Goal with this seminar

- Learn a systematic approach for analysing ECGs:” 5+1”
- Recognize and understand normal ECGs
- Interpret abnormalities in rhythm, conduction or morphology
Physiology

- The sinoatrial node (SA node) contains the fastest physiological pacemaker cells of the heart; therefore, they determine the heart rate.
- SA-node initiates depolarization, first atria, than ventricles by help of specialized conduction system.
- Repolarization follows depolarization.
• Depolarization of myocardial cells causes positive electrical charges which can be measured by electrodes.

• Electrical charges moving towards an electrode causes positive deflection relative to the isoelectric line.

• Electrical charges moving away from an electrode causes negative deflection.
Electrical axis of heart

- The electrical heart axis is an average of all depolarizations in the heart.
- The depolarization wave begins in the right atrium and proceeds to the left and right ventricle.
- Because the left ventricle wall is thicker than the right wall, the arrow indicating the direction of the depolarization wave is directed to the left.
Placements of electrodes (leads)
Limb leads (Vertical plane)

- Info from limb electrodes are combined to produce six limb leads.
- Try to look at the leads as eyes “looking” at the heart from different angles.
- Group the leads together into right, left and inferior-posterior.
Chest leads (horizontal plane)
• The **PQ interval** starts at the beginning of the atrial contraction and ends at the beginning of the ventricular contraction (0.12 – 0.20 seconds)

• The **QRS duration** indicates how fast the ventricles depolarize (normal < 0.10 seconds)

• The **normal QTc (corrected) interval** indicates how fast the ventricles are repolarized, becoming ready for a new cycle. (below 0.45 seconds in men and below 0.46 in women)
Normal ECG – “Sinus ryhtm”

• A P-wave (atrial contraction) precedes every QRS complex
• The rhythm is regular, but varies slightly during respirations
• The rate ranges between 60 and 100 beats per minute
• The P waves maximum height at 2.5 mm in II and/or III
• The P wave is positive in I and II, and biphasic in V1
Normal ECG
Arrhythmias

Classifications based on heart rate:

1. Tachycardia (HR > 100 bpm)
2. Bradycardia (HR < 60 bpm)

Classification based on origin of impulse:

1. Supraventricular – ”Narrow QRS complex” (< 0.12 ms)
2. Ventricular - ”Wide QRS complex” (> 0.12 ms)
Tachyarrhythmias

- Supraventricular
  - Atrial
  - Nodal
- Ventricular
An overview of pathological supraventricular arrhythmias and their origin

- AV nodal re-entry tachycardia (AVNRT)
- Atrial flutter (most common around tricuspid annulus)
- Atrial tachycardia (single atrial focus)
- Atrial fibrillation (multiple atrial wavelets)
- AV re-entry tachycardia (re-entry through accessory bundle)
Re-entry mechanism

atrium
av-node
His

slow
fast

atrial echo
# Sinustachycardia

Atrial frequency | 100 – 180 bpm  
Ventricular frequency | 100 – 180 bpm  
Regularity | Regular  
Origin | Sinus node  
P-wave | Positive in II, aVF  
Effect of Adenosine | No (can lead to temporary AV block

![ECG Waveform](image)
Sinustachycardia

Causes:

• Exercise, anxiety, alcohol, caffeine, drugs
• Fever
• Hypoxia
• Bleeding
• Anemia
• Hyperthyroidism
• ++++++
### Atrial tachycardia

<table>
<thead>
<tr>
<th>Description</th>
<th>Details</th>
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</thead>
<tbody>
<tr>
<td>Atrial frequency</td>
<td>&gt; 100</td>
</tr>
<tr>
<td>Ventricular frequency</td>
<td>&gt; 100</td>
</tr>
<tr>
<td>Regularity</td>
<td>Regular</td>
</tr>
<tr>
<td>Origin</td>
<td>Ectopic foci in atrium (re-entry)</td>
</tr>
<tr>
<td>P-wave</td>
<td>Negative in I, aVF (different morphology)</td>
</tr>
<tr>
<td>Effect of Adenosine / Vagal stimulation</td>
<td>Slow down rythm (AV-conduction)</td>
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</tbody>
</table>

![ECG tracing](image)
Atrial fibrillation (AF)
Atrial Fibrillation (AF)

<table>
<thead>
<tr>
<th>Description</th>
<th>Details</th>
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</thead>
<tbody>
<tr>
<td>Atrial frequency</td>
<td>400-600 bpm</td>
</tr>
<tr>
<td>Ventricular frequency</td>
<td>75-175 bpm</td>
</tr>
<tr>
<td>Regularity</td>
<td>Irregular</td>
</tr>
<tr>
<td>Origin</td>
<td>Atria (SVT)</td>
</tr>
<tr>
<td>P-wave</td>
<td>Absent</td>
</tr>
<tr>
<td>Effect of Adenosine</td>
<td>Reduces heart rate</td>
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</tbody>
</table>

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[Heart rate diagram]
Atrial Fibrillation (AF)
Atrial flutter
**Atrial flutter**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Atrial frequency</td>
<td>250-350 bpm</td>
</tr>
<tr>
<td>Ventricular frequency</td>
<td>75-150 bpm (3:1 or 2:1 block)</td>
</tr>
<tr>
<td>Regularity</td>
<td><strong>Regular</strong></td>
</tr>
<tr>
<td>Origin</td>
<td>Atria (SVT)</td>
</tr>
<tr>
<td>P-wave</td>
<td>Negative sawtooth in lead II</td>
</tr>
<tr>
<td>Effect of Adenosine</td>
<td>Temporary reduced AV conduction</td>
</tr>
</tbody>
</table>

![ECG waveform](image)
Atrial flutter
AV-nodal re-entry tachycardia (AVNRT)
<table>
<thead>
<tr>
<th>Description</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial frequency</td>
<td>180-250 bpm</td>
</tr>
<tr>
<td>Ventricular frequency</td>
<td>180-250 bpm</td>
</tr>
<tr>
<td>Regularity</td>
<td>Regular</td>
</tr>
<tr>
<td>Origin</td>
<td>AV-node</td>
</tr>
<tr>
<td>P-wave</td>
<td>Inside or right after QRS-complex</td>
</tr>
<tr>
<td>Effect of Adenosine</td>
<td>Terminates arrhythmia</td>
</tr>
</tbody>
</table>

AVNRT
AVNRT
Atrioventricular Re-entrant Tachycardia (AVRT)

• Also called "Wolf-Parkinson-White-syndrome" (WPS) and is a part of "Preexitation syndromes”

• Caused by an abnormal accessory conduction pathway between atria and ventricles

• Ventricles might be stimulated prematurely, resulting in an atrioventricular re-entry tachycardia

• Can also cause diffuse ECG-changes resembling ischaemia
Wolf-Parkinson-White Syndrome
Wolf-Parkinson-White syndrome
Ventricular tachycardias

Premature ventricular contractions (PVC)

• Most common of ventricular arrhythmias
Ventricular tachycardia (VT)
• Regular, HR 110-250

Ventricular fibrillation (VF)
• Irregular, HR 400-600 bpm

Torsade de pointes
• Regular, HR 150-300
Bradyarrhythmias

- AV-block 1st degree
- AV-block 2nd degree (Wenckebach and Mobitz type II)
- AV-block 3rd degree (complete AV-block)
- AV-blocks
- Sick sinus syndrome
AV-blocks

Incomplete
- Delay
- Mobitz I
- Mobitz II

Complete
- Dropped beats
- High Grade
- Third degree or Complete
• AV-block type 1

• AV-block type 2 – Mobitz I (Wencheback)

• AV-block type 2 – Mobitz II
• AV-block type III (complete block)
• No relation between P-waves and QRS complexes
• Atrial rhythm 60-100 bpm (or AF)
• Ventricular rhythm might be nodal, ventricular or absent
Conduction system

- AV node
- SA node
- Internodal pathways
- Right bundle branch
- Purkinje fibers
- Bundle of His
- Left bundle branch
- Left posterior fascicle
- Left anterior fascicle
Left bundle branch block (LBBB)

- Slowed conduction in left bundle, causing delayed depolarization of left ventricle
- QRS > 0.12 sec
- Deep S-waves in V1-V3
- Late R-waves in V5-V6
- ST-segment depression in lateral leads (I, aVL, V5-V6)
- Always a pathological finding in patients!
Right bundle branch block (RBBB)

- Conduction in right bundle branch is slow, causing late depolarization of right ventricle
- QRS > 0.12 sec
- rSR ("rabbit ears") in V1-V2
- Late deep S-waves in lateral leads (I, aVL, V5-V6)
- Commonly a physiological finding in patients!
# Myocardial ischemia and infarction

<table>
<thead>
<tr>
<th>Time from onset of symptoms</th>
<th>ECG</th>
<th>Changes in the heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>minutes</td>
<td>hyperacute T waves (tall T waves), ST-elevation</td>
<td>reversible ischemic damage</td>
</tr>
<tr>
<td>hours</td>
<td>ST-elevation, with terminal negative T waves, negative T waves (these can last for days to months)</td>
<td>onset of myocardial necrosis</td>
</tr>
<tr>
<td>days</td>
<td>Pathologic Q Waves</td>
<td>scar formation</td>
</tr>
</tbody>
</table>
Evolution of ECG changes in Myocardial infarction
Diagnosis of myocardial infarction

- Elevated cardiac enzymes in blood (Troponin T, C and CKMB)

AND one of the following:

- Typical symptoms (chest pain > 20 min)
- ECG changes (ST elevation, ST depression or pathological Q-waves)
ECG-changes

ST-elevation (STEMI)
- Men > 0.2 mV in V2-V3, and/or > 0.1 mV in other leads
- Women > 0.1 mV in two or more leads

ST-depression
- New horizontal or downsloping ST-depression > 0.05 mV in two contiguous leads

T-wave inversion
- > 0.1 mV in two contiguous leads

Q-wave (old infarction, develops after hours/days)

New LBBB!!
Location of ECG-changes
Location of ECG-changes
Where is MI located?
Where is MI located?
PAUSE!
How to read ECG

"5+1":

1. Rhythm
2. Rate
3. Conduction (PQ, QRS, QT)
4. Heart axis
5. Morphology (P wave, QRS, ST-segment)
6. Compare current ECG with a previous one
1. Ryhtm

- Is it sinus rhythm?
- Regular or irregular?
- Prolonged recording from one lead is used to provide a rhythm strip (usually lead II)
2. Heart rate

- Determine the time between two QRS complexes (RR)
- IF paper speed is 25 mm/second, count number of big squares and divide with 300 (only in regular ryhtm)
- If paperspeed is 50 mm/second, divide with 600 (only in regular ryhtm)
- "ECG-rulers"
3. Conduction

- **PQ interval** starts at the beginning of the atrial contraction and ends at the beginning of the ventricular contraction (0.12 – 0.20 seconds)
- The **QRS duration** indicates how fast the ventricles depolarize (normal < 0.10 seconds)
- The **normal QTc** (corrected) interval indicates how fast the ventricles are repolarized, becoming ready for a new cycle. (below 0.45 seconds in men and below 0.46 in women)
4. Heart axis

- Look at lead aVF and I, should normally be positive.
- Lead II is also positive, lead III can be positive or negative.
- In normal axis both has predominant positive deflections.
5. Morphology

- Normal p wave?
- No pathological Q-waves?
- Prolonged QRS?
- Normal R-wave progression in V1-V6?
- ST elevation or depression?
- Abnormal T-wave?
6. Compare to old ECG

- Are the presenting ECG-changes new?
- Remember that new LBBB is treated as STEMI.
For more ECG traces click:

http://ecg.bidmc.harvard.edu/maven/maven/main.asp