal</td>
</tr>
<tr>
<td></td>
<td>Elevation of 1–3 mm</td>
</tr>
<tr>
<td></td>
<td>Most marked in $V_2$</td>
</tr>
<tr>
<td></td>
<td>Concave</td>
</tr>
<tr>
<td>Early repolarization</td>
<td>Most marked in $V_4$, with notching at J point</td>
</tr>
<tr>
<td></td>
<td>Tall, upright T waves</td>
</tr>
<tr>
<td></td>
<td>Reciprocal ST depression in aVR, not in aVL, when limb leads are involved</td>
</tr>
<tr>
<td>ST elevation of normal variant</td>
<td>Seen in $V_2$ through $V_5$ with inverted T waves</td>
</tr>
<tr>
<td></td>
<td>Short QT, high QRS voltage</td>
</tr>
</tbody>
</table>
Electrocardiograms Showing Normal ST-Segment Elevation and Normal Variants.

Tracing 1

Tracing 2

Tracing 3

<table>
<thead>
<tr>
<th>Condition</th>
<th>Other Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular hypertrophy</td>
<td>Concave</td>
</tr>
<tr>
<td></td>
<td>Other features of left ventricular hypertrophy</td>
</tr>
<tr>
<td>Left bundle-branch block</td>
<td>Concave</td>
</tr>
<tr>
<td></td>
<td>ST-segment deviation discordant from the QRS</td>
</tr>
<tr>
<td>Acute pericarditis</td>
<td>Diffuse ST-segment elevation</td>
</tr>
<tr>
<td></td>
<td>Reciprocal ST-segment depression in aVR, not in aVL</td>
</tr>
<tr>
<td></td>
<td>Elevation seldom &gt;5 mm</td>
</tr>
<tr>
<td></td>
<td>PR-segment depression</td>
</tr>
<tr>
<td>Hyperkalemia</td>
<td>Other features of hyperkalemia present:</td>
</tr>
<tr>
<td></td>
<td>Widened QRS and tall, peaked, tented T waves</td>
</tr>
<tr>
<td></td>
<td>Low-amplitude or absent P waves</td>
</tr>
<tr>
<td></td>
<td>ST segment usually downsloping</td>
</tr>
<tr>
<td>Condition</td>
<td>Description</td>
</tr>
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<td>--------------------------------</td>
<td>----------------------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| Brugada syndrome               | rSR' in $V_1$ and $V_2$  
ST-segment elevation in $V_1$ and $V_2$, typically downsloping |
| Pulmonary embolism             | Changes simulating myocardial infarction seen often in both inferior and anteroseptal leads |
| Cardioversion                  | Striking ST-segment elevation, often $>10$ mm, but lasting only a minute or two immediately after direct-current shock |
| Prinzmetal's angina            | Same as ST-segment elevation in infarction, but transient                               |
| Acute myocardial infarction    | ST segment with a plateau or shoulder or upsloping  
Reciprocal behavior between aVL and III |
Electrocardiograms Showing ST-Segment Elevation in Various Conditions.

Embolism Who Had a Normal Coronary Angiogram (Tracing 1) and a Patient with Transient ST-Segment Elevation Immediately after Direct-Current (DC) Countershock to the Precordium (Tracing 2).
Classic HCM pattern with asymmetrical septal hypertrophy

- Voltage criteria for left ventricular hypertrophy.
- Deep narrow Q waves < 40 ms wide in the lateral leads I, aVL and V5-6.
Dagger-like Q waves
Figure 1
An electrocardiogram ordered on admission showed sinus rhythm, first-degree heart block, normal QT/QTc intervals, marked scooped ST elevations in leads V1 through V5, and scooped ST depressions in inferior leads.
Milk- alkali syndrome w hypercalcemia

Figure 2
The electrocardiogram obtained prior to discharge demonstrated resolution of first-degree heart block and ST elevations.
Fig. 1
12 lead ECG showing ST segment elevation in leads II, III, aVF, V-4, V-5 & V-6.
Ogilvie’s syndrome
Fig. 2

A: White arrows showing indentation of right atrial free wall. B: Preserved geometry of LV cross section during systole. C: Deformation of inferior infero-lateral segment secondary to elevated hemi-diaphragm.
Supplementary Fig. 1

Twelve-lead ECG demonstrating 2-mm ST elevation in V₁ to V₃ and 1-mm ST depression in lead I, along with 2-mm ST depression in V₅ and 3-mm depression in V₆ as well as a new incomplete right bundle-branch block.
Example of ECG of patient with pericarditis. Although there is clear ST elevation in the inferior leads, lead aVL lacks ST depression.
Fig. 4

ECG, few hours later, showing prominent diffuse ST-segment elevation with PR-segment depression.
A 12-Lead Electrocardiogram from a Patient with Acute Pericarditis, Demonstrating Widespread ST-Segment Elevation and PR-Segment Depression.
Atrial Flutter With 2:1 AV Conduction-KH

Frank G. Yanowitz, M.D.

Atrial flutter with 2:1 AV block is one of the most frequently missed ECG rhythm diagnoses because the flutter waves are often hard to find. In this example two
Example (b) – Atrial Flutter with 2:1 Block

[ECG waveform showing atrial flutter with 2:1 block]
Typical ECG findings with left main coronary artery (LMCA) occlusion:

- Widespread horizontal ST depression, most prominent in leads I, II and V4-6
- ST elevation in aVR ≥ 1mm
- ST elevation in aVR ≥ V1

ST Elevation in aVR may also be seen with:

- Proximal left anterior descending artery (LAD) occlusion
- Severe triple-vessel disease (3VD)
- Diffuse subendocardial ischaemia – e.g. due to O2 supply/demand mismatch, following resuscitation from cardiac arrest
Proximal LAD occlusion

- ST elevation in aVR and V1 of similar magnitude.
- Widespread ST depression (V3-6, I, II, III, aVF)
These are images from two different patients. The left main artery, as can been seen above, gives rise to the two main arteries that supplies the heart, the left anterior descending artery (LAD) and the circumflex artery (Lcx). If the left main artery goes totally down, so do the other arteries! Chances of survival are slim with a left main artery heart attack. This is why it’s the mother of all Widowmakers.
LMCA Occlusion

- Marked ST elevation in aVR >> V1
- ST depression in multiple leads (V2-6, I, II, aVL, aVF), to some extent masked by a non-specific interventricular conduction delay
Digoxin effect:

- This is the classic picture of digoxin effect with the “sagging” ST segments and T waves taking on the appearance of “Salvador Dali’s moustache”.

• The morphology of the QRS complex / ST segment is variously described as either “slurred”, “sagging” or “scooped” and resembling either a “reverse tick”, “hockey stick” or (my personal favourite) “Salvador Dali’s moustache”!
A 21 year old male of Asian descent has presented to your ED following a brief episode of syncope. He feels fine now and wants to go home. His ECG looks like this:
- LV aneurysm
Accelerated Junctional rhythm
ST elevation consider inferior injury or acute infarct
Prolonged QT

ACUTE MI

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A

B
LVH by voltage criteria: S wave in V2 + R wave in V5 > 35 mm

LV strain pattern: ST depression and T wave inversion in the lateral leads
Good example of LVH:

- Markedly increased LV voltages: huge precordial R and S waves that overlap with the adjacent leads (SV2 + RV6 >> 35 mm).
- R-wave peak time > 50 ms in V5-6 with associated QRS broadening.
- LV strain pattern with ST depression and T-wave inversions in I, aVL and V5-6.
- ST elevation in V1-3.
- Prominent U waves in V1-3.
- Left axis deviation.
Advanced Hyperkalemia

Marked widening of the QRS duration combined with tall, peaked T waves are suggestive of advanced hyperkalemia. Note the absence of P waves, suggesting a junctional rhythm, but in hyperkalemia the atrial muscle may be paralyzed while still in sinus rhythm. The sinus impulse conducts to the AV node through internodal tracts without activating the atrial muscle.
Hyperkalemia and Old Inferior MI

Frank G. Yanowitz, M.D. ©1998

The T waves are tall, peaked and have a narrow base, making them very uncomfortable to sit on! These changes are characteristic of hyperkalemia. The QRS is also slightly widened, another feature of hyperkalemia. Q waves in III and aVF indicate an old inferior MI.
Figure 1. Spectrum of ECG patterns observed in 3 patients with psychologically triggered, reversible LV dysfunction that mimics myocardial infarction or acute coronary syndrome.