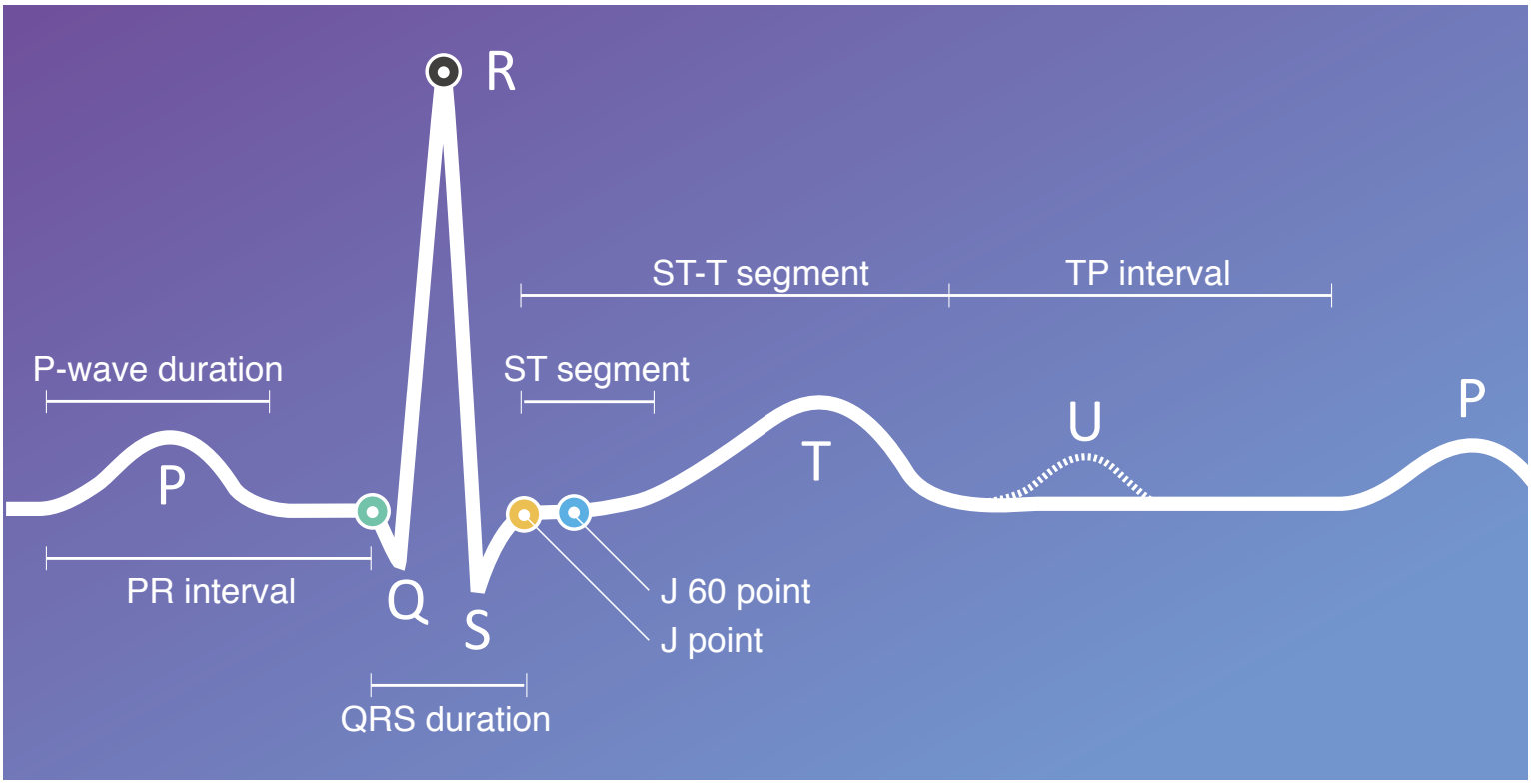

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POCKET GUIDE TO ECG INTERPRETATION

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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

ASSESSMENTS	EVALUATION
Assess ventricular (RR intervals) and atrial (PP intervals) rate and rhythm. ♥ Is ventricular rhythm regular? What is the ventricular rate (beats/min)? ♥ Is atrial rhythm regular? What is the atrial rate (beats/min)? ♥ P-waves should precede every QRS complex and the P-wave should be positive in lead II.	♥ 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. ♥ 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). ♥ Causes of tachycardia (tachyarrhythmia) with narrow QRS complexes (QRS duration <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. ♥ 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).

2. P-wave and PR interval

ASSESSMENTS	EVALUATION
♥ P-wave always positive in lead II (actually always positive in leads II, III and aVF). ♥ P-wave duration should be <0,12 s (all leads).	♥ P-wave must be positive in lead II, otherwise the rhythm cannot be sinus rhythm. ♥ P-wave may be biphasic (diphasic) in V1 (the negative deflection should be <1 mm). It may have a prominent second hump in the inferior limb leads (particularly lead II).

♥ P-wave amplitude should be ≤2,5 mm (all leads). PR interval must be 0,12–0,22 s (all leads).	♥ P mitrale: increased P-wave duration, enhanced second hump in lead II and enhanced negative deflection in V1. ♥ P pulmonale: increased P-wave amplitudes in lead II and V1. ♥ If P-wave not clearly visible: look for retrograde (inverted) P-waves, which can be located anywhere between the J point and the terminal part of the T-wave. ♥ PR interval >0,22 s: first-degree AV block. ♥ PR interval <0,12 s: Pre-excitation (WPW syndrome). ♥ Second-degree AV-block Mobitz type I (Wenckebach block): repeated cycles of gradually increasing PR interval until an atrial impulse (P-wave) is blocked in the atrioventricular node and the QRS complex does not appear. ♥ Second-degree AV-block Mobitz type II: intermittently blocked atrial impulses (no QRS seen after P) but with constant PR interval. ♥ Third-degree AV-block: All atrial impulses (P-waves) are blocked by the atrioventricular node. An escape rhythm arises (cardiac arrest ensues otherwise), which may have narrow or wide QRS complexes, depending on its origin. There is no relation between P-waves and the escape rhythm's QRS complexes, and atrial rhythm is typically faster than the escape rhythm (both rhythms are typically regular).

3. QRS complex

ASSESSMENTS	EVALUATION
♥ QRS duration must be <0,12 s (normally 0,07-0,10 s). ♥ There must be at least one limb lead with R-wave amplitude >5 mm and at least one chest (precordial) lead with R-wave amplitude >10 mm; otherwise there is low voltage. ♥ High voltage exists if the amplitudes are too high, i.e if the following condition is satisfied: S-waveV1 or V2 + R-waveV5 >35 mm. ♥ Look for pathological Q-waves. Pathological Q-waves are ≥0,03 s and/or amplitude ≥25% of R-wave amplitude in same lead, in at least 2 anatomically contiguous leads. ♥ Is the R-wave progression in the chest leads (V1–V6) normal?	♥ **Wide QRS complex (QRS duration ≥0.12 s)**: Left bundle branch block. Right bundle branch block. Nonspecific intraventricular conduction disturbance. Hyperkalemia. Class I antiarrhythmic drugs. Tricyclic antidepressants. Ventricular rhythms and ventricular extrasystoles (premature complexes). Artificial pacemaker which stimulates in the ventricle. Aberrant conduction (abberancy). Pre-excitation (Wolff-Parkinson-White syndrome). ♥ **Short QRS duration**: no clinical relevance. ♥ **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. ♥ **Low voltage**: Normal variant. Misplaced leads. Cardiomyopathy. Chronic obstructive pulmonary disease. Perimyocarditis. Hypothyreosis (typically accompanied by bradycardia). Pneumothorax. Extensive myocardial infarction. Obesity. Pericardial effusion. Pleural effusion. Amyloidosis. ♥ **Pathological Q-waves**: Myocardial infarction. Left-sided pneumothorax. Dextrocadia. Perimyocarditis. Cardiomyopathy. Amyloidosis. Bundle branch blocks. Anterior

♥ Is the electrical axis normal? Electrical axis is assessed in limb leads and should be between –30° to 90°.	fascicular block. Pre-excitation. Ventricular hypertrophy. Acute cor pulmonale. Myxoma. ♥ **Fragmented QRS complexes** indicates myocardial scarring (mostly due to infarction). ♥ **Abnormal R-wave progression**: Myocardial infarction. Right ventricular hypertrophy (reversed R-wave progression). Left ventricular hypertrophy (amplified R-wave progression). Cardiomyopathy. Chronic cor pulmonale. Left bundle branch block. Pre-excitation. ♥ **Dominant R-wave in V1/V2**: Misplaced chest electrodes. Normal variant. Situs inversus. Posterolateral infarction/ischemia (if patient experiences chest discomfort). Right ventricular hypertrophy. Hypertrophic cardiomyopathy. Right bundle branch block. Pre-excitation. ♥ **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

ASSESSMENTS	EVALUATION
♥ 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. ♥ ST segment deviation (elevation and depression) is measured in the J point.	♥ **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). ♥ ST-segment depression is uncommon among healthy individuals. ST-segment depression is particularly suspicious in the chest leads. Guidelines recommend that <0.5 mm ST-segment depression be accepted in all leads.

	♥ **Causes of ST-segment elevation**: Ischemia. ST segment elevation myocardial infarction (STEMI/STE-AKS). Prinzmetal's angina (coronary vasospasm). Male/female pattern. Early repolarization. Perimyocarditis. Left bundle branch block. Nonspecific intraventricular conduction disturbance. Left ventricular hypertrophy. Brugada syndrome. Takotsubo cardiomyopathy. Hyperkalemia. Post cardioversion. Pulmonary embolism. Pre-excitation. Aortic dissection engaging the coronary arteries. Left ventricular aneurysm. ♥ **Causes of ST-segment depression**: Ischemia. Non-ST segment elevation myocadial infarction (NSTEMI/NSTE-AKS). Physiological ST-segment depression. Hyperventilation. Hypokalemia. High sympathethic tone. Digoxin. Left bundle branch block. Right bundle branch block. Pre-excitation. Left ventricular hypertrophy. Right ventricular hypertrophy. Heart failure. Tachycardia. ♥ **Causes of waves/deflections in the J point (J wave syndromes)**: Brugada syndrome. Early repolarization.

5. T-wave

ASSESSMENTS	EVALUATION
♥ Should be concordant with the QRS complex. Should be positive in most leads. ♥ T-wave progression should be normal in chest leads. ♥ In limb leads the amplitude is highest in lead II, and in the chest leads the amplitude is highest in V2–V3.	♥ **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). ♥ **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. ♥ **T-wave inversion with simultaneous ST-segment deviation**: acute (ongoing) myocardial ischaemia. ♥ **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.

6. QTc interval and U-wave

ASSESSMENTS	EVALUATION
♥ QTc duration men ≤0,45 s. ♥ QTc duration women ≤0,46 s. ♥ Prolonged QTc duration may cause malignant arrhythmias (torsade de pointes, which is a type of ventricular tachycardia). ♥ Shortened QTc duration (≤0.32 s) is rare, but may also cause malignant ventricular arrhythmias. ♥ 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.	♥ **Acquired QT prolongation:** anti arrhythmic drugs (procainamide, disopyramide, amiodarone, sotalol), psychiatric medications (tricyclic antidepressants, SSRI, lithium etc); antibiotics (macrolides, kinolones, atovaquone, klorokine, amantadin, foscarnet, atazanavir); hypokalemia, hypocalcemia, hypomagnesemia; cerebrovascular insult (bleeding); myocardial ischemia; cardiomyopathy; bradycardia; hypothyroidism; hypothermia. A complete list of drugs causing QT prolongation can be found here. ♥ **Congenital QT prolongation:** genetic disease of which there are approximately 15 variants. ♥ **Short QTc syndrome (≤ 0,32 s):** caused by hyperkalcemia and digoxin treatment. May cause malignant ventricular arrhythmia. ♥ **Negative U-wave:** high specificity for heart disease (including ischemia).

7. Compare with earlier ECG tracings

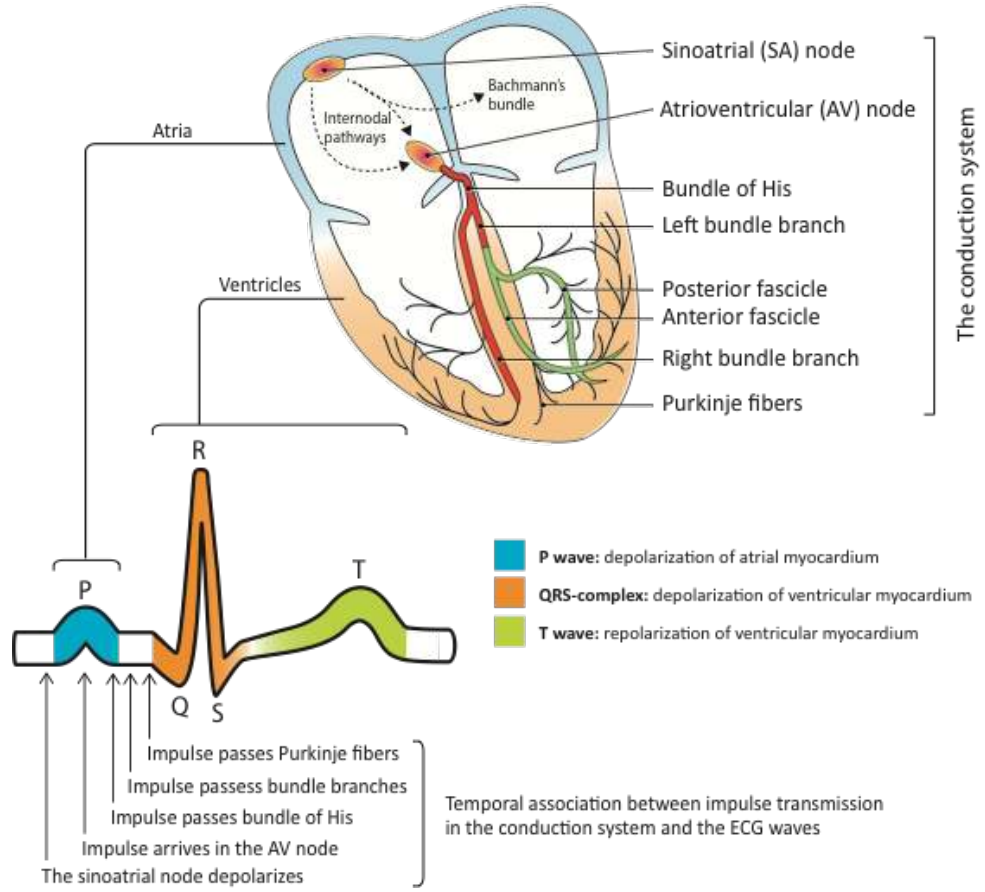
It is fundamental to compare the current ECG with previous recordings. All changes are of interest and may indicate pathology.

8. Clinical context

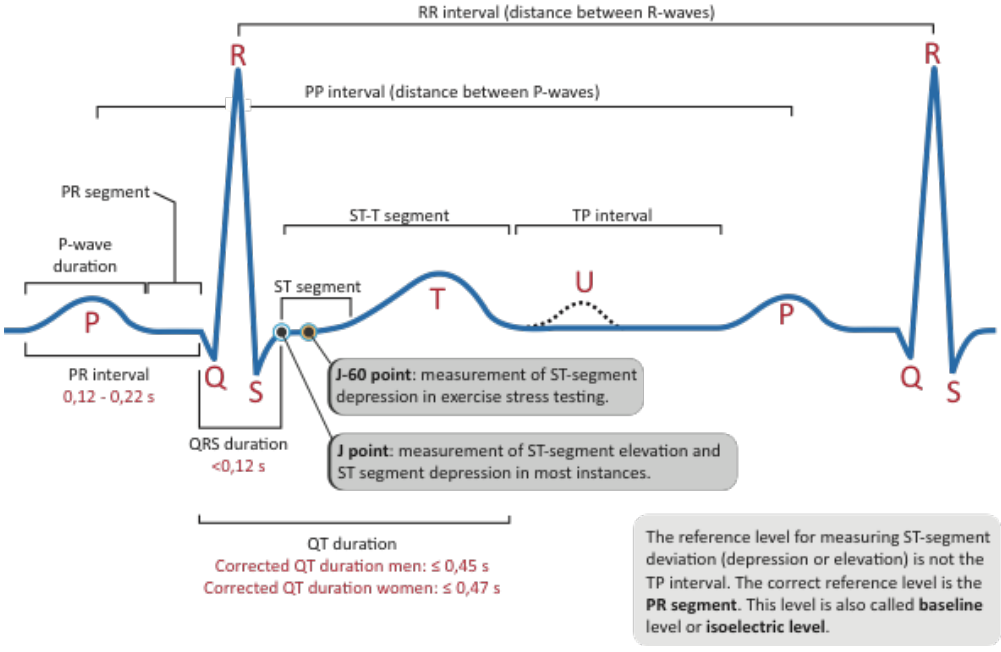
ECG changes should be put into a clinical context. For example, ST-segment elevations are common in the population and should not raise suspicion of myocardial ischemia if the patient do not have symptoms suggestive of ischemia.

The guide continues on the next page.

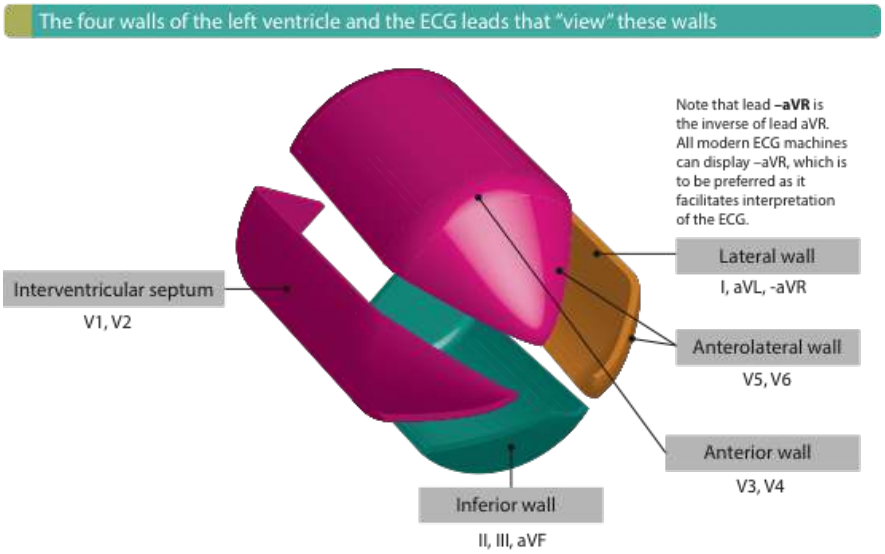
The cardiac conduction system



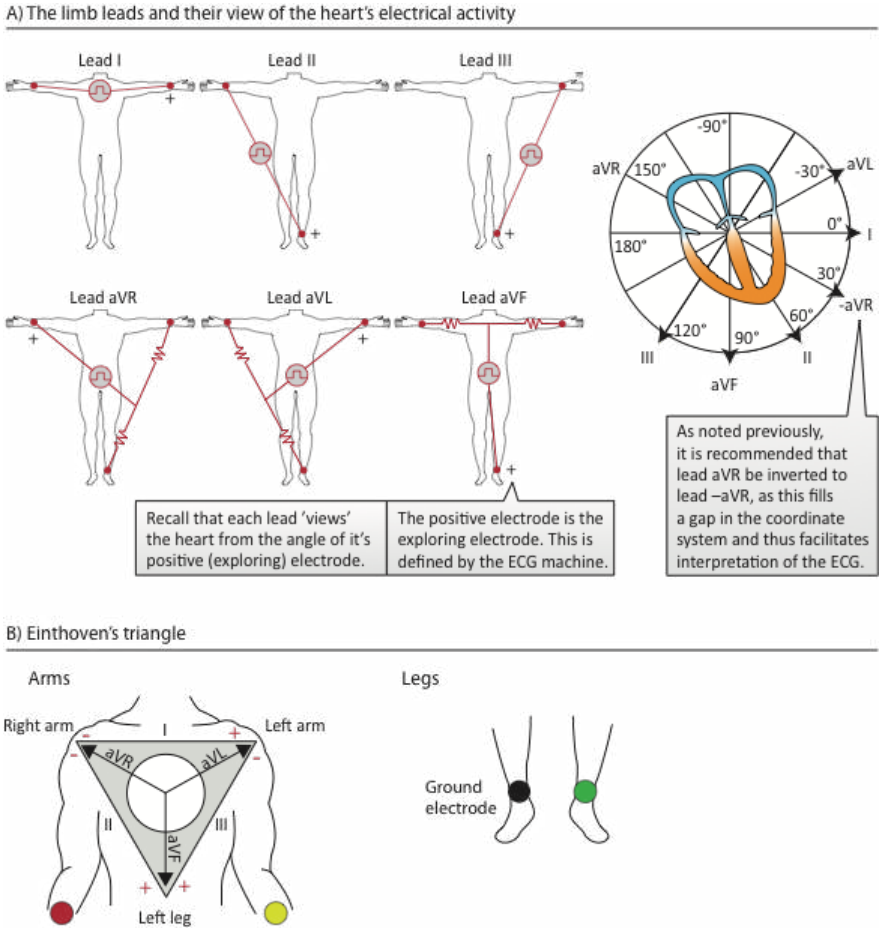
Waves, intervals and durations on the ECG



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

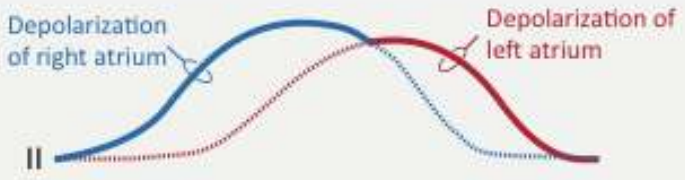


The ECG leads



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

P-mitrale

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.

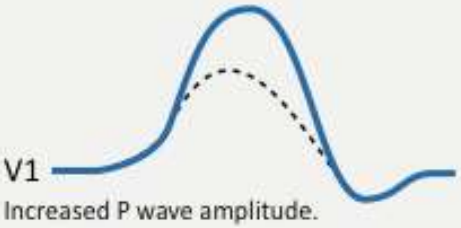
Enhanced second hump in lead II.

Enhanced negative deflection in V1.

P-pulmonale

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.

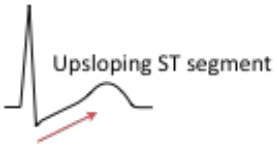
Increased P wave amplitude.



Increased P wave amplitude.

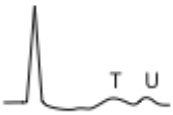
ST segment depressions

A Physiological ST-segment depressions

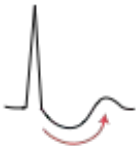


Upsloping ST-segment depression is a normal finding during physical exercise. It should be considered a normal finding, provided that T-waves are not inverted. Hyperventilation may cause similar ST-segment depressions.

B Non specific ST-segment depression



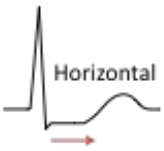
Hypokalemia and high sympathetic tone causes ST-segment depressions with flat T-waves and more marked U-waves. High sympathetic tone also causes tachycardia.



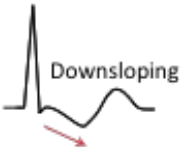
Digoxin (a drug used to treat atrial fibrillation and some caess of heart failure) causes a curved ST-segment depressions.

C ST-segment depressions caused by acute ischemia

Characteristics

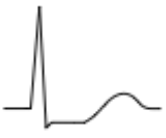


Very typical of ischemia.

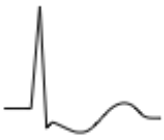


Typical of ischemia.

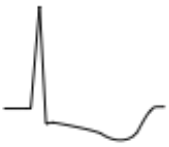
Real life examples



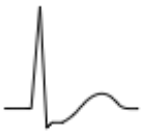
Horizontal depression with distinct ST-segment.



Downsloping with positive T-wave



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

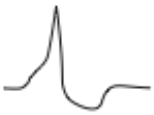
de Winter's sign is an exception to the rule that upsloping ST-segment depressions are not ischemic. de Winter's sign implies the presence of upsloping ST-segment depressions with prominent T-waves in the majority of the precordial (chest) leads. This is a sign of acute ischemia, most often caused by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

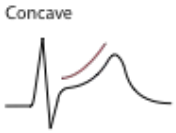
Right ventricular hypertrophy Large R-waves and ST-segment depressions in V1–V3. In case of chest discomfort, one must consider possibility of postero-lateral transmural ischemia as a differential diagnosis.

ST segment elevations

A Characteristics of ST-segment elevations caused by ischemia

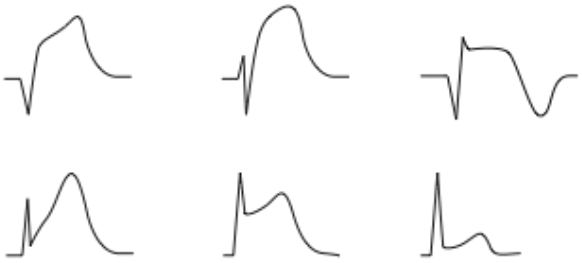
ST-segment elevations caused by ischemia typically displays a convex or straight ST-segment. Such ST-segment elevations in presence of chest discomfort are strongly suggestive of transmural myocardial ischemia. Note that the straight downsloping variant is unusual.

B Typical non-ischemic ST-segment elevation



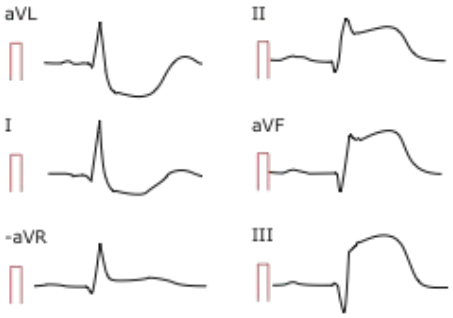
Non-ischemic ST-segment elevations are extremely common in all populations. They are characterized by a concave ST-segment and a greater distance between the J point and the T wave apex.

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.

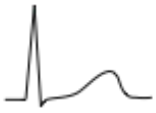
D Real life example (limb leads shown)



ECG from a male patient (age 61) 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

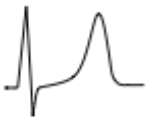
A Normal T-waves



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.

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

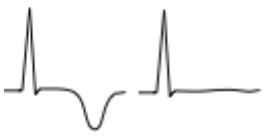
C Biphasic (diphasic) T-waves

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.

Whenever 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

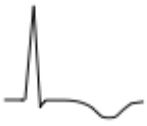


Post-ischemic
Symmetric T wave, with varying depth. Ranges from flat T wave to very deep T wave inversion. Inverted T waves do not equate acute (ongoing) ischemia, but rather appear after an episode of ischemia!

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!

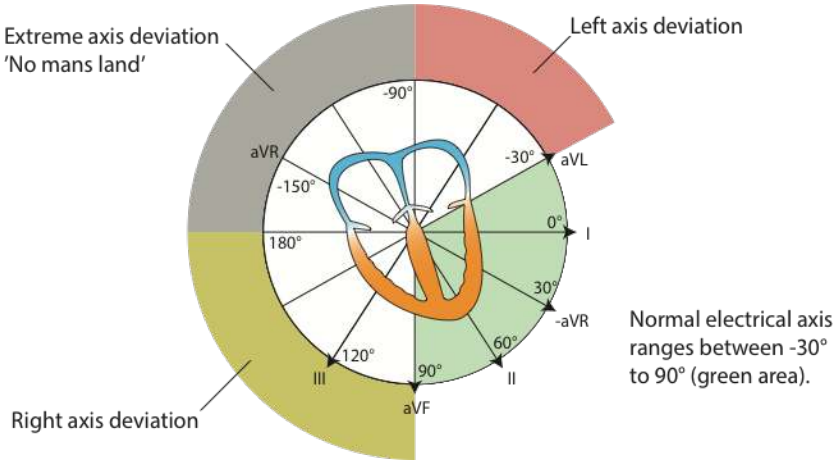
Cerebrovascular insult pattern
Very deep (gigantic) T wave inversions in the chest leads. Some studies report this finding in up to 30% of patients with intracerebral hemorrhage.

Hypertrophic cardiomyopathy
Symmetric T wave inversions, most comonly 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.

Electrical axis of the heart



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

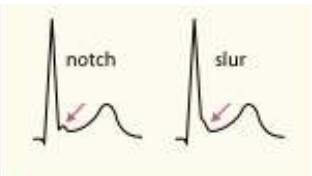
Evidence of previous myocardial infarction and high risk of ventricular tachcyardia and ventricular fibrillation.

Delta wave, pre-excitation

Risk of AVRT.
May be accompanied by atrial fibrillation (pre-excited atrial fibrillation).

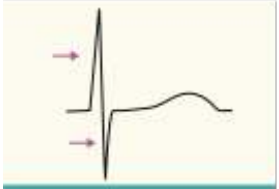
Brugada syndrome: ST elevation V1–V4. Shark tail appearance or saddle formed

Risk of ventricular tachycardia and ventricular fibrillation



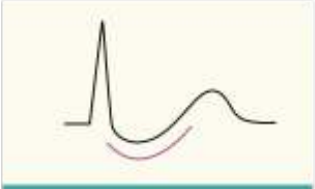
Early repolarization: slurring or notching at end-QRS

5 times increased risk of ventricular tachycardia and ventricular fibrillation.



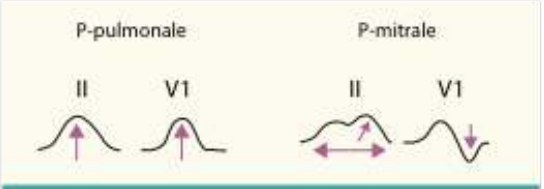
Hypertrophic cardio-myopathy: deep S, large R

Risk of ventricular tachycardia and ventricular fibrillation



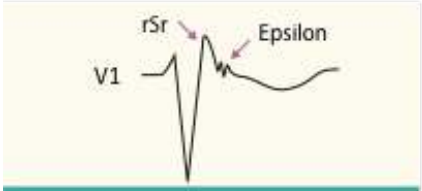
Digoxin effect: ST depression with "sagging" appearance

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.

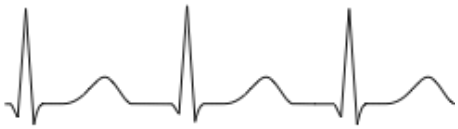
Assessment of RP interval for tachyarrhythmias

RP interval Short and <70 ms

Typical AVNRT. AVRT is unusual.

RP interval No visible P-wave

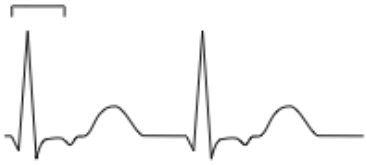
Typical AVNRT



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

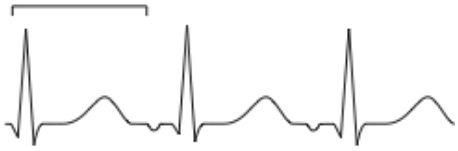
RP interval Short but >70 ms

In most cases AVRT. Occasionally atypical AVNRT or AT.

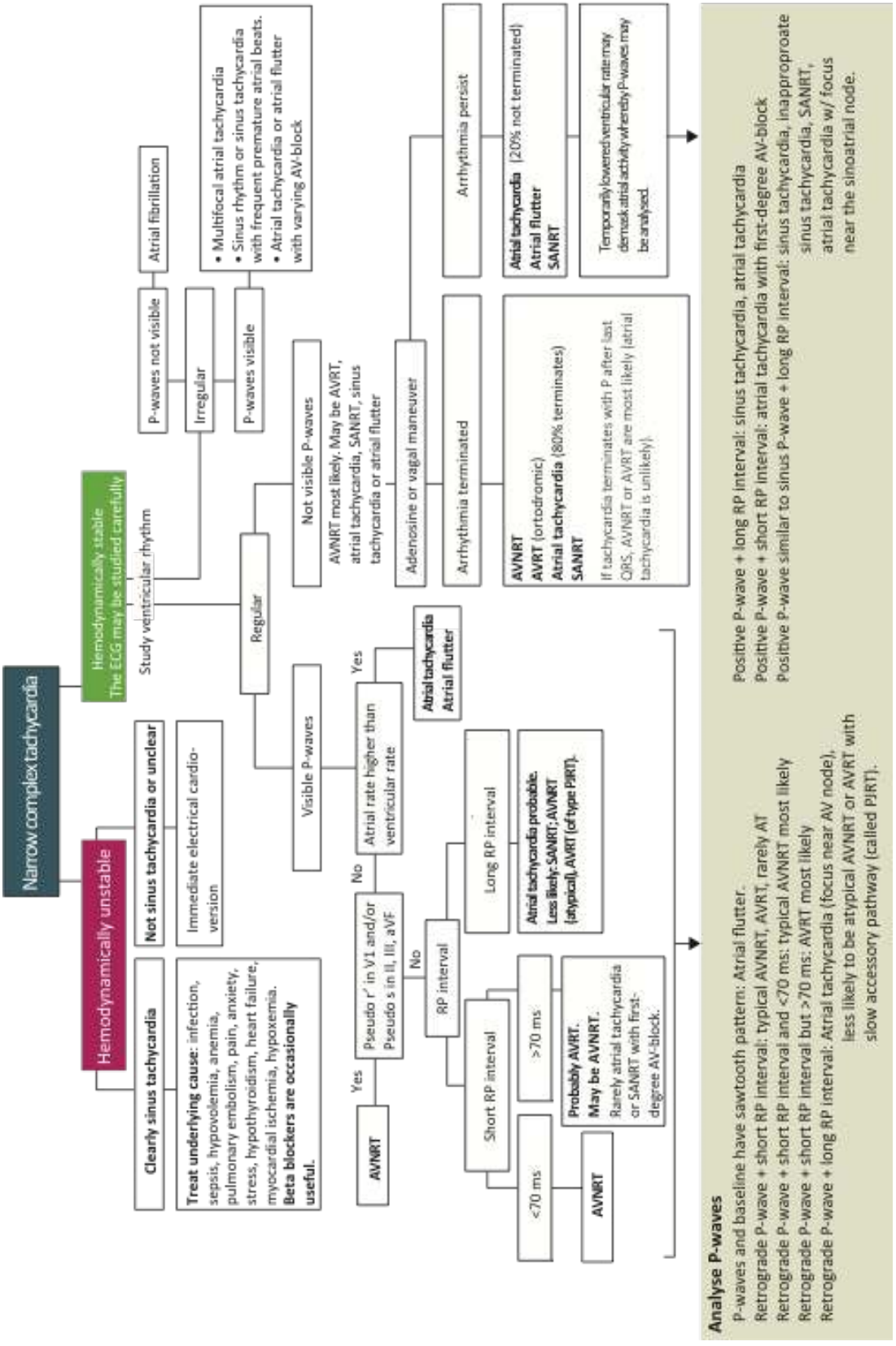


RP interval Long

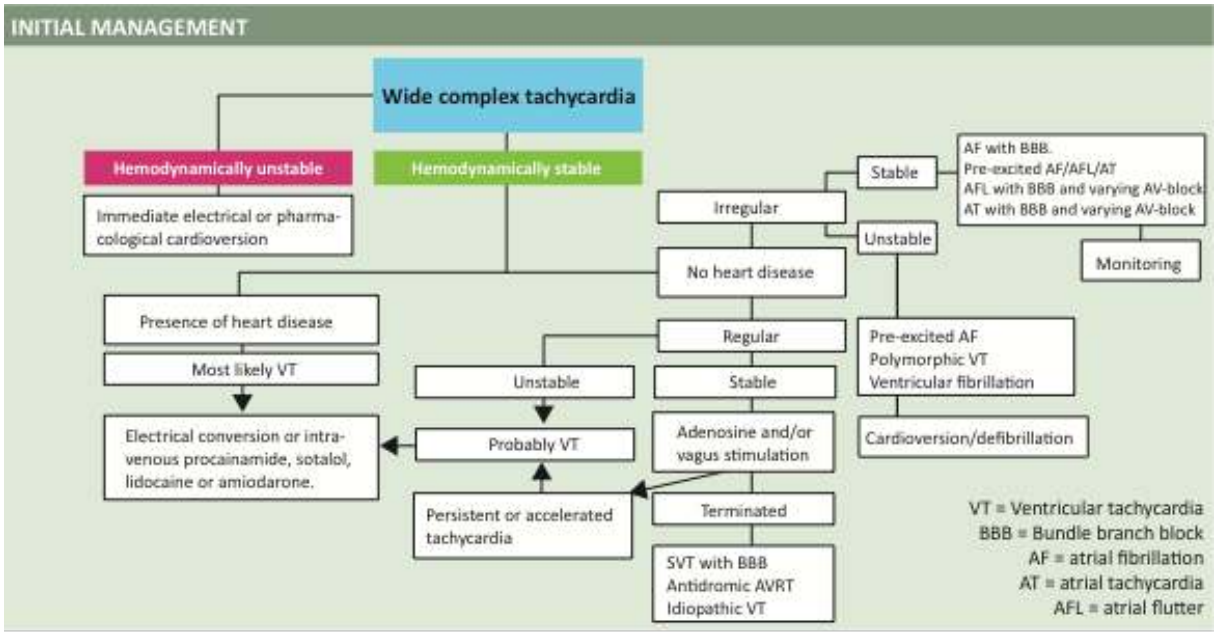
In most cases AT. Occassionally atypical AVNRT. Rarely PJRT.

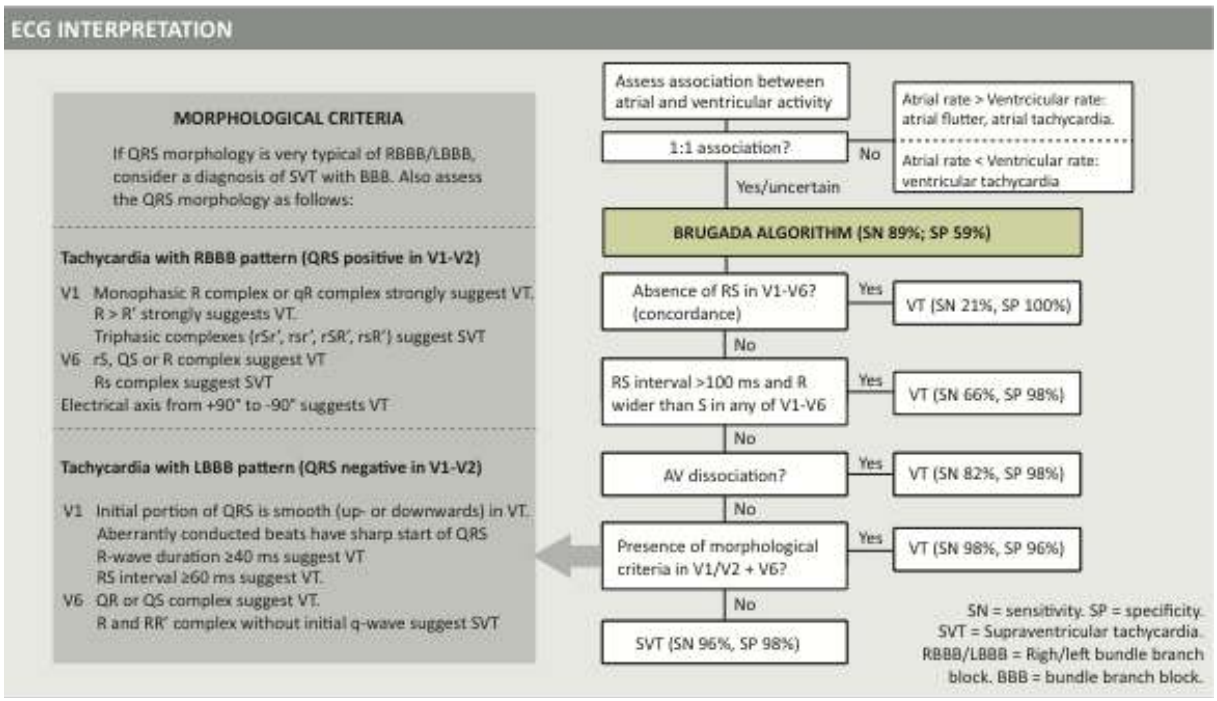


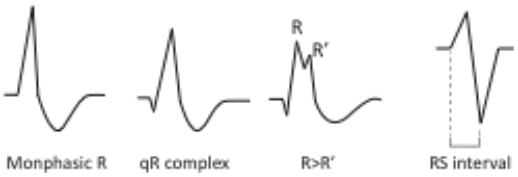
Diagnosis and management of tachyarrhythmias with narrow QRS complex



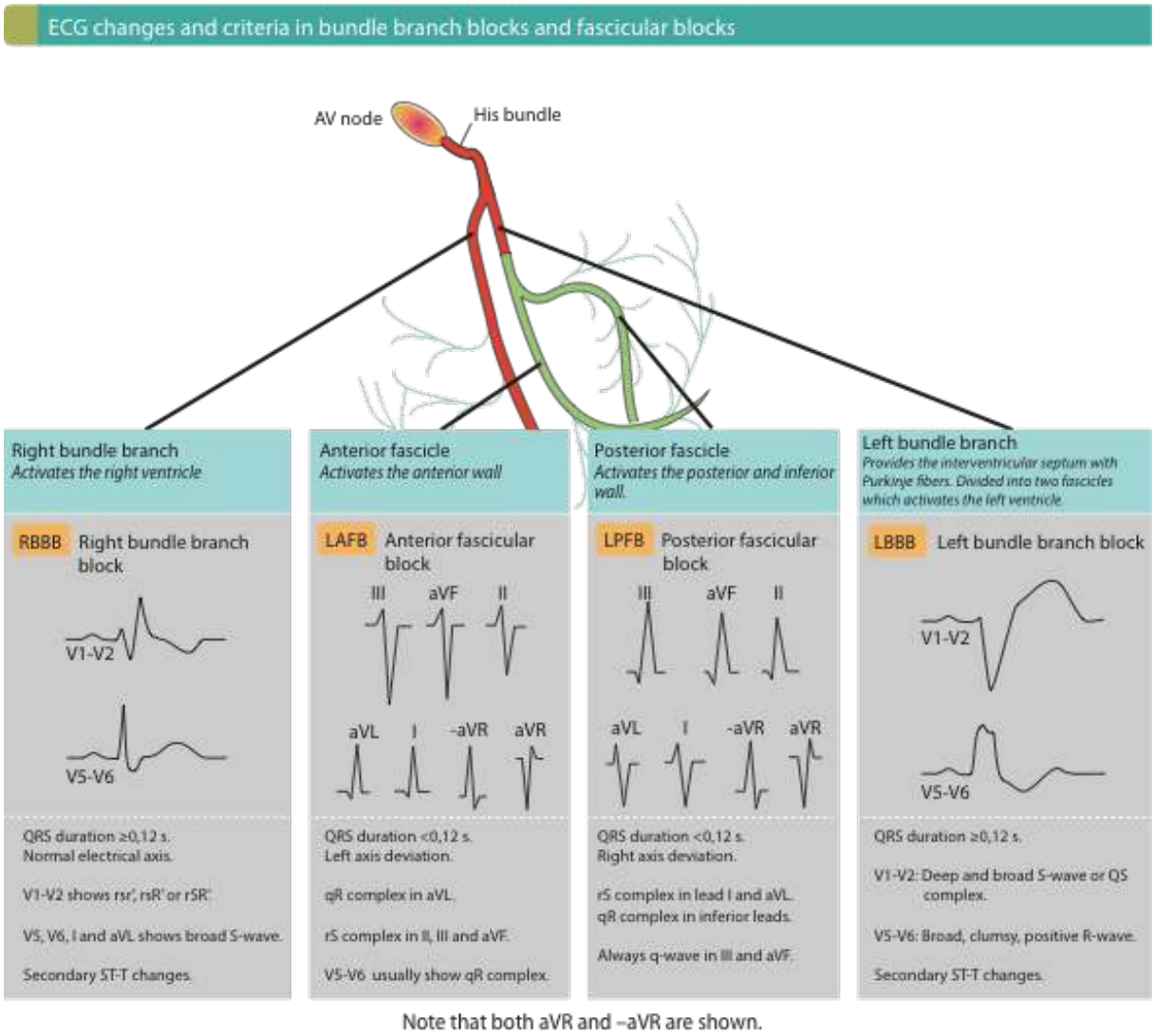
Diagnosis and management of tachyarrhythmias with wide QRS complex







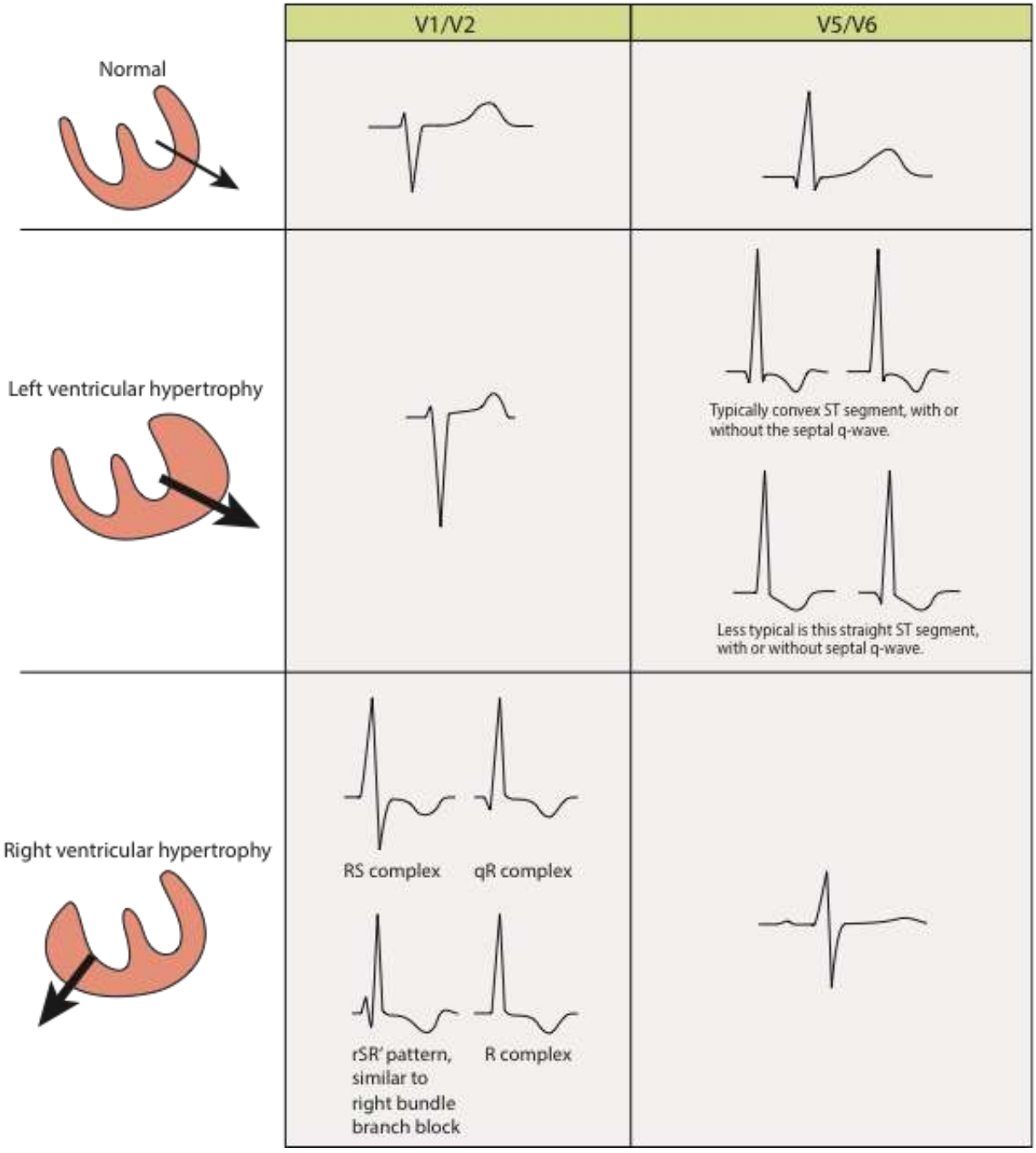
Intraventricular conduction defects



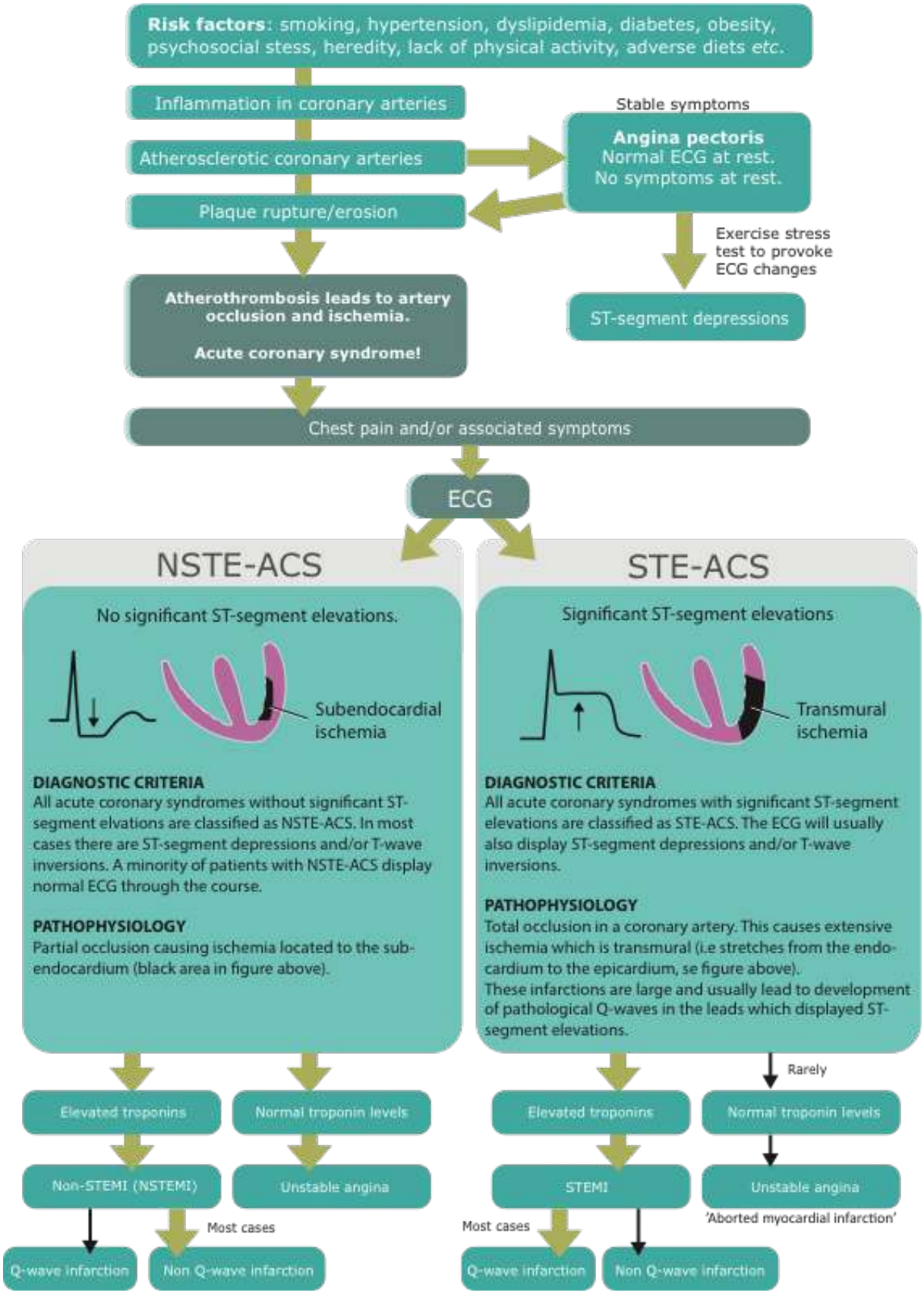
Note that both aVR and -aVR are shown.

Hypertrophy and dilatation

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



Classification of acute coronary syndromes (ACS)



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.