ECG ABNORMALITIES

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• When we interpret an ECG we compare it instantaneously with the normal ECG and normal variants stored in our memory; these memories are stored visually in the posterior parts of the cerebrum and intellectually in the frontal parts. If these reservoirs contain only question marks, we are as helpless and lost in our interpretation as someone wandering through a snowstorm without a compass. It is important therefore to fill, or reactivate, these reservoirs.
P wave Abnormalities

- Right atrial enlargement results in a P wave that is peaked and narrower than usual called P Pulmonale.
P wave Abnormalities

- Left atrial enlargement results in a notched P wave with prolonged duration called P Mitrale
QRS Abnormalities

- Increased QRS width:
  - Cardiac hypertrophy or dilatation
  - Bundle branch block (QRS > 0.12)
High voltage:

- Cardiac hypertrophy

Low voltage:

- Old myocardial infarctions
- Pericardial effusion
Pathological Q wave

Q waves are considered pathological if:

- > 0.04 sec
- > 2 mm deep
- > 25% of depth of QRS complex
- Seen in V1 or V2

Pathological Q waves usually indicate ongoing or prior myocardial infarction.
T wave abnormalities

- T wave inversion:
  1. Mild ischemia
  2. Ventricular hypertrophy
  3. Bundle Branch Block
  4. Digoxin Toxicity
  5. Normal finding in aVR, V1, & lead III sometimes
• Peaked & tall T waves:
  1. Early stages of MI
  2. Hyperkalemia
  3. Normal finding

• Flattened T Wave
  1. Hypokalemia
  2. Ischemia
RIGHT VENTRICULAR HYPERTROPHY

- Best seen in the right ventricular leads (especially V1).
- The height of the R wave exceeds the depth of the S wave in V1.
- Deep S wave in lead V6.
- Right axis deviation.
- In severe cases inversion of the T waves in leads V1 and V2, and sometimes in lead V3 or V4.
- P pulmonale.
LEFT VENTRICULAR HYPERTROPHY

- A tall R wave (greater than 25 mm) in lead V5 or V6
- A deep S wave in lead V1 or V2
- With significant hypertrophy, inverted T waves are seen in leads I, aVL, V5 and V6.
- Left axis deviation occasionally occurs

✓ It is difficult to diagnose minor degrees of left ventricular hypertrophy from the ECG.
Arrhythmias

- An abnormal heart rhythm, the abnormality occurs in the rate or the regularity of heart beat due to abnormalities in the cardiac excitatory and conductive system.
Abnormal sinus Rhythm

- Tachycardia
- Bradycardia
- Sinus Arrhythmia
Tachycardia

- Fast heart rate above 100 beats per minute (B.P.M)
- Causes:
  - Fever
  - Sympathetic stimulation
Bradycardia

- Slow heart rate below 60 B.P.M
- Seen normally in athletes
- Vagal stimulation can trigger bradycardia
Sinus Arrhythmia

- The heart rate increases with inspiration & decreases with expiration this is especially evident in young people
- If the deviation exceeds 15% it is sinus arrhythmia

Note
- One P wave per QRS complex
- Constant PR interval
- Progressive beat-to-beat change in the R-R interval
Atrioventricular Block

- Results from conditions that can either decrease the rate of impulse conduction in the AV bundle or block the impulse entirely. Like Ischemia or compression of the AV bundle by a scar tissue.

- First degree block
- Second degree Block
- Third degree Block
- Stokes- Adams Syndrome
First Degree Heart Block

• when the PR interval increases to greater than 0.20 second, the P-R interval is said to be prolonged and the patient is said to have first-degree incomplete heart block.
Second Degree Block

- Occurs when the P-R interval length is 0.25 - 0.45 second, the action potential is sometimes strong enough to pass through the bundle into the ventricles and sometimes not strong enough.
- So there will be **dropped beats**; an atrial P wave but no QRS-T wave.

*Figure 13-6  Second degree A-V block, showing occasional failure of the ventricles to receive the excitatory signals (lead $V_3$).*
Third degree block

- Occurs with complete block of the impulse from the atria into the ventricles.

- The ventricles spontaneously establish their own signal, usually originating in the AV node or AV bundle. Therefore, the P waves become dissociated from the QRS-T complexes.

- In this figure the atrial rate is 115 B.P.M, whereas the ventricular rate is 48 B.P.M

Figure 13-7  Complete A-V block (lead II).
Stokes-Adams Syndrome

- The total block comes and goes
- The duration of block may be a few seconds, a few minutes, a few hours, or even weeks
- Overdrive suppression prevents the ventricles from beating until after a delay of 5 to 30 seconds.
Atrial fibrillation

- The normal regular electrical impulses generated by the SA node are overridden by disorganized electrical impulses usually originating in the roots of the pulmonary veins.
- On the ECG either no P waves are seen or only a fine, high frequency, very low voltage wavy record. The QRS-T complexes are normal in shape but are irregular.
Atrial flutter

• The electrical signal travels along a circular pathway within the right atrium, causing the atria to beat faster than the ventricles.
• Atrial flutter causes a rapid rate of contraction of the atria, usually between 200 and 350 B.P.M.
• P waves are strong.
• QRS-T complex follows an atrial P wave only once for every two to three beats of the atria, giving a 2:1 or 3:1 rhythm.
Figure 13-20 Pathways of impulses in atrial flutter and atrial fibrillation.
Ventricular Fibrillation

- The most serious of all cardiac arrhythmias, if not stopped within 1 to 3 minutes, is almost invariably fatal.
- The ventricular muscle contraction is not coordinated. So no pumping of blood occurs.
- ECG is bizarre and shows no regular rhythm of any type.
- Voltages of the waves in the ECG are usually about 0.5 millivolt when ventricular fibrillation first begins, but they decay rapidly.
Figure 13-17  Ventricular fibrillation (lead II).
ECG changes seen in MI

- ECG is very useful for diagnosing MI and locating areas of infarction.
- ST segment elevation occurs in the leads corresponding to the part of the heart that is damaged:
  - Leads V1-V4 with anterior wall infarction,
  - Lead aVL, I, V5 & V6 with lateral wall infarction
  - Leads II, III and aVF with inferior wall infarction.
• To be considered significant, more than 1 mm of ST segment elevation in at least two contiguous limb leads (e.g. I and VL; III and VF), or more than 2 mm of ST segment elevation in at least two contiguous precordial leads

• Within a day or so, the ST segments return to the baseline, the T waves in the affected leads become inverted, and Q waves develop. These ECG changes are usually permanent
Development of inferior infarction

1 h after onset of pain:

<table>
<thead>
<tr>
<th>I</th>
<th>II</th>
<th>III</th>
<th>VR</th>
<th>VL</th>
<th>VF</th>
</tr>
</thead>
</table>

6 h after onset of pain:

<table>
<thead>
<tr>
<th>I</th>
<th>II</th>
<th>III</th>
<th>VR</th>
<th>VL</th>
<th>VF</th>
</tr>
</thead>
</table>

24 h after onset of pain:

<table>
<thead>
<tr>
<th>I</th>
<th>II</th>
<th>III</th>
<th>VR</th>
<th>VL</th>
<th>VF</th>
</tr>
</thead>
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Note

- Three ECGs have been recorded over 24 h, and have been arranged horizontally
- Sinus rhythm with a normal cardiac axis in all three ECGs
- The first record is essentially normal
- 6 h after the onset of pain, the ST segments have risen in leads II, III and VF, and the ST segment is depressed in leads I, VR and VL. A Q wave has developed in lead III
- 24 h after the onset of pain, a small Q wave has appeared in lead II, and more obvious Q waves can be seen in leads III and VF. The ST segments have returned to the baseline, and the T waves are now inverted in leads III and VF
Abnormal ECGs
- P waves are replaced by rapidly quivering small deflection of variable amplitude
- Irregularly irregular ventricular rate
- Narrow QRS complexes

- Atrial Fibrillation
- Sinus rhythm, rate 63/min
- Right axis deviation (deep S waves in lead I)
- Dominant R waves in lead $V_1$
- Deep S waves in lead V (clockwise rotation)
- Inverted T waves in leads II, III, VF and $V_1$–$V_3$
- Flat T waves in leads $V_4$–$V_5$

Severe right ventricular hypertrophy
• Sinus rhythm, rate 83/min
• Normal axis
• Tall R waves in leads V₅–V₆ (R wave in lead V₅, 40 mm) and deep S waves in leads V₁–V₂
• Inverted T waves in leads I, VL and V₅–V₆

Left ventricular hypertrophy
• Sinus rhythm, rate 70/min
• Normal axis
• Q waves in leads III and VF
• Normal QRS complexes
• Raised ST segments in leads II, III and VF
• Inverted T waves in lead VL (abnormal) and in lead V₁ (normal)

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**Acute inferior infarction**
- Sinus rhythm, rate 60/min
- Normal axis
- Normal QRS complexes
- ST segments depressed horizontally in leads $V_3 - V_5$
- Normal T waves

ST segment depression in unstable angina
- Sinus rhythm, rate 75/min
- Normal axis
- Normal QRS complexes
- ST segments elevated in leads $V_1 - V_5$
- Normal T waves

ST segment elevation in acute anterior myocardial infarction
- HR = 83 b.p.m
- Left axis deviation (-71)
- QRS complex has low voltage
- Pathological Q waves in leads II & III
- Inverted T waves in leads II & III

- **Old inferior MI**
• Atrial rate is 100 b.p.m & ventricular rate is 37.5 b.p.m
• Right axis deviation (105 degrees)
• Prolonged QRS
• Loss of progression of QRS in chest leads
• Inverted T wave in v1-v4

• 3rd Degree AV Block
• Acute anterior MI
- Sinus tachycardia
- Right axis deviation
- P pulmonale (in the inferior leads)
- Loss of QRS progression in chest leads
- Low QRS voltage.
- Slight ST segment depressions in the aVF & V3

- **Right bundle branch block**
Second degree heart block
- Sinus rhythm, rate 47/min
- Normal QRS complexes, ST segments and T waves

**Sinus bradycardia in an athlete**
- Sinus rhythm, rate 80/min
- PR interval prolonged at 336 ms
- Constant PR interval in all beats
- Loss of the R wave in lead V₃ could indicate an old anterior infarction, otherwise QRS complexes, ST segments and T waves are normal

First degree heart block
THANK YOU

BEST OF LUCK