Advanced ECG Interpretation

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DISCLOSURES

None of the planners or presenters of this session have disclosed any conflict or commercial interest
Objectives

• Identify ECG changes related to hypertrophy, bundle branch blocks, and MI’s

• Review approach to interpretation of wide complex tachycardia

• Describe other miscellaneous causes of ECG abnormalities: pericarditis, electrolyte abnormality, medication effects, and hypothermia

• Practice using a systematic approach to interpreting 12 lead ECGs
• Positive in leads I, II, V₄ & V₆
• Negative in aVR
• Morphology changes depending on pacemaker
• .08-0.11
Atrial Enlargement
PR Interval
PR Interval
Ventricular Hypertrophy
Less than 5 mm in all limb leads
Less than 10 mm high in all precordials

Obesity, pleural effusion, pericardial effusion
Q Waves

• Pathologic Q wave = Dead Myocardium

• Septal Q waves in Lead I and aVL

• QS in V1

• Less than 0.04 and less than 1/3 the height of the R wave
Benign QS waves

Pathologic QS waves
• Isolate the smallest and most isoelectric limb lead
• Identify the lead perpendicular to this lead
• Is the QRS positive or negative in this lead?
• Is the isoelectric lead more positive or negative?
Right Bundle Branch Block

- QRS > 0.12
- Slurred S wave in leads I and V6
- RSR’ pattern in V1 or V2
Left Bundle Branch Block

- QRS $\geq 0.12$

- Broad, monomorphic R waves in I and V6

- Broad monomorphic S waves in V1

QRS Morphology

Discordant ST-Segments and T-Waves

Normal for LBBB and paced rhythm
Concordant ST elevation > 1 mm in leads with a positive QRS complex (score 5)

Concordant ST depression > 1 mm in V1-V3 (score 3)

Excessively discordant ST elevation > 5 mm in leads with a negative QRS complex (score 2).

A total score of ≥ 3 is reported to have a specificity of 90% for diagnosing myocardial infarction.

Sgarbossa’s Criteria
Intraventricular Conduction Delay
Intraventricular Conduction Delay
Left Anterior Hemiblock

Left Axis Deviation -30 to -90

qR or and R wave in lead I

rS in lead III, and probably II & aVF
Left Posterior Hemiblock

- Right Axis Deviation 90-180
- S wave in lead I and q in III
- Exclusion of RAE and/or RVH
### Bifascicular Blocks

**Lead I**
- Slurred S
- LPH: Green
- LAH: Blue

**Lead II**
- LAH: Blue

**Lead III**
- aVF: Green
- LPH: Blue
- LAH: Blue

**Lead V₁**
- Rabbit Ears

**Lead V₆**
- Slurred S
Bifascicular Blocks
Bifascicular Blocks
## Wide Complex Tachycardia vs. V-tach

<table>
<thead>
<tr>
<th>Complexes &gt; 160 ms</th>
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<tbody>
<tr>
<td>Absence of RBBB or LBBB morphology</td>
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<tr>
<th>Extreme axis deviation: Negative in I + aVF</th>
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<td>AV dissociation</td>
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<th>Capture beats</th>
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<td>Fusion beats</td>
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<th>Positive or negative concordance in precordial leads with no RS seen</th>
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<tr>
<td>Brugada’s sign- Onset of QRS to nadir of S wave &gt;100ms</td>
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<tr>
<th>Josephson’s sign – Notching near the nadir of the S-wave</th>
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<td>RSR’ complexes with a taller “left rabbit ear”.</td>
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T Waves

- **Shape**
- **Polarity**
- **Size**

Figure 14-8

- Asymmetrical Positive
- Asymmetrical Negative
- Tall, Peaked Narrow
- Tall, Broad
- Very Broad! Positive
- Very Broad! Negative

Watch out for CNS events in these very broad types!
ST segments
AMI Location Correlation

I Lateral  aVR  V₁ Septal  V₄ Anterior

II Inferior  aVL  Lateral  V₂ Septal  V₅ Lateral

III Inferior  aVF Inferior  V₃ Anterior  V₆ Lateral
Reciprocal

- Mirror image when 2 electrodes viewing the MI from opposite angles
• How do we determine what is happening with the posterior wall of the heart?

• Reciprocal changes will occur in what leads?

• Can perform a posterior ECG with lead V7 to V10

Posterior MI
Posterior MI
RV infarct

- Complicates an estimated 40% of inferior MI’s
- Very uncommon to have isolated RV infarct
- Very preload sensitive
- ST elevation in V1
- ST elevation in lead II > III
- How can we look at the right side of the heart?
Inferior-RV-Posterior

- **I**: Lateral, aVR
- **II**: Inferior, aVL, High Lateral
- **III**: Inferior, aVF, Inferior (Lead III>II)
- **V1**: Septal
- **V4R**: Right-sided
- **V2**: ST elevation ≥0.5X, ST depression*
- **V3**: Anterior
- **V6R**: Right-sided
RVH with Strain

Figure 14-18: Leads V₁ to V₂.

- Increased R:S ratio
- ST Depression
- Flipped Asymmetric T wave
- Concave Downward
LVH with Strain
### Miscellaneous Abnormalities

<table>
<thead>
<tr>
<th>Notching</th>
<th>Osborn Waves</th>
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<tr>
<td><img src="image1.png" alt="Heart ECG" /></td>
<td><img src="image2.png" alt="Heart ECG" /></td>
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</table>

- **Notching**
  - Sever hypothermia

- **Osborn Waves**
  - Large deflection at the end of the QRS complex
  - Can also cause ST dep. and flipped T waves
Pericarditis
Osborn Waves
Hypocalcemia

- Prolongation of the ST segment with lengthening of the QTc interval
- At risk for the development of torsades de pointes
- ST segment depression in inferior leads ? ischemia
- This ECG also shows LVH with possible strain. Flipped symmetrical T waves in lateral leads.
Hypokalemia

- Mild ST depression
- Mild decreased amplitude of T waves
- Minimal prolongation of the QRS interval
- Prominent U wave
<table>
<thead>
<tr>
<th>Drugs</th>
<th>Possible Toxic Effects</th>
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<tr>
<td>Class I antiarrhythmics</td>
<td>• Lengthened QRS and QTc intervals</td>
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<td>• Possible AV blocks</td>
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<tr>
<td></td>
<td>• Slowed or completely blocked SA node</td>
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<tr>
<td></td>
<td>• Arrhythmias</td>
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<tr>
<td>Calcium Channel Blockers</td>
<td>• Blocked AV node</td>
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<tr>
<td>Beta-blockers</td>
<td>• Slowed automaticity of the SA node</td>
</tr>
<tr>
<td></td>
<td>• Blocked AV node</td>
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<tr>
<td>Amiodarone</td>
<td>• Slowed conduction everywhere: SA node, atrium, AV node, ventricles</td>
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<tr>
<td>Phenothizines and tricyclic antidepressants</td>
<td>• Widened QRS and QTc interval</td>
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<tr>
<td></td>
<td>• T wave abnormalities</td>
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Digoxin Effects
References


