POCKET GUIDE TO ECG INTERPRETATION

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Methodological ECG Interpretation

The ECG must always be interpreted systematically. Failure to perform a systematic interpretation of the ECG may be detrimental. The interpretation algorithm presented below is easy to follow and it can be carried out by anyone. The reader will gradually notice that ECG interpretation is markedly facilitated by using an algorithm, as it minimizes the risk of missing important abnormalities and also speeds up the interpretation.

1. Rhythm

<table>
<thead>
<tr>
<th>ASSESSMENTS</th>
<th>EVALUATION</th>
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<tbody>
<tr>
<td>Assess ventricular (RR intervals) and atrial (PP intervals) rate and rhythm.</td>
<td>♥ Sinus rhythm (which is the normal rhythm) has the following characteristics: (1) heart rate 50–100 beats per minute; (2) P-wave precedes every QRS complex; (3) the P-wave is positive in lead II and (4) the PR interval is constant.</td>
</tr>
<tr>
<td>♥ Is ventricular rhythm regular? What is the ventricular rate (beats/min)?</td>
<td>♥ Causes of bradycardia: sinus bradycardia, sinoatrial block, sinoatrial arrest/inhibition, second-degree AV block, third-degree AV block. Note that escape rhythms may arise during bradycardia. Also note that bradycardia due to dysfunction in the sinoatrial node is referred to as sinus node dysfunction (SND). If a person with ECG signs of SND is symptomatic, the condition is classified as sick sinus syndrome (SSS).</td>
</tr>
<tr>
<td>♥ Is atrial rhythm regular? What is the atrial rate (beats/min)?</td>
<td>♥ Causes of tachycardia (tachyarrhythmia) with narrow QRS complexes (QRS duration &lt;0,12 s): sinus tachycardia, inappropriate sinus tachycardia, sinoatrial re-entry tachycardia, atrial fibrillation, atrial flutter, atrial tachycardia, multifocal atrial tachycardia, AVNRT, AVRT (pre-excitation, WPW). Note that narrow complex tachyarrhythmia rarely causes circulatory compromise or collapse.</td>
</tr>
<tr>
<td>♥ P-waves should precede every QRS complex and the P-wave should be positive in lead II.</td>
<td>♥ Causes of tachycardia (tachyarrhythmia) with wide QRS complexes (QRS duration ≥0,12 s): ventricular tachycardia is the most common cause and it is potentially life-threatening. Note that 10% of wide complex tachycardias actually originate from the atria but the QRS complexes become wide due to abnormal ventricular depolarization (e.g. sinus tachycardia with simultaneous left bundle branch block).</td>
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</table>

2. P-wave and PR interval

<table>
<thead>
<tr>
<th>ASSESSMENTS</th>
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</tr>
</thead>
<tbody>
<tr>
<td>♥ P-wave always positive in lead II (actually always positive in leads II, III and aVF).</td>
<td>♥ P-wave must be positive in lead II, otherwise the rhythm cannot be sinus rhythm.</td>
</tr>
<tr>
<td>♥ P-wave duration should be &lt;0,12 s (all leads).</td>
<td>♥ P-wave may be biphasic (diphasic) in V1 (the negative deflection should be &lt;1 mm). It may have a prominent second hump in the inferior limb leads (particularly lead II).</td>
</tr>
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</table>
**3. QRS complex**

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<tr>
<th>ASSESSMENTS</th>
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<tbody>
<tr>
<td>♥ There must be at least one limb lead with R-wave amplitude &gt;5 mm and at least one chest (precordial) lead with R-wave amplitude &gt;10 mm; otherwise there is low voltage.</td>
<td>♥ Short QRS duration: no clinical relevance.</td>
</tr>
<tr>
<td>♥ High voltage exists if the amplitudes are too high, i.e if the following condition is satisfied: S-waveV1 or V2 + R-waveV5 &gt;35 mm.</td>
<td>♥ High voltage: Hypertrophy (any lead). Left bundle branch block (leads V5, V6, I, aVL). Right bundle branch block (V1–V3). Normal variant in younger, well-trained and slender individuals.</td>
</tr>
</tbody>
</table>
Is the electrical axis normal? Electrical axis is assessed in limb leads and should be between –30° to 90°.

| ♥ Fragmented QRS complexes indicates myocardial scarring (mostly due to infarction). |
| ♥ Right axis deviation: Normal in newborns. Right ventricular hypertrophy. Acute cor pulmonale (pulmonary embolism). Chronic cor pulmonale (COPD, pulmonary hypertension, pulmonary valve stenosis). Lateral ventricular infarction. Pre-excitation. Switched arm electrodes (negative P and QRS-T in lead I). Situs inversus. Left posterior fascicular block is diagnosed when the axis is between 90° and 180° with rS complex in I and aVL as well as qR complex in III and aVF (with QRS duration <0.12 seconds), provided that other causes of right axis deviation have been excluded. |
| ♥ Left axis deviation: Left bundle branch block. Left ventricular hypertrophy. Inferior infarction. Pre-excitation. Left anterior fascicular block is diagnosed if the axis is between -45° and 90° with qR-complex in aVL and QRS duration is 0.12 s, provided that other causes of left axis deviation have been excluded. |
| ♥ Extreme axis deviation: Rarely seen. Probably misplaced electrodes. If the rhythm is wide QRS complex tachycardia, then the cause is probably ventricular tachycardia. |

4. ST segment

<table>
<thead>
<tr>
<th>ASSESSMENTS</th>
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<tbody>
<tr>
<td>♥ The ST-segment should be flat and isoelectric (in level with the baseline). It may be slightly upsloping at the transition with the T-wave.</td>
<td>♥ Benign ST segment elevation is very common in the population, particularly in the precordial leads (V2–V6). Up to 90% (in some age-ranges) of healthy men and women display concave ST-segment elevations in V2–V6 (this is called male/female pattern). ST-segment elevations which are not benign nor due to ischemia are rather common (listed below).</td>
</tr>
<tr>
<td>♥ ST segment deviation (elevation and depression) is measured in the J point.</td>
<td>♥ ST-segment depression is uncommon among healthy individuals. ST-segment depression is particularly suspicious in the chest leads. Guidelines recommend that &lt;0.5 mm ST-segment depression be accepted in all leads.</td>
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</table>
5. T-wave

<table>
<thead>
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<tbody>
<tr>
<td>♥ Should be concordant with the QRS complex. Should be positive in most leads.</td>
<td>♥ Normal variants: An isolated (single) T-wave inversion is accepted in lead V1 and lead III. In some instances the T-wave inversions from childhood may persist in V1–V3(V4), which is called persistent juvenile T-wave pattern. Rarely, all T-waves remain inverted, which is called global idiopathic T-wave inversion (V1–V6).</td>
</tr>
<tr>
<td>♥ T-wave progression should be normal in chest leads.</td>
<td>♥ T-wave inversion without simultaneous ST-segment deviation: This is not a sign of ongoing ischemia, but may be post-ischemic. One type of post-ischemic T-wave inversion is especially acute, namely Wellen's syndrome (characterized by deep T-wave inversions in V1–V6 in patient with recent episodes of chest pain). Cerebrovascular insult (bleeding). Pulmonary embolism. Perimyocarditis (after normalization of the ST-segment elevation, T-waves become inverted in perimyocarditis). Cardiomyopathy.</td>
</tr>
<tr>
<td>♥ In limb leads the amplitude is highest in lead II, and in the chest leads the amplitude is highest in V2–V3.</td>
<td>♥ T-wave inversion with simultaneous ST-segment deviation: acute (ongoing) myocardial ischaemia.</td>
</tr>
<tr>
<td></td>
<td>♥ High T-waves: Normal variant. Early repolarization. Hyperkalemia. Left ventricular hypertrophy. Left bundle branch block. Occasionally perimyocarditis. High (hyperacute) T-waves may be seen in the very early phase of STEMI.</td>
</tr>
</tbody>
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6. QTc interval and U-wave

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<thead>
<tr>
<th>ASSESSMENTS</th>
<th>EVALUATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>♥ QTc duration men ≤0.45 s.</td>
<td>♥ Acquired QT prolongation: anti arrhythmic drugs (procainamide, disopyramide, amiodarone, sotalol), psychotropic medications (tricyclic antidepressants, SSRI, lithium etc); antibiotics (macrolides, kinolones, atovaquone, klorokine, amantadine, foscarnet, atazanavir); hypokalemia, hypocalcemia, hypomagnesemia; cerebrovascular insult (bleeding); myocardial ischemia; cardiomyopathy; bradycardia; hypothroidism; hypothermia. A complete list of drugs causing QT prolongation can be found here.</td>
</tr>
<tr>
<td>♥ QTc duration women ≤0.46 s.</td>
<td>♥ Congenital QT prolongation: genetic disease of which there are approximately 15 variants.</td>
</tr>
<tr>
<td>♥ Prolonged QTc duration may cause malignant arrhythmias (torsade de pointes, which is a type of ventricular tachycardia).</td>
<td>♥ Short QTc syndrome (≤0.32 s): caused by hyperkalemia and digoxin treatment. May cause malignant ventricular arrhythmia.</td>
</tr>
<tr>
<td>♥ Shortened QTc duration (≤0.32 s) is rare, but may also cause malignant ventricular arrhythmias.</td>
<td>♥ Negative U-wave: high specificity for heart disease (including ischemia).</td>
</tr>
<tr>
<td>♥ The U-wave is seen occasionally, especially in well-trained individuals, and during low heart rate. It is largest in V3–V4. Amplitude is one fourth of T-wave amplitude.</td>
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</tbody>
</table>

7. Compare with earlier ECG tracings

It is fundamental to compare the current ECG with previous recordings. All changes are of interest and may indicate pathology.

8. Clinical context

ECG changes should be put into a clinical context. For example, ST-segment elevations are common in the population and should not raise suspicion of myocardial ischemia if the patient do not have symptoms suggestive of ischemia.

_The guide continues on the next page._
The cardiac conduction system

Waves, intervals and durations on the ECG

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The walls of the left ventricle and the leads that view these walls

The ECG leads

A) The limb leads and their view of the heart’s electrical activity

Recall that each lead ‘views’ the heart from the angle of its positive (exploring) electrode.

The positive electrode is the exploring electrode. This is defined by the ECG machine.

As noted previously, it is recommended that lead aVR be inverted to lead –aVR, as this fills a gap in the coordinate system and thus facilitates interpretation of the ECG.

B) Einthoven’s triangle
P-wave changes

Contour of the normal P wave

The P-wave is always positive in lead II if the rhythm is sinus rhythm. The P-wave may, however, display two humps, as shown here. This is due to the fact that the atria are not depolarized (activated) simultaneously.

The P-wave in lead V1 may be biphasic, due to the negative deflection caused by depolarization of the left atrium (the electrical vector is directed away from V1).

Abnormal P-waves

<table>
<thead>
<tr>
<th>P-mitrale</th>
<th>P mitrale is a consequence of left atrial enlargement (often caused by mitral stenosis). Enlargement of the left atrium amplifies its contribution to the contour of the P wave.</th>
</tr>
</thead>
<tbody>
<tr>
<td>II</td>
<td>Enhanced second hump in lead II.</td>
</tr>
<tr>
<td>V1</td>
<td>Enhanced negative deflection in V1.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>P-pulmonale</th>
<th>P pulmonale is a consequence of right atrial enlargement. This is often a consequence of pulmonary valve stenosis or increased resistance in the pulmonary circulation. Enlargement of the right atrium causes an increased P wave amplitude in both leads.</th>
</tr>
</thead>
<tbody>
<tr>
<td>II</td>
<td>Increased P wave amplitude.</td>
</tr>
<tr>
<td>V1</td>
<td>Increased P wave amplitude.</td>
</tr>
</tbody>
</table>
ST segment depressions

A. Physiological ST-segment depressions

Upsloping ST segment

Upsloping ST-segment depression is a normal finding during physical exercise. It should be considered a normal finding, provided that T-waves are not inverted. Hyperventilation may cause similar ST-segment depressions.

B. Non specific ST-segment depression

Hypokalemia and high sympathetic tone causes ST-segment depressions with flat T-waves and more marked U-waves. High sympathetic tone also causes tachycardia.

Digoxin (a drug used to treat atrial fibrillation and some cases of heart failure) causes a curved ST-segment depressions.

C. ST-segment depressions caused by acute ischemia

Characteristics | Real life examples
---|---
Horizontal | Horizontal depression with distinct ST-segment.
Downsloping | Down sloping with positive T-wave.

Very typical of ischemia. Typical of ischemia.

Horizontal depression with short ST-segment.

Downsloping with inverted T-wave.

Horizontal depression with short ST-segment.

Note: When considering myocardial ischemia, deviations in the ST-segment always indicates ongoing ischemia. ST-segment deviation may be accompanied by T-wave changes, but it is the ST-deviation that indicates acute ischemia.

de Winter’s sign

de Winter’s sign is an exception to the rule that upsloping ST-segment depressions are not ischemic. de Winter’s sign implies the presence of upsloping ST-segment depressions with prominent T-waves in the majority of the precordial (chest) leads. This is a sign of acute ischemia, most often caused by a proximal occlusion of the left anterior descending (LAD) artery.

D. Secondary repolarization abnormalities (secondary ST- and T-wave changes)

Left bundle branch block (lead V6) | Left ventricular hypertrophy (lead V6) | Right bundle branch block (lead V1) | Pre-excitation (delta wave)

Right ventricular hypertrophy

Large R-waves and ST-segment depressions in V1–V3. In case of chest discomfort, one must consider possibility of posterolateral transmural ischemia as a differential diagnosis.
ST segment elevations

A. Characteristics of ST-segment elevations caused by ischemia

Convex | Straight upsloping | Straight horizontal | Straight downsloping

ST-segment elevations caused by ischemia typically display a convex or straight ST-segment. Such ST-segment elevations in presence of chest discomfort are strongly suggestive of transmural myocardial ischemia. Note that the straight downsloping variant is unusual.

B. Typical non-ischemic ST-segment elevation

Concave

Non-ischemic ST-segment elevations are extremely common in all populations. They are characterized by a concave ST-segment and a greater distance between the J point and the T wave apex.

C. Examples of ST-segment elevations caused by ischemia

ST-segment elevation can vary markedly in appearance. These six examples were retrieved from six different patients with STEMI.

D. Real life example (limb leads shown)

ECG from a male patient (age 51) who experienced chest pain while driving to work. Note ST-segment elevations as well as reciprocal ST-segment depressions. There are also pathological Q-waves (leads III, aVF and perhaps II).
T-wave changes

A Normal T-waves

Normal T wave
Smooth transition from ST-segment to T wave. T wave is slightly asymmetric with a steeper downslope.

Normal variant
Large, asymmetric T wave with broad base. Often in conjunction with slight J point elevation in leads V2-V4.

B Large T-waves

Hyperkalemia
Large, symmetric, pointed with short base.

Hypersensitive T wave
can be seen in transmural ischemia. High, broad based, symmetric, not pointed. Almost always seen in conjunction with ST-segment elevation.

C Biphasic (diphasic) T-waves

Both these T waves are negative (inverted) since the terminal portions are negative.

This T wave is positive by definition since the terminal portion is positive.

Wherever spotting a biphasic T wave, try to determine whether it is actually a positive or negative (inverted) T-wave by viewing the terminal portion of the T wave.

D Negative (inverted) T-waves

Post-ischemic
Symmetric T wave, with varying depth. Ranges from flat T wave to very deep T wave inversion. Inverted T waves do not equate acute (ongoing) ischemia, but rather appear after an episode of ischemia.

Acute (ongoing) ischemia
T wave inversion with simultaneous ST-segment deviation (most commonly ST-depression). Note that it is the ST-segment deviation that represents the acute ischemia.

Cerebrovascular insult
Pattern
Very deep (gigantic) T wave inversions in the chest leads. Some studies report this finding in up to 30% of patients with intracerebral hemorrhage.

Hypertrophic cardiomyopathy
Symmetric T wave inversions, most commonly in V1–V3. Often very deep and accompanied by large R waves. Occasionally accompanied by ST-segment depression.

PERIMYOKARDIT
T wave inversions occur after normalization of ST-segment elevations in perimyocarditis. T wave inversions often seen in most leads.
As evident from the figure above, the normal heart axis is between −30° and 90°. If the axis is more positive than 90° it is referred to as right axis deviation. If the axis is more negative than −30° it is referred to as left axis deviation. The axis is calculated (to the nearest degree) by the ECG machine. The axis can also be approximated manually by judging the net direction of the QRS complex in leads I and II. The following rules apply:

- Normal axis: Net positive QRS complex in leads I and II.
- Right axis deviation: Net negative QRS complex in lead I but positive in lead II.
- Left axis deviation: Net positive QRS complex in lead I but negative in lead II.
- Extreme axis deviation (−90° to 180°): Net negative QRS complex in leads I and II.
Pro-arrhythmic ECG changes during sinus rhythm

- **Q-waves or fragmented QRS complexes**: Evidence of previous myocardial infarction and high risk of ventricular tachycardia and ventricular fibrillation.

- **Delta wave, pre-excitation**: Risk of AVRT. May be accompanied by atrial fibrillation (pre-excited atrial fibrillation).

- **Brugada syndrome: ST elevation V1–V4, Shark tail appearance or saddle formed**: Risk of ventricular tachycardia and ventricular fibrillation.

- **Early repolarization: slurring or notching at end-QRS**: 5 times increased risk of ventricular tachycardia and ventricular fibrillation.

- **Hypertrophic cardiomyopathy: deep S, large R**: Risk of ventricular tachycardia and ventricular fibrillation.

- **Digoxin effect: ST depression with “sagging” appearance**: Digoxin may cause virtually all known arrhythmias.

- **P-pulmonale (right atrial abnormality) and P-mitrale (left atrial abnormality)**: Associated with atrial tachyarrhythmias.

- **Arrhythmogenic right ventricular cardiomyopathy: rSr-pattern in V1 with epsilon wave after QRS**: Risk of ventricular tachycardia and ventricular fibrillation.

- **Long QT interval (LQTS) or Short QT interval (SQTS)**: Long QT syndrome is fairly common and poses risk of polymorphic ventricular tachycardia referred to as torsade de pointes. Short QT syndrome is very rare but may also cause torsade de pointes.
Assessment of RP interval for tachyarrhythmias

<table>
<thead>
<tr>
<th>RP interval</th>
<th>Short and &lt;70 ms</th>
<th>Typical AVNRT, AVRT is unusual.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td><img src="waveform1.png" alt="ECG Waveform" /></td>
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<table>
<thead>
<tr>
<th>RP interval</th>
<th>No visible P-wave</th>
<th>Typical AVNRT.</th>
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<tbody>
<tr>
<td></td>
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<td><img src="waveform2.png" alt="ECG Waveform" /></td>
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</table>

If the P-wave is invisible, it is classified as short RP interval.

<table>
<thead>
<tr>
<th>RP interval</th>
<th>Short but &gt;70 ms</th>
<th>In most cases AVRT. Occasionally atypical AVNRT or AT.</th>
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<td></td>
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<td><img src="waveform3.png" alt="ECG Waveform" /></td>
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<table>
<thead>
<tr>
<th>RP interval</th>
<th>Long</th>
<th>In most cases AT. Occasionally atypical AVNRT. Rarely PJRT.</th>
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<td><img src="waveform4.png" alt="ECG Waveform" /></td>
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Diagnosis and management of tachyarrhythmias with narrow QRS complex
Diagnosis and management of tachyarrhythmias with wide QRS complex

INITIAL MANAGEMENT

Wide complex tachycardia

Hemodynamically unstable

- Immediate electrical or pharmacological cardioversion
- Presence of heart disease
- Most likely VT

Hemodynamically stable

- Stable
- Unstable
- Regular
- Irregular
- No heart disease

Electrical conversion or intravenous procainamide, lidocaine or amiodarone.

Persistant or accelerated tachycardia

Adenosine and/or vagus stimulation

SVT with BBB

Antihemodynamic AVRT

Idiopathic VT

AF with BBB

Pre-excited AF/AFL/AT

AF with BBB and varying AV-block

At with BBB and varying AV-block

VT = Ventricular tachycardia

BBB = Bundle branch block

AF = Atrial fibrillation

AT = Atrial tachycardia

AFL = Atrial flutter

ECG INTERPRETATION

MORPHOLOGICAL CRITERIA

If QRS morphology is very typical of RBBB/LBBB, consider a diagnosis of SVT with BBB. Also assess the QRS morphology as follows:

Tachycardia with RBBB pattern (QRS positive in V1-V2)

- V1: Monomorphic R complex or qR complex strongly suggest VT. R > R′ suggests VT.
- QRS complexes (qR′, qR′′, qR′′′) suggest SVT
- V6: Rs, QS or R complexes suggest VT
- Rs complex suggest SVT
- Electrical axis from +90° to -90° suggests VT

Tachycardia with LBBB pattern (QRS negative in V1-V2)

- V1: Initial portion of QRS is smooth (up- or downwards) in VT. Aberrantly conducted beats have sharp start of QRS
- R-wave duration >60 ms suggest VT
- RS interval ≥60 ms suggest VT
- V6: QR or QS complex suggest VT.
- R and R′ complex without initial q-wave suggest SVT

Assess association between atrial and ventricular activity

- 1:1 association?
- Yes/uncertain
- No

Atrial rate > Ventricular rate: atrial flutter, atrial tachycardia.

BRUGADA ALGORITHM (SN 89%; SP 59%)

- Absence of RS in V1-V6 (concordance)
- Yes: VT (SN 22%, SP 100%)
- No
- RS interval >100 ms and R wider than S in any of V1-V6
- Yes: VT (SN 66%, SP 98%)
- No
- AV dissociation?
- Yes: VT (SN 82%, SP 98%)
- No: SVT (SN 95%, SP 98%)

SN = sensitivity. SP = specificity.

SVT = Supraventricular tachycardia.

RBBB/LBBB = Right/left bundle branch block.

BBB = Bundle branch block.

RS interval

Morphasic R qR complex R-R′
Intraventricular conduction defects

ECG changes and criteria in bundle branch blocks and fascicular blocks

**Right bundle branch block**
- Activates the right ventricle
- QRS duration $>0.12$ s.
- Normal electrical axis.
- V1-V2 shows rs', rSr or rSr'.
- V5, V6, I and aVF shows broad S-wave.
- Secondary ST-T changes.

**Anterior fascicular block**
- Activates the anterior wall
- QRS duration $<0.12$ s.
- Left axis deviation.
- qR complex in aVL.
- rS complex in II, III and aVF.
- V5-V6 usually show qR complex.

**Posterior fascicular block**
- Activates the posterior and inferior wall.
- QRS duration $<0.12$ s.
- Right axis deviation.
- rS complex in lead I and aVL.
- qR complex in inferior leads.
- Always q-wave in III and aVF.

**Left bundle branch block**
- Provides the intraventricular septum with Purkinje fibers. Divided into two fascicles which activates the left ventricle.

Note that both aVR and -aVR are shown.
**Hypertrophy and dilatation**

Use leads V1, V2, V5 and V6 to spot ventricular hypertrophy. These leads show characteristic QRS changes in hypertrophy.

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<thead>
<tr>
<th></th>
<th>V1/V2</th>
<th>V5/V6</th>
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<tbody>
<tr>
<td>Normal</td>
<td><img src="image1" alt="Normal ECG" /></td>
<td><img src="image2" alt="Normal ECG" /></td>
</tr>
<tr>
<td>Left ventricular</td>
<td><img src="image3" alt="Left ventricular hypertrophy ECG" /></td>
<td><img src="image4" alt="Left ventricular hypertrophy ECG" /></td>
</tr>
<tr>
<td>Right ventricular</td>
<td><img src="image5" alt="Right ventricular hypertrophy ECG" /></td>
<td><img src="image6" alt="Right ventricular hypertrophy ECG" /></td>
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</tbody>
</table>

- Typically convex ST segment with or without the septal q-wave.
- Less typical is this straight ST segment, with or without septal q-wave.
- RS complex
- qR complex
- rSR' pattern, similar to right bundle branch block
- R complex
Classification of acute coronary syndromes (ACS)

**Risk factors:** smoking, hypertension, dyslipidemia, diabetes, obesity, psychosocial stress, heredity, lack of physical activity, adverse diets etc.

- Inflammation in coronary arteries
- Atherosclerotic coronary arteries
- Plaque rupture/erosion

**Angina pectoris**
Normal ECG at rest. No symptoms at rest.

- Exercise stress test to provoke ECG changes
- ST-segment depressions

**Atherothrombosis leads to artery occlusion and ischemia.**
**Acute coronary syndrome!**

- Chest pain and/or associated symptoms

**ECG**

**NSTE-ACS**
- No significant ST-segment elevations.

**Pathophysiology**
Partial occlusion causing ischemia located to the subendocardium (black area in figure above).

**Diagnostic Criteria**
All acute coronary syndromes without significant ST-segment elevations are classified as NSTE-ACS. In most cases there are ST-segment depressions and/or T-wave inversions. A minority of patients with NSTE-ACS display normal ECG through the course.

- Elevated troponin
- Normal troponin levels
- Non-STEMI (NSTEMI)
- Unstable angina
- Most cases

- Q-wave infarction
- Non Q-wave infarction

**STE-ACS**
- Significant ST-segment elevations

**Pathophysiology**
Total occlusion in a coronary artery. This causes extensive ischemia which is transmural (i.e., stretches from the endocardium to the epicardium, see figure above). These infarctions are large and usually lead to development of pathological Q-waves in the leads which displayed ST-segment elevations.

- Elevated troponin
- Normal troponin levels
- STEMI
- Unstable angina
- ‘Aborted myocardial infarction’
- Most cases

- Q-wave infarction
- Non Q-wave infarction

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Criteria for acute myocardial infarction (AMI)

STE-ACS (STEMI) – ST elevation acute myocardial infarction

Criteria for STEMI New ST segment elevations in at least two anatomically contiguous leads:

- **Men age ≥40 years**: ≥2 mm in V2-V3 and ≥1 mm in all other leads.
- **Men age <40 years**: ≥2,5 mm in V2-V3 and ≥1 mm in all other leads.
- **Women (any age)**: ≥1,5 mm in V2-V3 and ≥1 mm in all other leads.
- **Men & women V4R and V3R**: ≥0,5 mm, except from men <30 years in whom the criteria is ≥1 mm.
- **Men & women V7-V9**: ≥0,5 mm.

NSTE-ACS (NSTE-ACS) – Non ST elevation acute myocardial infarction: NSTEMI and unstable angina

- New horizontal or downsloping ST segment depressions ≥0,5 mm in at least two anatomically contiguous leads.
- T wave inversion ≥1 mm in at least two anatomically contiguous leads. These leads must have evident R-waves, or R-waves larger than S-waves.