**Summary:** There are many ways to read an ECG. During this session, we will teach a particular method of ECG interpretation. We will emphasize this systematic approach with examples that range in difficulty from “basic” to “attending” level. We will work together to realize when an ECG is “abnormal,” and try to describe the abnormality. By integrating the clinical cases with the ECG findings, we will try to determine a diagnosis.

**Objectives:**
- Develop a systematic approach to ECG interpretation.
- Engage in an interactive, clinical-case based session
- Learn some of the nuances of ECG reading we hope will be useful to you on wards
- Feel comfortable to read an ECG during morning report if called on

**Instructor notes:**
*In this guide, we provide a “suggested approach” to ECG interpretation. When leading the session, it may be useful to briefly review the 6 core steps in reading an ECG. The teaching points will come up with each sample ECG. Please spend the majority of time going through the various examples.*
*Some of the “scenario” text included in the instructor guide is not available to the interns or on the powerpoint slides. You may choose to give this information as the interns proceed through the interpretation of each ECG. This may help to focus their attention to particular details and emphasize the importance of using the clinical history as an adjunct to their interpretation.*
Free online ECG resources:
1. http://ecg.bidmc.harvard.edu
2. http://ecg.utah.edu/

A Suggested Approach to the ECG

1. **What is the rate?**
   Two simple methods for rate estimation:
   - Regular rhythm: approximate by using the “300, 150, 100, 75, 60, 50” approach by counting large boxes. Take “300 bpm” and divide by the number of large boxes.
   - Irregular rhythms: count the number of total beats on the strip and multiply by 6 (ECG is a 10 second strip).

2. **What is the rhythm?**
   Some important questions to guide your approach to rhythm:
   - Is the rhythm regular? (Tip: march out p waves and QRS complexes on a piece of paper, or use a caliper. Marching out with caliper opened to include 2-3 QRS complexes will often facilitate accuracy.)
   - For sinus rhythm, must be able to answer “yes” to the following 3 questions:
     - Is there a P before every QRS complex?
     - Is there a QRS before every P?
     - Is the QRS narrow? (<3 little boxes/120 msec)

3. **What is the axis?**
   Normal QRS axis ranges from -30° to + 90° (up in I and II).
   - LAD is < -30° (up in I, down in II).
   - RAD: > + 90° (down in I, up in aVF).
Normal P wave axis ranges 0° to +75°. Roughly, the P waves should be upright in I and II.
- Abnormal P wave axis DDX: Limb lead reversal, ectopic atrial focus, retrograde atrial activation (retrograde P wave)

4. **How long are the intervals?**
   PR interval: conduction through the AV node
   - The PR interval should be 3-5 little boxes or < 1 large box (120-200 msec).
   - If > 200, first degree heart block
   - If <120, likely preexcitation: accessory pathway between atria and ventricles (ex. WPW) or AV nodal (junctional) rhythm

   QRS complex: ventricular activation
   - The QRS should be 60-100 msec (3 little boxes = 120 msec)
   - If <100-120, incomplete BBB, nonspecific intraventricular conduction delay (IVCD), left anterior or posterior fascicular block in some cases
   - If >120 ms, ddx includes BBB, Vtach or other ectopic rhythms, IVCD, hyperK

   QT interval: ventricular depolarization and repolarization
   - \( QT_c = QT / \sqrt{RR} \) (sec). Should be ≤ 40 msec
   - Ddx for prolonged QT includes: hereditary long QT syndrome; drugs (ex. psych meds, antiarrythmics, antiemetics); low K, Ca or Mg; CNS disease, post-MI.

5. **Is there hypertrophy?**

   **RAE (right atrial enlargement):** In right atrial hypertrophy, look for “P pulmonale.” The P wave in II is more than 2.5 little boxes tall.

   **LAE (left atrial enlargement):** In left atrial hypertrophy, look for “P mitrale.” The P wave in II is more than 3 mm wide and often has 2 humps. Also, look in V1 for a biphasic P wave with a terminal deflection more than 1 mm wide.

   **Right ventricular hypertrophy:** For RVH to be present, look for tall R waves in V1 (> 7mm tall) and RAD.

   **Left ventricular hypertrophy:** For LVH to be present, refer to the criteria in the table.

   **Note:** these are generally specific, but insensitive. Satisfying any one of these criteria is generally sufficient.
6. Is there evidence for acute or past infarction?
   - Is there ST elevation? If so, is it in a particular vascular territory?
   - Is there ST depression? If so, check for reciprocal ST elevation.
   - Are there Q waves? Remember a pathologic Q wave is one box wide and two boxes deep or 25% of the height of the R wave. Pathologic if in V1-3.
   - Are there T wave changes? (see below)

7. Are the waveforms normal?
   P waves:
   - Is the shape funny? If so, there may be an ectopic atrial focus.
   - Does it look upside down? Perhaps there is retrograde atrial depolarization.

QRS complex
Is there evidence for pre-excitation? (i.e. slurred upstroke)
- Is there low voltage? (No QRS greater than 10 mm tall or no limb lead > 5 mm tall)
- Is the transition across the precordium normal? (i.e. normal R wave progression. R>S by V4.

Is there evidence for bundle branch block?
- RBBB: Wide QRS and RSR’ in V1 or V2
- LBBB: Wide QRS and broad monophasic R wave in I and V6
- LAFB: LAD –45 to –90 degrees (i.e. up in I, down in II), qR complex in I, rS in aVF
- LPFB: RAD 100-180 degrees (i.e. down in I and up in aVF), rS complex in I and qR in aVF

T waves
- Are the T waves flattened, inverted or peaked?

Interpreting ECGs: Instructor’s guide to the sample ECGs

- **Note**: Scenario text in italics is not included on the Powerpoint slide
- Go around and ask a student to try to identify ECG abnormalities and integrate findings with clinical scenario. Ask them to try to be brief (<1 min), so the majority of time may be spent on teaching. Ex. Rate, rhythm, axis, intervals, hypertrophy, ischemia, waveforms, synthesis
- Specific Qs for group, slide summaries, and transitions included under teaching notes.

<table>
<thead>
<tr>
<th>ECG</th>
<th>Scenario</th>
<th>Dx</th>
<th>Key ECG findings</th>
<th>Teaching Notes / Take Home Pearls</th>
</tr>
</thead>
<tbody>
<tr>
<td>PART 1: ST Segments</td>
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</tbody>
</table>
| 1   | 64 F with chest pain (Left Main thrombus, cardiogenic shock) | NSTEMI           | ▪ ST depressions in V3-V6  
▪ TWI in V3-V6  
▪ aVR elevation | ▪ Let the history focus your attention on key aspects of the ECG.  
▪ aVR elevation is an ominous sign. Can often suggest LM disease, proximal LAD, or severe 3-vessel disease. AVR is electrically |
### PART 1: COMMON cardiology CONDITIONS

<table>
<thead>
<tr>
<th>Case</th>
<th>Clinical Presentation</th>
<th>Diagnosis</th>
<th>ECG Findings</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>54M with chest pain (recent URI, pain radiates to L shoulder, improves with leaning forward, rub on exam)</td>
<td>Pericarditis</td>
<td>Diffuse STE - PR depressions - Changes reversed in aVR</td>
<td>What makes this reassuringly less likely STEMI? Hx and diffuse changes. For STEMI, look for regionalization of STE. Slide on the ddx of STE: Tracings 1-7: 1. LVH; 2. LBBB; 3. Pericarditis (STE diffuse including precordial leads and II, also PR dep); 4. Pseudoinfarct of ↑K – T wave tall, narrow and tented; 5. Acute anteroseptal MI; 6. Acute anteroseptal MI + RBBB; 7. Brugada (RSR’, STE only V1+V2, long QTc)</td>
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<tr>
<td>3</td>
<td>68F in CCU (recent anterior MI, 3v CAD, 90% LAD culprit lesion)</td>
<td>Anterior MI, subacute</td>
<td>Anterior Q waves - LAD - LAFB - Poor R wave progression</td>
<td>Slide on DDx of Poor R wave progression: (Anterior MI, Cardiomyopathy, LVH, RVH/COPD, clockwise rotation of the heart, LBBB, Lead misplacement); Slide on DDx of LAD: 1. LAFB (Axis is &lt; -45 degrees); 2. LVH (Usually not extreme LAD); 3. IMI; 4. LBBB (Usually not extreme LAD) - transition to Part 2 emergencies -</td>
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</tbody>
</table>

### PART 2: COMMON EMERGENCIES

<table>
<thead>
<tr>
<th>Case</th>
<th>Clinical Presentation</th>
<th>Diagnosis</th>
<th>ECG Findings</th>
<th>Interpretation</th>
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<tbody>
<tr>
<td>4</td>
<td>55M found down (dizziness, new renal failure, K 7.6)</td>
<td>Hyperkalemia</td>
<td>Peaked T waves - Wide QRS - AV block - Bradycardia</td>
<td>Slide: ECG in hyperkalemia; Treatments include: Calcium, Insulin/dextrose, Beta agonists, Kayexelate (+miralax or lactulose), Lasix, Dialysis</td>
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<td>5</td>
<td><strong>62M with NSCLC and dyspnea</strong>&lt;br&gt;(large effusion with RV collapse. 1L drained)</td>
<td>Pericardial effusion</td>
<td>▪ Low voltage&lt;br▪ Electrical alternans&lt;br▪ You’ll only find it if you think of it.&lt;br▪ Pericardial effusions are high on the differential in the oncology patient.&lt;br▪ If you have an onc pt with dypnea/CP, tachycardia, or hypotension, check JVP and pulsus.&lt;br▪ Next steps: check pulsus, call IR, think preload dependent.</td>
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<tr>
<td>6</td>
<td><strong>71F with acute SOB</strong></td>
<td>PE</td>
<td>▪ Sinus tach&lt;br▪ S1Q3T3&lt;br▪ ECG findings assoc w/ acute PE can be seen in other conditions that lead to acute pHTN&lt;br▪ Most common ECG finding in PE is sinus tach (&gt;40%)&lt;br▪ S1Q3T3 is not sensitive or specific (only 20% of acute PEs)&lt;br▪ P pulmonale (RAE)&lt;br▪ RBBB: ~18% pts and assoc with incr. mortality&lt;br▪ R axis deviation&lt;br▪ RV strain: TWIs in V1-V4 +/- inf (II/III/aVF): ~up to 1/3 of pts&lt;br▪ Signs of RV dilatation such as prominent R in V1 or clockwise rotation (R/S transition pt toward V6 with S wave in V6)&lt;br▪ Afib, Aflutter or Atach only in 8%</td>
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<td><strong>PART 3: RHYTHM</strong></td>
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<td>7</td>
<td><strong>47F feels fine</strong>&lt;br&gt;(congenital heart block 2/2 neonatal lupus)</td>
<td>Complete Heart Block</td>
<td>▪ AV dissociation&lt;br▪ Junctional escape rate of ~45bpm.&lt;br▪ Call EP, patient will need a PPM</td>
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<td>8</td>
<td><strong>85M with palpitations</strong></td>
<td>AVNRT</td>
<td>▪ SVT&lt;br▪ Retrograde P waves&lt;br▪ Regular rhythm&lt;br▪ Absence of flutter waves&lt;br▪ Short RP tachycardia likely ANRT&lt;br▪ Old ECG is helpful to compare QRS and P wave morphologies</td>
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<tr>
<td>9</td>
<td><strong>67F with palpitations</strong>&lt;br&gt;(history of pAF)</td>
<td>AFib with RVR</td>
<td>▪ Irreg irregular&lt;br▪ Would review quickly given common&lt;br▪ Q: What do you need to know next? (A: Patient’s Blood pressure)</td>
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<tr>
<td>No.</td>
<td>Description</td>
<td>Rhythm</td>
<td>Details</td>
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</table>
| 10  | **72M with severe COPD p/w palpitations**                                   | MAT    | - 3 diff p wave morphologies  
- Irreg irreg tach  
- MAT often associated with severe lung disease.  
- If HR<100 with at least 3 p wave morphologies = wandering atrial pacemaker |
| 11  | **77M with palpitations (CAD s/p CABG, prior inf MI, inf scar. VT resolved w/ shock. ICD implanted)** | Monomorphic VT | - Wide QRS  
- Precordial RS >100msec  
- Regular, wide complex tachycardia in a patient with a hx of CAD → VT until proven otherwise!  
- Next Q: Do they have a pulse? (if not, start chest compressions, will need to shock)  
- *Slide: Review ACC Algorithm for Wide Complex Tachycardia*  
  *transition to Part 3 -- rhythm* |