Systematic EKG Interpretation

Index

Introduction

This document presents a systematic interpretation of the EKG organized according to the mnemonic **OPQRST+**. O stands for Overview.

Systematic EKG Interpretation

O	Rate	Regularity
P	P-wave	PR-segment
Q	Q-wave	QRS-complex
R	QRS-axis	LVH, RVH
S	LVH, RVH	ST-segment
T	T-wave	QT-interval
+	Additional findings	Summary: rhythm + suspected conditions (e.g. ischemia)

Rate

When the paper speed of the rhythm strip is 25 mm/sec, one large box (5 mm per side) corresponds to 0.2 seconds. The heart rate in beats per minute (bpm) can be estimated by dividing 300 by the number of large boxes between two adjacent QRS-complexes. When the paper speed is 50 mm/sec, the heart rate is 600 divided by the number of large boxes between two adjacent QRS-complexes. When the paper speed is 12.5 mm/sec, the heart rate is 150 divided by the number of large boxes.

Differential Diagnosis of Tachycardia

	Regular	**Irregular**
QRS < 120 msec	• ST • MRAT (e.g. atrial flutter) • EAT • AVNRT • Orthodromic AVRT	• AF • MRAT with varying AV block • EAT with varying AV block • MAT
QRS ≥ 120 msec	• VT, monomorphic • Antidromic AVRT • ST, MRAT, EAT, AVNRT or orthodromic AVRT with o Bundle branch block o Na-channel blockade o Hyperkalemia o Accessory pathway	• VT, polymorphic (e.g. torsade de pointes) or monomorphic • AF, MRAT/EAT with varying AV-block, or MAT with o Bundle branch block o Na-channel blockade o Hyperkalemia o Accessory pathway

ST: sinus tachycardia
MRAT: macro-re-entrant atrial tachycardia
AVNRT: atrioventricular nodal re-entrant tachycardia
AVRT: atrioventricular re-entrant tachycardia
AF: atrial fibrillation
EAT: ectopic atrial tachycardia
MAT: multifocal atrial tachycardia
VT: ventricular tachycardia

Regularity

This step consists in noting whether the QRS-complexes show up at regular intervals or not, and if the rhythm is irregular, whether there is a pattern (e.g. three QRS-complexes appearing with a common time-interval between QRS-complexes; then a pause; then three QRS-complexes appearing again with the same common time-interval between QRS-complexes).

- Sinus rhythm is usually regular, but some cycle length variation may occur due to alternations in autonomic input and changes in cardiac filling during inspiration. If the P-P interval varies by > 120 msec (and the QRS-complexes appear to be triggered by the P-waves), sinus arrhythmia is considered present [1]
- Atrial flutter: the cycle length between P-waves is very regular, with an absolute cycle length difference of < 24 msec and a typical standard deviation of 2-5 msec [2]. The heart rate is hence regular if the conduction rate remains the same (e.g. 2:1 AV-conduction). If the AV-conduction frequence varies, the heart rate becomes irregularly irregular.
- Monomorphic VT is regular with < 50 msec beat-to-beat cycle length variation [3]
- Second degree AV-blocks can be regularly irregular, with e.g. 3:2 AV-conduction
- Atrial fibrillation is irregularly irregular

Regularity is one of the aspects of the EKG that allows for determination of the underlying rhythm. Other factors—e.g. presence of P-waves, interval between P-waves and QRS-complexes, QRS-morphology—are assessed during the following steps in the systematic EKG-interpretation. Determining the rhythm is hence part of the last step in the systematic interpretation.

P-Wave

The P-wave is a small deflection preceding the QRS-complex and corresponds to atrial depolarization. A normal P-wave axis is usually around 60°, and hence positive in lead II [4]. At rest, a normal P-wave is < 2.5 mm (0.25 mV) in height and < 0.12 seconds in duration [4].

Sinus rhythm is likely when:
- there is a P-wave preceding each QRS-complex
- there is a QRS-complex following each P-wave
- the P-wave is positive in lead II
- the PR-interval is between 0.12 and 0.2 seconds.

Negative P-waves in lead II suggests one of the following:
- lead placement error
- a retrograde P-wave with the atrial depolarization originating from the AV node
- ectopic atrial focus

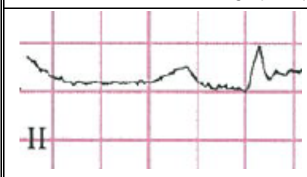
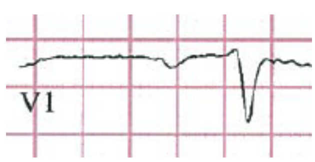
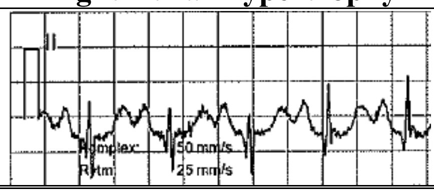
Atrial hypertrophy may be suggested by the appearance of the P-waves in leads II and V1 [4]:

Left Atrial Hypertrophy

Suggestive Findings		Differential Diagnosis
II	Humped or notched P-wave and a duration > 0.12 sec	• Hypertensive heart disease • Aortic stenosis, aortic insufficiency • Mitral stenosis, mitral insufficiency • Cardiomyopathy
V1	Biphasic P-wave with a terminal negative deflection of > 0.04 sec or > 1 mm (0.1 mV) in depth	

Right Atrial Hypertrophy

Suggestive Findings		Differential Diagnosis
II	P-wave > 2.5 mm, < 0.12 sec	• Pulmonary hypertension, acute or chronic • Pulmonary stenosis, atrial septal defects, Ebstein's anomaly, Tetralogy of Fallot
V1	P-wave > 2.5 mm, occasionally negative, < 0.12 sec	

Left Atrial Hypertrophy		Right Atrial Hypertrophy
		

Small P-waves may be caused by hyperkalemia. As hyperkalemia worsens, the P-waves may disappear altogether despite the continued presence of sinus activity [5].

Absence of P-waves may be due to:

- sinus arrest
- atrial fibrillation
- hyperkalemia (P-wave amplitude is so reduced that P-waves are invisible)
- P-waves hidden with the QRS-complexes

PR-Segment

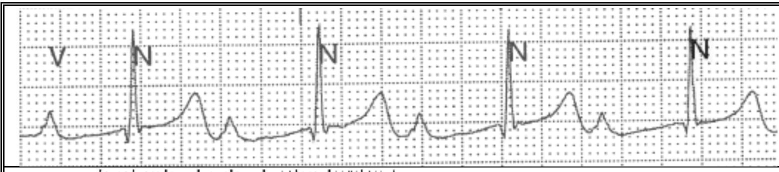
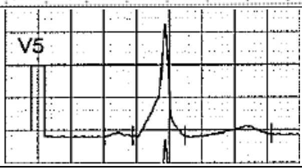
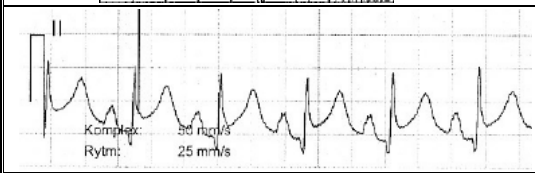
The PR-interval is the time from the beginning of the P-wave until the beginning of the QRS-complex [4] and corresponds to the delay between the onset of atrial depolarization and the onset of ventricular depolarization. A normal PR-duration is between 0.12 and 0.2 seconds [4] and the PR-interval is usually isoelectric with the EKG baseline.

Prolonged PR-interval, i.e. a PR-interval > 200 msec, may be caused by the conditions listed above in the table Differential Diagnosis of Conduction Blocks [4]. First degree AV-block refers to a rhythm with a uniformly prolonged PR-interval (i.e. no missing QRS-complexes following the P-waves).

Short PR-interval may occur with:

- A non-sinus origin of the P-wave
- Pre-excitation, in which the PR-interval is cut short by the premature depolarization of the ventricle through an accessory bypass tract, resulting in a slurred upstroke of the QRS-complex called a **delta wave**.

Depressed PR-segment relative to the TP-segment EKG baseline suggests pericarditis [6, 7] but is not specific for this condition [8]

	**First degree AV block**
	**Delta Wave, resulting from pre-excitation, and associated with a short PR-segment.**
	**PR-segment depression in the setting of acute pericarditis. The baseline is the TP-segment.**

First Degree AV-Block is present when the PR-interval exceeds 0.2 seconds and when each P-wave is followed by a QRS-complex.

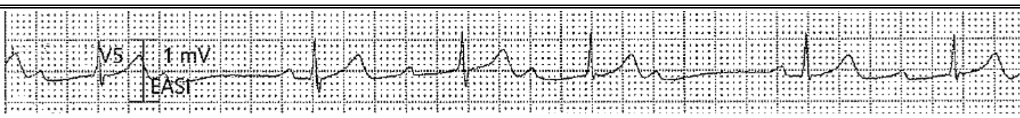
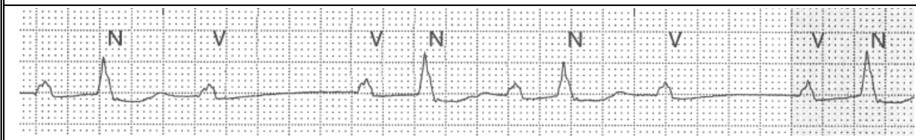
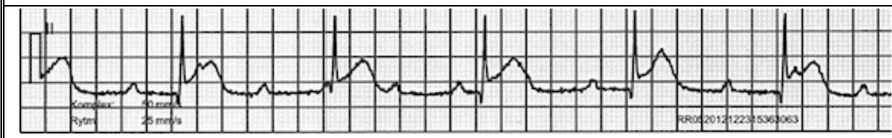
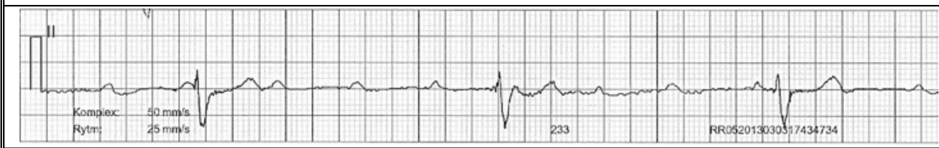
Second Degree AV-Block is present when occasional P-waves are not followed by a QRS-complex.

- Second degree AV-block Mobitz type 1 (also known as Wenckebach) is present when the PR-interval gradually lengthens until no QRS-complex follows the P-wave. The conduction abnormality is more likely to be in the AV-node.
- Second degree AV-block Mobitz type 2 is present when the PR-interval is constant and P-waves are intermittently not conducted. The conduction abnormality is more likely to be below the AV-node, and second degree AV-block type 2 is more likely to progress to third degree AV-block than second degree AV-block type 1.

When every second P-wave is not conducted, the surface EKG cannot distinguish between Mobitz type 1 and type 2. The following can help:

- A PR-interval > 300 msec when the P-wave is conducted suggests Mobitz type 1
- A narrow QRS-complex suggests Mobitz type 1
- If atropine (0.25 - 0.5 mg) converts the block from 2:1 to 3:2, that confirms Mobitz type 1
- If carotid sinus massage converts the block from 2:1 to 3:1 or worse, that suggests Mobitz type 1; if it converts the block from 2:1 to 3:2, that suggests Mobitz type 2

Third Degree AV-Block is a bradyarrhythmia characterized by the absence of temporal relationship between the P-waves and the QRS-complexes, so-called AV-dissociation.
- The QRS is narrow if generated by a pacemaker located proximal to the bifurcation of the bundle of His, in which case the heart rate is typically 40-60 beats/min and the rhythm is referred to as an AV-nodal escape rhythm.
- The QRS is wide if generated in the ventricle, in which case the heart rate is typically 20-40 beats/min and referred to as a ventricular escape rhythm.

	**Second degree AV block Mobitz type 1**
	**Second degree AV block Mobitz type 2**
	**Third degree AV block**
	**Third degree AV block**

Differential Diagnosis of Conduction Blocks [4, 9]

Pathophysiology	Examples
Degenerative*	Fibrosis, sclerosis, calcification
Ischemia**	Ischemic heart disease, including acute myocardial infarction
Infectious	Lyme disease, toxoplasmosis, infective endocarditis, myocarditis
Infiltrative	Amyloidosis, sarcoidosis, hemochromatosis, lymphomas
Drugs	Beta blocker, verapamil, cardizem, digoxin, antiarrhythmics
Neurological	Increased vagal tone (sleep, pain, carotid sinus massage)
Trauma	Valve surgery, ablations
Electrolytes	Hyperkalemia
Endocrine	Severe hypo- and hyperthyroidism

* Degenerative/idiopathic is the leading cause of conductions blocks (50-67% of cases)
** Second most common cause

Q-Wave

The Q-wave refer to the initial deflection of the QRS-complex when the deflection is negative (i.e. below the EKG baseline) [4].

Differential Diagnosis of Q-Waves [10]:

Pathophysiology	Examples
Physiologic and positional effects	• Misplacement of chest lead electrodes • Dextrocardia, • Left pneumothorax • Chronic obstructive pulmonary disease (COPD)
Myocardial injury or replacement*	• Myocardial ischemia • Dilated cardiomyopathy • Myocarditis • Replacement of myocardial tissue by electrically inert material (amyloidosis, metastatic tumor, scleroderma, sarcoidosis) • Duchenne muscular dystrophy
Ventricular enlargement	• Chronic obstructive pulmonary disease • Acute pulmonary embolism • Hypertrophic cardiomyopathy • Heart failure with reduced or preserved left ventricular ejection fraction
Altered ventricular conduction	• Left bundle-branch block (QS in right and mid precordial leads) • Wolff-Parkinson-White pre-excitation pattern

* Any process, acute or chronic, that causes sufficient loss of regional electromotive potentials or (bi)ventricular dilation

One source emphasizes the following [10]:

- not all Q-waves are pathological
- not all pathological Q-waves are caused by myocardial infarction
- consensus regarding the precise criteria for the diagnosis of pathologic Q waves (with respect to their width, extent, and location) is lacking

The author recommends taking the clinical context into account when interpreting the presence of Q-waves and echocardiography as the first-line noninvasive test to assess unexplained Q waves [10].

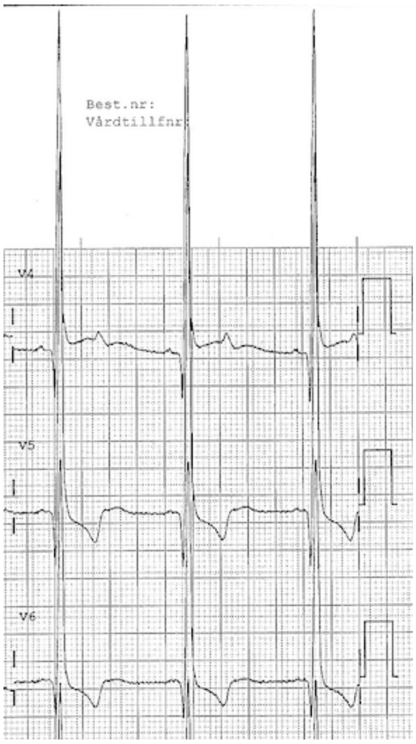
According to the fourth universal definition of myocardial infarction [11], Q-wave criteria are associated with prior myocardial infarction when present in the absence of left ventricular hypertrophy and left bundle branch block.

EKG changes associated with prior myocardial infarction (in the absence of left ventricular hypertrophy and left bundle branch block) [11]

Leads	Pathological Findings
V2 or V3	• Q-wave > 20 msec • QS-complex
Consecutive lead groupings: • I, aVL • II, III, aVF	Either of the following in two consecutive leads: • Q wave ≥ 30 msec and ≥ 1 mm deep • QS-complex

• V1–V9	
V1 or V2	• R wave > 40 msec and R/S > 1 with a concordant positive T wave in absence of conduction defect.

Narrow (< 0.03 sec), deep Q-waves in I, aVL, V5 and/or V6 in a patient with high left ventricular voltage who presents with syncope or presyncope suggests hypertrophic obstructive cardiomyopathy (Amal Mattu http://ekgumem.tumblr.com/post/28409061231/ecg-findings-in-hypertrophic).



QRS-complex

The QRS-complex stretches from the beginning of the Q-wave (or the beginning of the R-wave in the absence of Q-wave) to the J-point, i.e. the junction between the QRS-complex and the ST-segment. It represents the time required for depolarization to spread throughout the ventricles [4].

- **R-wave** refers to the first positive deflection of the QRS-complex
- **S-wave** refers to the negative deflection of the QRS-complex following an R-wave
- **R'-wave** refers to positive QRS-complex deflection following an S-wave
- **QS-wave** refers to an entirely negative QRS-complex [4]

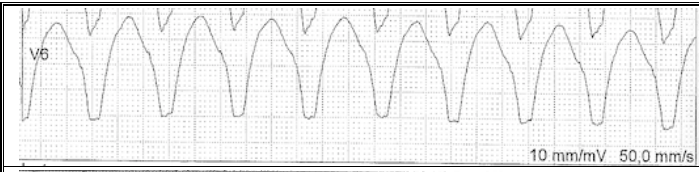
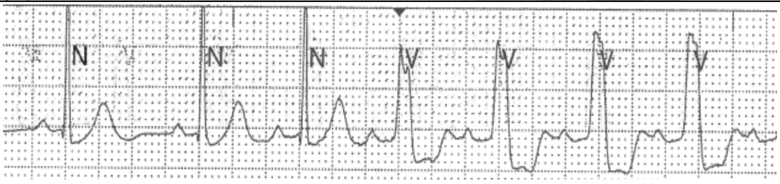
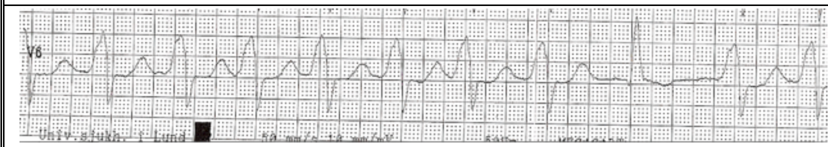
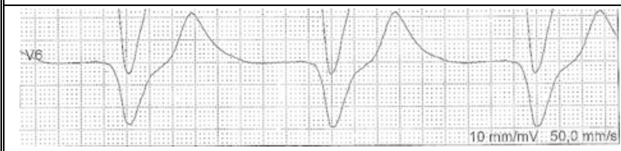
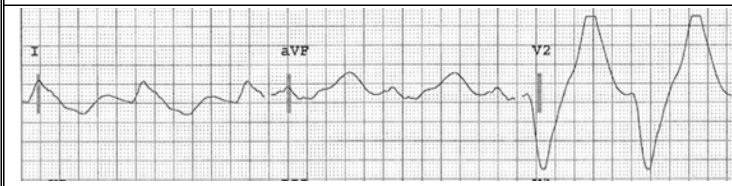
A normal QRS-duration in adults is ≤ 100 msec [4]; such 'narrow' QRS-complexes indicate that the ventricular activation has originated from a site proximal to the bifurcation of the bundle of His [12], that the His-Purkinje system distal to the bifurcation is functioning properly and that the myocytes are depolarizing normally.

Wide QRS-Complexes indicate one of the following:

- that the depolarization of the ventricles has not occurred via a healthy HIS-Purkinje system [4], e.g. in the presence of a left bundle branch block (LBBB) or right bundle branch block (RBBB)
- abnormally functioning fast sodium channels, e.g. due to hyperkalemia or poisoning

Differential Diagnosis of Wide QRS-Complexes

Pathophysiology	Examples
Depolarization originates in the ventricle	• **Premature ventricular beat** • **Ventricular escape rhythm** in AV-block III • **Paced ventricular beats**. In the setting of right ventricular pacing, the EKG shows a LBBB pattern. If a patient with a pacemaker shows a RBBB pattern, it indicates that the pacemaker wire is stimulating the left ventricle [13, 14] • **Ventricular tachycardia**
Bundle branch block	• **Left bundle branch block** • **Right bundle branch block** +/- left fascicular blocks • **Tachycardia-related bundle branch block** [12] • **Ashman's phenomenon,** whereby a long RR-interval is followed by a short RR interval and a wide QRS-complex at the end of the short RR-interval due to incomplete repolarization of a bundle branch (typically right bundle branch)
Accessory pathway	• Part of the ventricle is prematurely depolarized through an accessory pathway, such as a with the Wolff-Parkinson-White syndrome • **Antidromic atrioventricular reentry tachycardia** (AVRT)
Hyperkalemia	• **Hyperkalemia** interferes with the functioning of sodium channels [15], thereby impending phase 0 and resulting in a wide QRS-complex
Membrane stabilization	• Intoxication with **class I antiarrhythmics** and other **sodium channel blocking agents** inhibits the fast cardiac sodium channels, a phenomenon known as ’membrane stabilization’ [16].

	**Monomorphic Ventricular Tachycardia**
	**Supraventricular tachycardia leading to a rate-related bundle branch block, and erroneously interpreted as a ventricular tachycardia**
	**Irregular wide QRS-complex tachycardia with an occasional narrow QRS-complex: atrial fibrillation with pre-excitation**
	**Broad QRS-complexes resulting from hyperkalemia. The heart rate is around 70 bpm. P-waves are not visible.**
	**Mild tachycardia (around 110 bpm; 50 mm/s) with very wide QRS-complexes resulting from poisoning with a sodium channel blocking agent (Amitryptiline in this case)**

Complete Bundle Branch Block is suggested by QRS-complex duration > 120 msec in adults, > 100 msec in children 4 to 16 years of age, and > 90 msec in children less than 4 years of age [17] accompanied by other morphological criteria.

Left Bundle Branch Block (LBBB) [4, 17]

Suggestive Findings	Differential Diagnosis
• Wide, entirely negative QS complex in V1 (rarely, a wide rS complex) • Wide, tall R-wave without a Q-wave in V6	• Long-standing hypertensive disease • Valvular lesion (e.g. aortic stenosis, aortic regurgitation) • Cardiomyopathies • Coronary artery disease • Degenerative changes

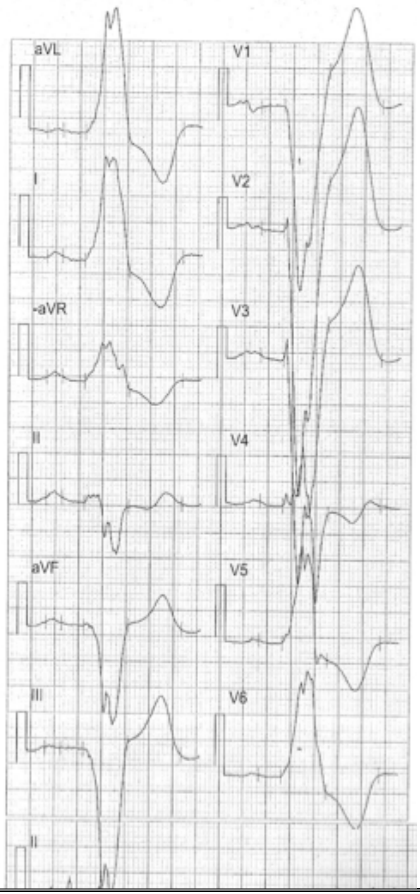
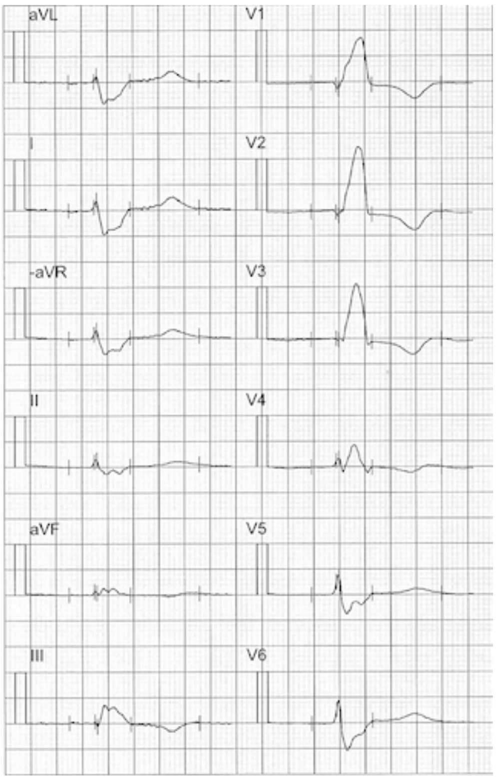
Right Bundle Branch Block (RBBB) [4, 17]

Suggestive Findings	Differential Diagnosis

• rSR’ appearance in V1 or V2. The R' deflection is usually wider than the initial r-wave. • qRS pattern in V6 with a wide S-wave of greater duration than the R-wave or > 40 msec in leads I and V6 in adults. • When a pure dominant R-wave with or without a notch is present in V1, the R peak time in leads V5 and V6 is normal while the R peak time in lead V1 is > 50 msec.	• Atrial septal defect with left-to-right shunt • Chronic pulmonary disease with pulmonary artery hypertension • Pulmonary stenosis • Cardiomyopathies • Coronary artery disease • Chronic degenerative changes

A **right bundle branch block** combined with a **left anterior fascicular block** results in
- A wide QRS-complex with a wide S-wave in V6
- A leftward axis

A **right bundle branch block** combined with a **left posterior fascicular block** results in
- A wide QRS-complex with a wide S-wave in V6
- A rightward axis

Left Bundle Branch Block	Right Bundle Branch Block with Left Posterior Fascicular Block
	

If a patient with **pacemaker** who initially has a LBBB pattern on the EKG (due to right ventricular pacing) now presents with a RBBB pattern, it indicates that the pacemaker wire has become dislodged and is now stimulating the left ventricle [13, 14].

The **Ashman phenomenon** refers to a bundle branch block pattern classically seen in patients with atrial fibrillation. A long RR cycle is followed by a short RR cycle ('long-short' rule), and the second beat of that combination shows a bundle branch pattern (usually RBBB) [12]. The Ashman phenomenon can lead to a succession of wide complex beats misinterpreted as non-sustained VT.

QRS-Axis

The mean QRS electrical axis refers to mean direction of the QRS-complex as seen on the coronal plane of the body. The EKG leads that map the heart's electrical activity onto the body's coronal plane are the limb leads. A normal QRS-axis lies between -30° and +100° [4]. When the QRS-complex is positive in both lead I and lead II, the QRS axis lies between -30° and +90° and is thus normal.

QRS-Axis Deviation can be detected by noting the polarity of the QRS-complexes in leads I and II [4]:

QRS-Axis Deviation

Lead I	Lead II	QRS-Axis	Differential Diagnosis
QRS -	QRS +	Rightward shift +90° to + 150°	• Right ventricular hypertrophy • Left posterior hemiblock • Lateral wall myocardial infarction • Chronic lung disease (e.g. emphysema) • Acute right ventricular overload (e.g. PE)
QRS +	QRS -	Leftward shift -30° to - 90°	• Left ventricular hypertrophy • Left anterior hemiblock • Left bundle branch block • Inferior wall myocardial infarction • Endocardial cushion defects (congenital)
QRS -	QRS -	"Northwest" axis +150° to -90°	• Incorrect lead placement • Situs inversus • Heart transplant
Isoelectric QRS	Isoelectric QRS	Indeterminate axis	• Normal variant • Intoxication with sodium channel blockers • Hyperkalemia

R-Wave

Ventricular hypertrophy may be suggested by the height of the R-wave in specific leads, by the sum of the R-wave and the S-wave in specific leads, or by additional findings. The criteria listed in the following tables have varying sensitivities and specificities [18].

Left Ventricular Hypertrophy (LVH) [4]

Suggestive Findings	Differential Diagnosis
• R_{aVL} > 11-13 mm • S_{V1} + R_{V5} or R_{V6} > 35 mm (i.e. > 3.5 mV) • S_{V3} + R_{aVL} > 28 mm in men; > 20 mm in women • Slight ST-segment depression followed by an asymmetrically inverted T-wave in V5-V6 • EKG findings of left atrial hypertrophy • Left axis deviation	• Systemic hypertension • Aortic stenosis, aortic regurgitation • Mitral regurgitation • Dilated, hypertrophic cardiomyopathy

The Cornell product is (S_{V3} + R_{aVL}) x QRS. A Cornell product > 1800 mm*msec suggests HFpEF (heart failure with preserved ejection fraction) and increased mortality [19].

Right Ventricular Hypertrophy (RVH) [4]

Suggestive Findings	Differential Diagnosis
• R-wave exceeding the S-wave in lead V1 • Right axis deviation • T-wave inversions in V1-V3 • EKG findings of right atrial hypertrophy	• Pulmonary hypertension • Pulmonary stenosis

Tall R-waves in lead V1, defined as an R/S ratio ≥ 1, are unusual. The QRS-complex in lead V1 usually shows as small R-wave (due to left-to-right septal depolarization) followed by a large S-wave (due to the dominance of left ventricular depolarization).

The differential diagnosis of tall R-waves in lead V1 is [20]:

- Normal variant occurring in 1% of the population
- Right bundle branch block
- Left ventricular ectopy
- Right ventricular hypertrophy
- Acute right ventricular dilatation (strain)
- Hypertrophic cardiomyopathy
- Progressive muscular dystrophy
- Dextrocardia
- Misplaced leads
- Posterior myocardial infarction: tall R-waves in V1-V2, associated with ST depression and upright T-waves, provide a "mirror image" of the Q-waves, ST elevation and inverted T-waves seen on the posterior leads V7-V9 in the setting of a posterior STEMI [21]

S-Waves

- The sum of the S-waves and R-waves in specific leads may suggest left ventricular hypertrophy (see previous section).
- A wide S-wave in V6 of greater duration than the R-wave or an S-wave > 40 msec in leads I and V6 in adults, combined with a wide QRS-complex, suggests a right bundle branch block.

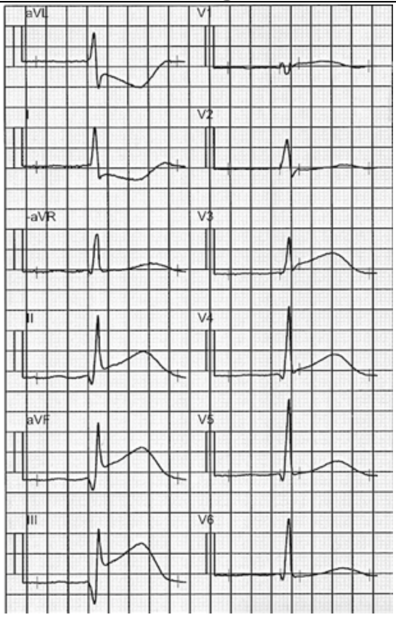
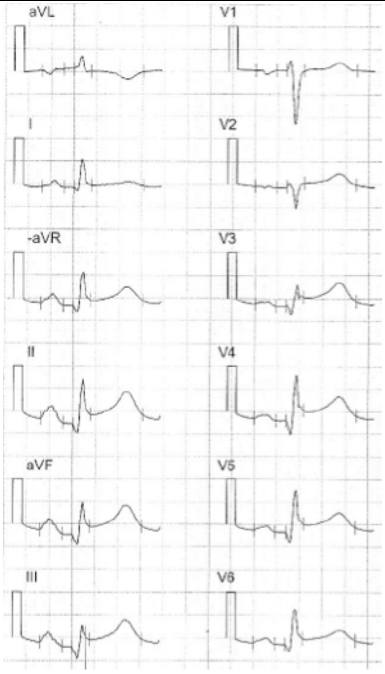
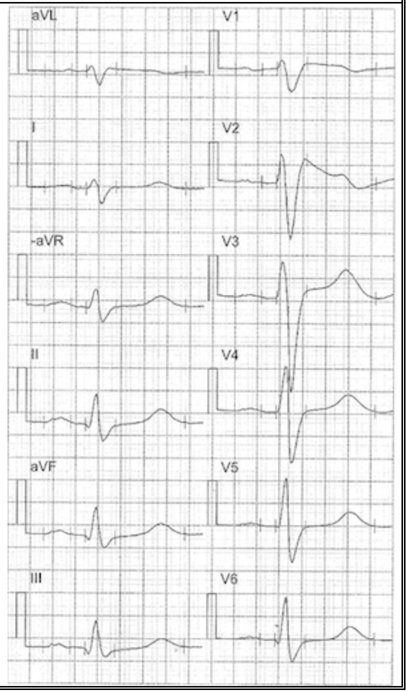
ST-Segment

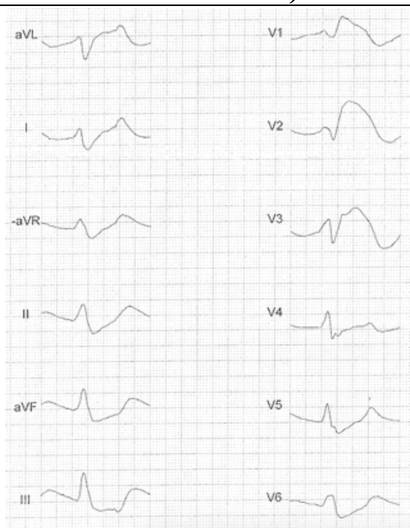
The ST-segment stretches from the end of the QRS-complex to the beginning of the T-wave and represents the beginning of ventricular repolarization [4]. The junction between the QRS-complex and the ST-segment is referred to as the **J-point** [4]. The differential diagnosis of ST-segment elevation and depression is presented in the following tables.

Differential Diagnosis of ST-Segment Elevation [22, 23]

Pathophysiology	Characteristics
STEMI	• **Horizontal or convex (dome-shaped)** ST-segment elevation suggests STEMI. The ST-segment elevation in STEMI may also be concave. • **ST-segment elevation in lead III > ST-segment elevation in lead II** strongly suggests STEMI instead of pericarditis (Mattu https://www.youtube.com/watch?v=cE71p9mfOq8) • **Check-mark sign** refers to a QR-T complex, i.e. a complex where the QR complex seems to merge directly with the T-wave without an intervening S-wave; this complex suggests STEMI [8] • **Reciprocal ST-segment depressions** may be present. One study reported that the presence of any ST depression in lead aVL in associated with inferior ST-segment elevation is highly sensitive for coronary occlusion in inferior myocardial infarction and very specific for differentiating inferior myocardial infarction from pericarditis [24]. • **The location of the ST-segment elevation** corresponds to the culprit lesion (see table below)
Diffuse Ischemia	• **Type 2 myocardial infarctions** (resulting e.g. from decreased perfusion) can lead to ischemic ST-segment elevations that are not limited to a specific coronary territory.
Normal	• **Normal ST-segment elevation** occurs in 90% of healthy young men in the precordial leads [23]. The ST elevations are concave up, and there are no reciprocal ST depressions.
Early Repolarization	• ST-segment elevation associated with a notch at the J point in V4 is referred to as 'early repolarization.' It is common in healthy young people in the anterior leads. The ST-segment is concave up and the T-waves are upright in V2 – V6. • Early repolarization occurring in the inferior leads, especially with a J-point elevation of > 0.2 mV, appears to be associated with an increased risk of death from cardiac causes and arrhythmias in middle-aged patients [25]. Another study showed an association between early repolarization in the inferolateral leads and ventricular fibrillation [26].

Pericarditis	• ST-segment elevations are **concave (saddle-shaped)** and **diffuse**, i.e. not limited to a specific coronary territory • **Reciprocal ST-segment depressions are absent.** • ST-segment elevation to T-wave amplitude ratio ≥ 0.25 in lead V6 strongly suggests pericarditis [27].
LVH	• ST-segment elevation in the precordial leads can occur in the context of left ventricular hypertrophy
LBBB	• A LBBB results in ST-segment elevation in the precordial leads. • A pacemaker that stimulates the right ventricle will also result in a LBBB pattern.
Hyperkalemia	• Elevated ST-segments can occur in the context of hyperkalemia, so-called pseudoinfarct pattern [5].
Hypercalcemia	• Elevated ST-segments can occur in the context of severe hypercalcemia [28-30]
Brugada	• The Brugada pattern consists of [31]: o a downward sloping ST-segment elevation in leads V1 + V2 o a complete or incomplete right bundle branch block • The Brugada pattern is due to a heritable defect in sodium channels in the myocytes. It may be associated with life-threatening arrhythmias and/or a family history of sudden cardiac death, in which case the criteria for the so-called Brugada syndrome are fullfilled and an implantable cardiodefibrillator is recommended.
Flutter	• Flutter-waves may lead to ST-segment elevation.
Takotsubo Cardiomyopathy	• Takotsubo cardiomyopathy is also referred to as apical ballooning syndrome, stress cardiomyopathy and broken heart syndrome [32] • ST-segment elevation on EKG which usually yields to T-wave inversions within hours • The condition is characterized by the acute onset of chest pain, dyspnea, shock, the absence of pathological coronary artery obstruction on angiogram, left ventricular systolic dysfunction with ballooning of the apex on angiography in conjunction with normal or increased contraction of the basilar area. Prior to angiography, the condition cannot be clinically distinguished from OMI.

ST-Elevation from STEMI Checkmark Sign lead III	ST-Elevation from Pericarditis	ST-Elevation from Brugada Pattern
		

ST-Segment Elevation in a healthy 22-year-old man	ST-Segment elevation from hyperkalemia ("Pseudoinfarct Pattern")	ST-Segment elevation from Flutter-waves
		

Culprit Coronary Lesion

The location of the ST-segment elevations suggests the location of the culprit lesion in the coronary circulation [33]. Reciprocal ST-segment depressions predict a larger infarct distribution, more severe pump failure, an increased severity of underlying coronary artery disease and a worse prognosis [34].

ST-SEGMENT ELEVATION	ST-SEGMENT DEPRESSION	CULPRIT LESION
• **V1-V4, I, aVL** • Often in aVR	• **II, III, aVF > 1 mm** • Often V5	• Proximal LAD above the first septal and first diagonal branches, resulting in extensive anterior wall / anterobasal ischemia [33]. • Elevation in V1 > 2.5 mm SN 100% SP 98%, ST-depression in II, III, aVF > 1 mm SN 34% SP 98% for proximal LAD occlusion [35].
• **V2-V4, aVL** • Absent in V1	• **III**	• LAD between the first septal and the first diagonal, resulting in anterior wall ischemia [33] • ST-elevation in I, aVL and V2 with ST depression in III suggests acute occlusion of the first diagonal branch of the LAD (D1), a so-called "high lateral" myocardial infarction [36], also referred to as the South African flag pattern [37]
• **V3-V6** • Sometimes in II, III, aVF • Absent in V1, aVR, aVL	• Absent in II, III,aVF	• LAD below the first septal and first diagonal branches, resulting in mid anterior wall ischemia [33]; SN 66% SP 73% [35]
• **V5, V6, I, aVL** • **II ≥ III**	• **V1-V3**	• Proximal LCx, resulting in lateral wall ischemia [33]; SN 83% SP 96% for LCx [35]
• **II, III, aVF**	•	• PDA resulting in inferior wall ischemia [33]
• **III > II**	• **I and/or aVL**	• STE III > II had SN 89% SP 68% for RCA as opposed to LCx. If the patient also has STD aVL > I then SN 80% SP 92% [38].
• **III > II, V4R** • Often in V1	• **I & aVL** • Absent in V1-V3	• Proximal RCA lesion, resulting in RV and inferior wall ischemia [33] • SN 79% SP 100% [35]. • ST-elevation in V4R SN 88% SP 78% for RV-infarction [39].
• **V7-V9**	• **V1-V3**	• Posterior wall ischemia [33].

Posterior Infarction

The following suggest posterior OMI [40]:

- Maximal findings V1-V4
- Horizontal ST-depression
- Upright T-waves
- R/S > 1 in V2

To assess the posterior wall, the posterior V7-V9 electrodes are placed on the back at the same segmental level as the V4-V6 leads with the V8 lead just inferior to the tip of the scapula [21]. V7-V9 electrodes can reveal an isolated posterior OMI and are recommended in the setting of ST-depressions in V1-V3 [41].

High lateral infarction

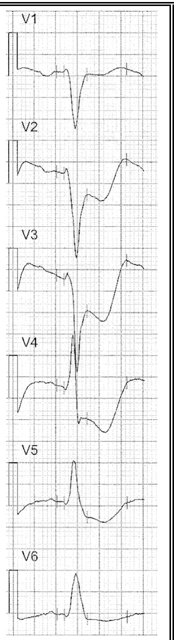
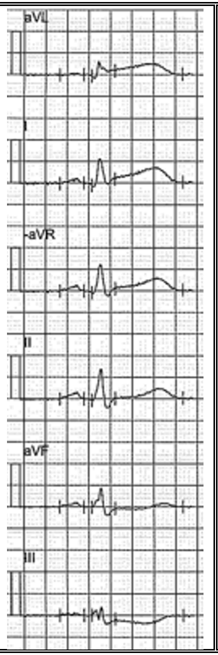
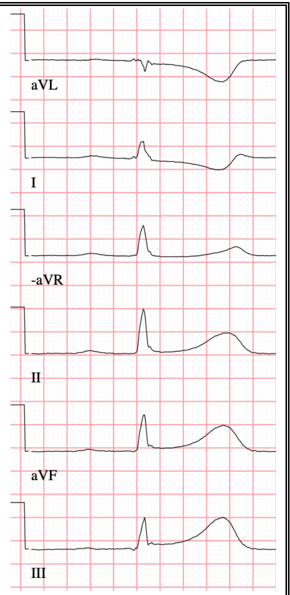
The high lateral wall of the left ventricle is supplied by either:

- the first diagonal branch of the left anterior descending (LAD) artery
- the obtuse marginal branches of the left circumflex (LCx) artery

A high lateral infarction manifests as:

- STE in aVL and I
- STD in III
- STE in V2

When the EKG is presented in the classic format, these EKG-manifestations are within the green part of the South African flag, hence the term "South African Flag sign" [37].

Posterior OMI		**High-Lateral OMI**		**Inferior OMI**	
Maximal ST-depression in V1-V4 and upright-T waves. To assess the posterior wall, V7-V9 leads placed on the back at the same level as the V4-V6 leads		ST-elevation in I, aVL and V2; ST-depression in III		ST-elevation in III; ST-depression in aVL	

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Right Ventricular Infarction
Right ventricular (RV) infarction is associated with 40% of inferior infarctions [21]. In patients with inferior STEMI, right ventricular infarction is suggested by (http://lifeinthefastlane.com/ecg-library/right-ventricular-infarction/):

- ST elevation in V1 - the only standard ECG lead that looks directly at the right ventricle
- ST elevation in V1 > ST elevation in V2
- Isoelectric ST-segment in V1 and markedly depressed ST-segment in V2
- ST elevation in lead III > lead II - because lead III is more 'rightward facing' than lead II and hence more sensitive to the injury current produced by the right ventricle

The right-sided chest lead V4R is more sensitive for the detection of right ventricular infarction and is recommended in the presence of an inferior OMI [41]. The V4R lead is obtained by placing the electrode in the right 5th intercostal space in the mid-clavicular line, 'mirroring' the placement of the V4 electrode. ST-elevation > 1 mm in V4R with an upright T wave is the most sensitive electrocardiographic sign of RV-infarction [35]. One study found that ST-elevation V4R had SN 88% and SP 78% for RV-infarction among patients with inferior OMI [39].

Aslanger Pattern
The following suggests acute inferior OMI (more often occlusion or near occlusion of the circumflex artery than the right coronary artery) with at least one accompanying stable but critical stenosis in one of the non-infarct-related arteries disease [42]:

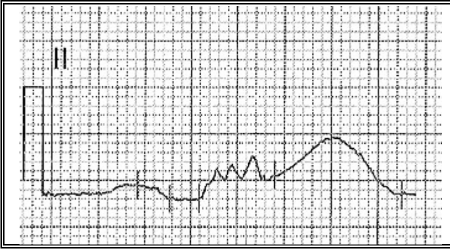
- Inferior ST-elevation isolated to lead III
- Concomitant ST-depression in any of V4-V6, with a positive/terminally positive T-wave
- ST-segment in V1 > V2

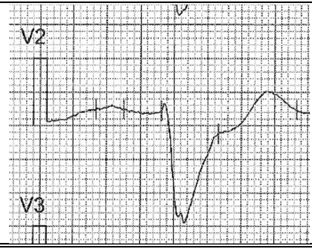
Left Main Coronary Artery (LMCA) Occlusion
Occlusion of the LMCA classically manifests as follows:

- ST-segment elevation in lead aVR, often with concurrent ST-segment elevation in lead aVL and/or lead I, is a key finding.
- This is frequently accompanied by widespread ST-segment depression, especially in the lateral and inferior leads (II, III, aVF, V4–V6).
- Absence of ST-segment elevation in V1 is a distinguishing feature compared to proximal LAD occlusion
- Widespread ST-segment depression, most pronounced in leads V4–V6, is common and reflects global subendocardial ischemia
- Anterior ST-segment elevation (V2–V6) and ST elevation in I and aVL may be seen, particularly in cases without significant collateral circulation.
- QRS left axis deviation, bifascicular block, fragmented QRS, and prolonged QRS and QTc intervals are associated with more severe ischemia and worse prognosis.
- Right bundle branch block (RBBB) with left anterior fascicular block and marked left axis deviation may be present.
- De Winter pattern (upsloping ST depression with tall, symmetrical T waves in precordial leads) and “triangular” or “lambda-like” QRS-ST-T complexes have also been reported in LMCA occlusion.
- The ECG pattern may be dynamic, reflecting the degree of ischemia and presence or absence of collateral flow.

Modified Sgarbossa and Barcelona Criteria for OMI in LBBB
Acute coronary occlusion if ≥ 1 of the following criteria is present:

Modified Sgarbossa	Barcelona
• ST-depression > 1 mm in V1, V2 or V3 • ST-elevation > 1 mm concordant with the QRS • ST-elevation > 1 mm and ≥ 25% of the amplitude of the S wave (STE/S wave ≤ -0.25)	• ST-deviation > 1 mm concordant with QRS • ST-deviation > 1 mm discordant with QRS in leads with max R or S ≤ 6 mm





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OMI with Pacemaker-Induced Left Bundle Branch Block

Both Modified Sgarbossa and Barcelona criteria can detect OMI in pacemaker-induced LBBB.		

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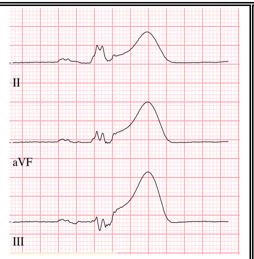
Hyperacute T-waves		de Winter EKG Pattern	
Tall, bulky symmetrical T-waves. Area under the curve more useful than height. Precedes ST-elevation		Upsloping 1- to 3-mm ST-segment depression at the J point in leads V1-V6 that continues into a tall, positive symmetrical T wave. Suggests occlusion of the proximal LAD	

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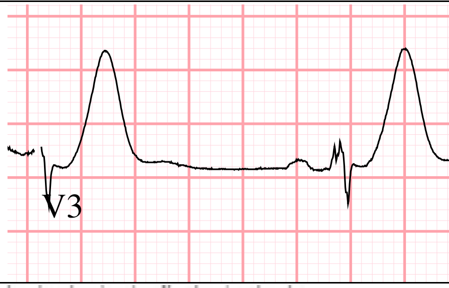
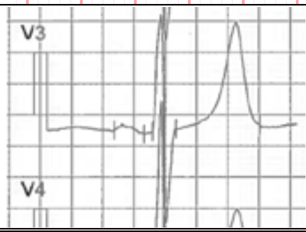
Differential Diagnosis of ST-Segment Depression (adapted from [4])

Pathophysiology	Characteristics
Ischemia	• ST-segment depression from subendocardial ischemia • Reciprocal ST-segment depression from a transmural myocardial infarction
LVH / RVH	• ST-segment depression resulting from ventricular hypertrophy is termed 'strain pattern.'
BBB	• Bundle branch blocks lead to ST-segment depression in certain leads.
Medications	• 'Scooping" or 'coving' ST-segment depression suggests a pharmacological effect, e.g. secondary to **digoxin** [43].
Metabolic	• **Hypokalemia** can result in ST-segment depression

T-Wave

The T-wave is the EKG deflection that follows the QRS-complex. Where the ST-segment ends and the T-wave beginnings is somewhat arbitrary [4]. The T-wave corresponds to part of ventricular repolarization [4]. Normal T-waves are asymmetrical, with a slower rise to peak or descent to trough and faster return to baseline [4].

Differential Diagnosis of Large Positive T-Waves

Pathophysiology	Characteristics	
Myocardial ischemia	• **Hyperacute T-waves** refer to tall, symmetrical, broad-based T-waves seen in the acute phase of a transmural infarction [4], possibly resulting from localized extracellular hyperkalemia [44]	
Hyperkalemia	• **'Tenting' and 'peaking' of the T-wave** refer to tall, symmetrical, narrow-based T-waves generally considered to be the earliest EKG sign of hyperkalemia [5]	

Hyperacute T-Waves in Leads V2-V3 Prior to the Development of ST-Elevation

	
Time: 19:56	**Time: 20:00**

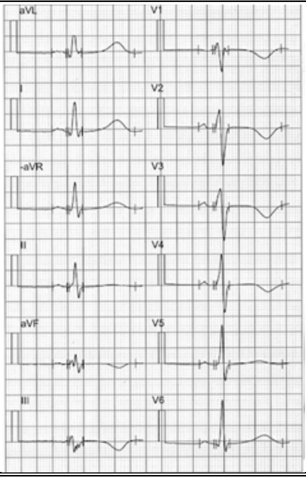
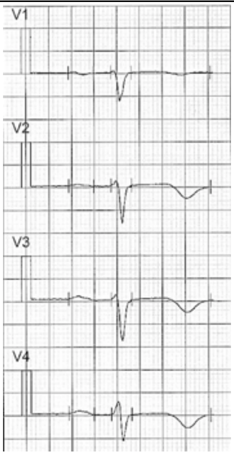
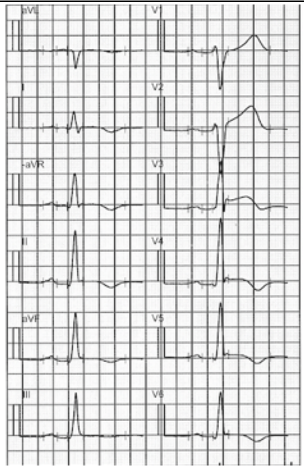
T-Wave in V1

A normal EKG has a flat or inverted T-wave in V1.

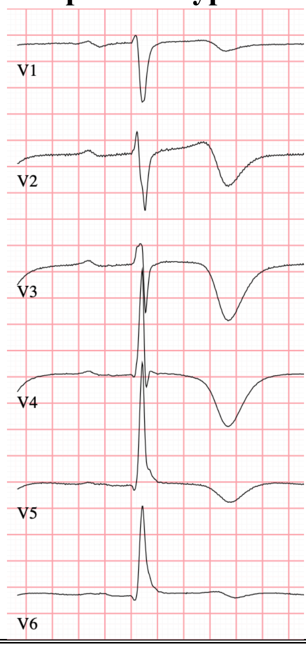
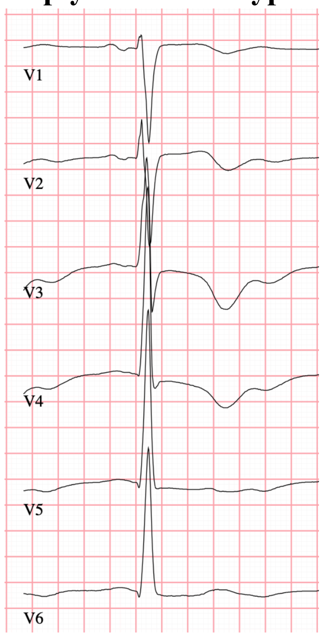
A new upright T-wave in V1 (compared with old EKG) (NTTV1) or a T-wave in V1 taller than T-wave in V6 ("loss of precordial T-wave balance") can be an early sign of ischemia [45] https://www.aliem.com/tall-t-wave-lead-v1/, https://litfl.com/t-wave-ecg-library/,

Differential Diagnosis of Negative T-Waves

Pathophysiology	Characteristics
Normal	• **Normal, negative T-waves** can be seen in leads with a negative QRS-complex, e.g. in V1 [4]
Left ventricular hypertrophy	• The typical left ventricular strain pattern consists of an initially convex (dome-shaped), gradually downward sloping ST-segment leading to an inverted, asymmetric T-wave with an abrupt return to the baseline in the lateral leads (I, aVL, V5, V6) [46]
Pulmonary embolism	• Negative T-waves in the precordial leads (V1-V4) are often seen in patients with acute coronary syndrome (ACS) and pulmonary embolism (PE). In a series of 300 patients with negative precordial T-waves, the presence of negative T-waves in both III and V1 suggested PE as opposed to ACS (SN 90%, SP 97%) [47]. Lead III faces the inferior region of the right ventricle while leads V1 and V2 face its anterior region [47]. Pressure overload may impair coronary flow and the inverted T-waves may reflect ischemia. The same findings may be expected in other conditions of RV pressure overload, e.g. pulmonary hypertension.
Myocardial infarction	• Negative T-waves occur during the evolving phase of a Q-wave myocardial infarction and sometimes with a non-Q-wave myocardial infarction. The negative T-wave results from a delay in regional repolarization produced by the ischemic injury [4].
Myocardial ischemia	• Deep symmetrical T-wave inversions (type 1) or biphasic T-wave changes (type 2) in V2 and V3, in a patient with a history of angina pain who is pain free, suggest tight LAD stenosis. This pattern is referred to as '**Wellens' syndrome**' or '**LAD coronary-T-wave syndrome**' and suggests left anterior descending artery stenosis [48-51]. Cardiac enzymes may be negative. These patients do not require immediate percutaneous intervention but should be admitted for coronary angiography.
Takotsubo	• Takotsubo (stress) cardiomyopathy is a cardiac syndrome characterized by ST-segment elevation, negative T-waves, elevated cardiac enzymes and transient left ventricular apical ballooning without obstructive coronary disease. In a series of 300 patients with negative T-waves in the precordial leads, negative T-waves in leads -aVR and no negative T-waves in lead V1 identified Takotsubo cardiomyopathy with SN 95% and SP 97% [47].
CVA-T-waves	• Very deep, widely splayed negative T-waves may occur in the setting of cerebrovascular accidents such as subarachnoid hemorrhage, and are referred to as 'CVA-T-waves' [4, 52]
Pericarditis	• Diffusely inverted T-waves may be seen weeks following acute pericarditis [4, 6]

T-wave Inversions from Pulmonary Embolism	Wellens Pattern B T-Wave Inversions	Wellens Pattern A Biphasic T-waves	T-wave Inversions from Subacute Pericarditis
			

Wellens T waves: critical LAD stenosis, occlusion followed by spontaneous reperfusion

Biphasic: Type A	Deeply Inverted: Type B
	

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Abnormal T-Wave Morphology

Pathophysiology	Characteristics
Pseudo-normalization	• Pseudonormalization of the T-waves refers to a normal T-wave replacing a negative T-wave in a patient with acute chest pain or angina equivalent. Such a phenomenon suggests acute coronary syndrome [48].
Biphasic, notched T-wave	• The T-waves of patients with hereditary long QT syndromes are frequently abnormal with a biphasic contour or a prominent notched component [53].

QT-Interval

The QT-interval is measured from the Q-wave until the end of the T-wave. It represents ventricular depolarization and repolarization.

QTc-Interval

The QT-interval varies with the heart rate. There are several formulas to "correct" for the heart rate and obtain a QTc, namely Bazett, Fridericia, Framinghame and Hodges. According to the Bazett, QTc = QT / square root of the RR interval expressed in seconds.

The relationship between heart rate and QT-interval varies between individuals, hence no formula has been universally accepted. The lower limit of a normal QTc interval is around 330 msec but has not been well defined [4].

Prolonged QTc-Interval (upper one percent) is > 470 msec in adult men, > 480 msec in adult women and > 460 msec in 1 – 15 year-olds [53]. QTc-prolongation is associated with certain arrhythmias, predominantly the polymorphic ventricular tachycardia called torsade de pointes.

Rischall et al studied the 'Half RR' rule where the QTc is considered prolonged if the QT is > half the RR [54]. Compared to the formulas named above, the Half-RR rule leads to false positives when the heart rate exceeds 60 bpm and false negatives when the heart rate is below 60 bpm. Rischall et al propose the following screening strategy for prolonged QTc:

- If the heart rate exceeds 60 bpm, the half-RR rule is a conservative strategy to rule-out prolonged QTc-interval (but it will yield some false positive results)
- If the heart rate is below 60 bpm, use a fixed uncorrected QT of 485 msec as cut-off

Differential Diagnosis of Prolonged QTc-Interval [4]

Pathophysiology	Examples
Electrolytes	• **Hypokalemia**, **hypomagnesemia**, **hypocalcemia** (less commonly)
Medications	• **Antiarrhythmics**, especially Class IA (Quinidine, pronainamide) and Class III (Ibutilide, Sotalol, Amiodarone) • **Antidepressants**, e.g. tricyclic antidepressants. • **Antipsychotics**, e.g. phenothiazines • **Antihistamines**, e.g. • **Miscellaneous**, see http://www.azcert.org for a complete list
Hereditary	• **Congenital Long QT Syndrome** is caused by 'channelopathies,' i.e. abnormal ion channel function in the heart that result in prolonged repolarization [4].
Ischemia	• **Myocardial ischemia** [4]
Other	• **Cerebrovascular accidents** [4] • **Hypothermia** prolongs the QT-interval by slowing the repolarization of myocardial cells [4]

Differential Diagnosis of Short QTc-Interval

Pathophysiology	Examples
Electrolytes	• **Hypercalcemia**, **hyperkalemia**
Medications	• **Digitalis**

Additional Findings

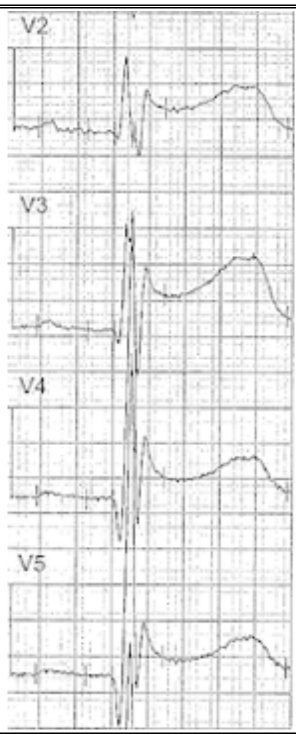
U-Waves

U-waves are positive deflections following the T-waves that are sometimes seen in leads V4-6. They represent the last phase of ventricular repolarization [4]. U waves are usually low voltage (< 0.2 mV) and have the same polarity as the T-wave [44]. Negative U waves may appear after positive T-waves in the setting of left ventricular hypertrophy and myocardial ischemia [4].

Differential Diagnosis of U-Waves

Pathophysiology	Examples
Electrolytes	• **Hypokalemia**, **hypercalcemia**
Metabolic	• **Thyrotoxicosis**
Medications	• **Sotalol**, **phenothiazines**, **digitalis** and other medications [4]
Other	• **Cerebrovascular accidents** can lead to prominent U waves in conjunction with CVA T-waves [4]

Osborn Waves

<table>
<tr>
<td>Osborn waves, also known as prominent J waves, are found in hypothermia [55]. The mechanism appears to be related to the differential effects of systemic cooling on the repolarization of different layers of the ventricles [4].</td>
<td>
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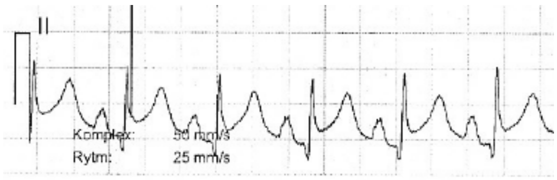
Epsilon Waves

Epsilon waves are low amplitude notches found right after the QRS in the right precordial leads (V1-V3). They suggest Arrhythmogenic Right Ventricular Dysplasia (ARVD), a genetic disorder leading to fibro-fatty changes that can cause sudden cardiac death in young people [56, 57]. Other EKG findings that may be present in patients with ARVD include [58]:

- QRS-duration ≥ 110 msec in V1-V3
- S-wave upstroke (from the nadir of the S-wave to the isoelectric line) ≥ 55 msec in V1-V3 (95% of patients); the interval between the nadir of the S-wave and the end of all depolarization deflections is referred to as the Terminal Activation Duration (TAD).
- T-wave inversions in V1-V3 (85% of patients)
- QRS duration > 110 msec in I [56]

Spodick Sign

Spodick sign refers to down sloping of the TP-segment and suggests acute pericarditis [8].



Rhythm

Sinus Rhythm: see under the Section P-wave above.

AV-blocks: see under the Section PR-interval above.

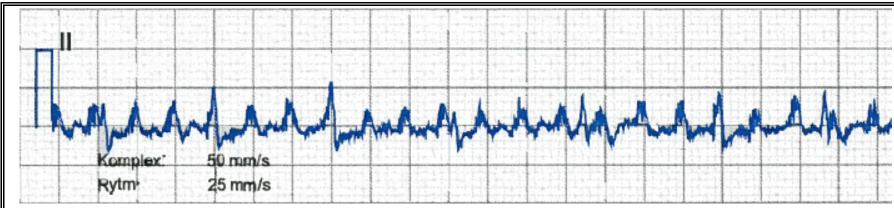
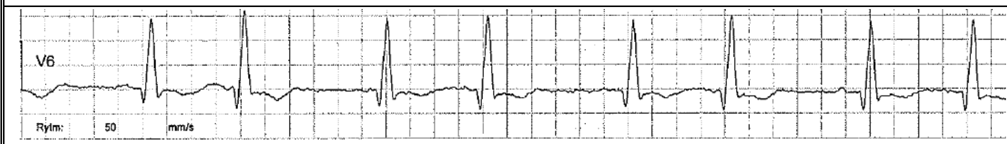
Atrial Flutter: the atrial rate is usually 300 bpm, but it can be slower in patients taking antiarrhythmic medications. Flutter-waves are best seen in lead V1; they may be superimposed upon the T-waves. Patients with atrial flutter and 2:1 conduction usually have a heart rate of approximately 150 bpm, while patients with 3:1 conduction usually have a heart rate of approximately 100 bpm. When the blocking is not constant, the rhythm is irregular.

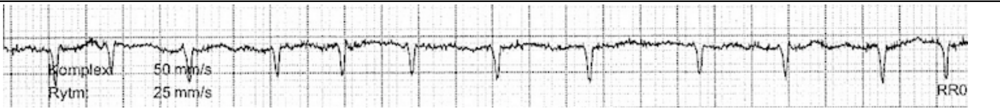
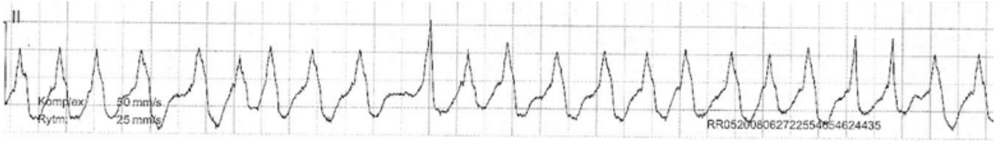
Atrial Fibrillation is likely when no P-waves are present and the rhythm is irregularly irregular, i.e. without any discernible pattern. When combined with a bundle branch block, inhibition of fast sodium channels or pre-excitation, atrial fibrillation results in a wide complex irregular tachycardia. Pre-excitation refers to the abnormal activation of the ventricles via an accessory pathway. An irregular rhythm with wide QRS-complexes and a heart rate > 220 bpm is pathognomonic for atrial fibrillation with pre-excitation [12]. The presence of narrow QRS-complexes amid a background of an irregular tachycardia with wide, monomorphic QRS-complexes, is highly suggestive of atrial fibrillation with pre-excitation.

Ectopic Atrial Tachycardia (EAT): the heart rate is usually 200 - 260 bpm, but may vary between 100 and 300 bpm [59-61]. The P-wave in lead II may be negative.

Atrioventricular Nodal Reentry Tachycardia (AVNRT) results from a re-entry circuit coursing within the AV-node. If the P-waves are visible, the RP-time is short (< 90 msec is 90% specific for AVNRT, < 70 msec even more specific) [59, 62]. The QRS-complexes are narrow.

Atrioventricular Reentry Tachycardia (AVRT) results from a re-entry circuit coursing through the AV-node and along an accessory bypass tract linking an atrium to a ventricle. If the electrical impulse activates the ventricles though the AV-node (and the atria through the bypass tract), the QRS-complexes are narrow and the AVRT is termed orthodromic. If the P-waves are visible, the RP-time is > 90 msec but shorter than the PR-time [62]. If the electrical impulse actives the ventricles through the accessory bypass tract (and the atria are activated retrogradely through the AV-node), then the AVRT is termed antidromic and the QRS-complexes are broad.

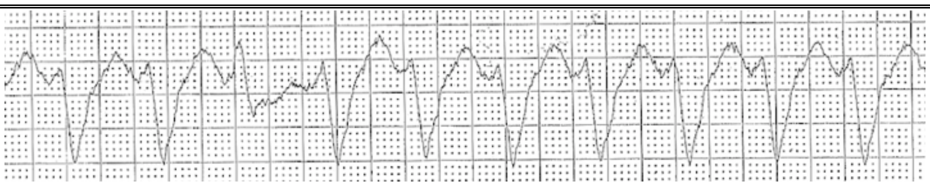
	**Atrial flutter with regular 3:1 conduction, resulting in a heart rate of 100 bpm**
	**Atrial flutter with 2:1 alternating with 3:1 conduction**

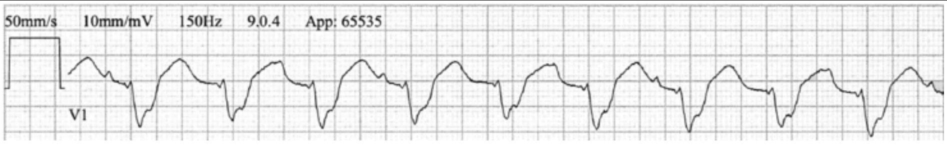
	**Atrial fibrillation (25 mm/s)**
	**Atrial fibrillation with pre-excitation (25 mm/s)**

Ventricular Tachycardia (VT) should be suspected if the heart rate exceeds 130 bpm [63], the rhythm is regular and the QRS-complexes are wide (> 120 msec). The presence of P-waves with a slower rate than the ventricular rate and no correlation with the QRS-complexes (AV-dissociation) is pathognomonic for VT. The presence of occasional QRS-complexes that are narrower than the main QRS morphology, so-called capture beats or fusion beats, indicated AV-dissociation and hence VT.

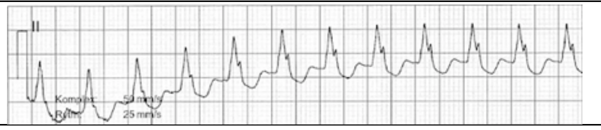
EKG features that suggest that a regular tachycardia with wide QRS-complexes is VT can be organized according to the ABCDEF mnemonic (https://litfl.com/vt-versus-svt-its-as-easy-as-abcde/).

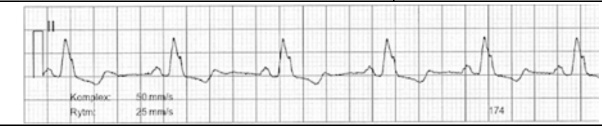
- **A for extreme Axis deviation:** a 'Northwest' axis (QRS negative in leads I and II) suggests VT
- **B for Broad QRS ≥ 160 msec** is strongly associated with VT but may also occur in the presence of class I antiarrhythmics and preexcitation
- **C for Concordance**: **Negative concordance** (all the QRS-complexes in V1-V6 are negative) is virtually diagnostic of VT generated from the anteroapical left ventricle. **Positive concordance** (all the QRS-complexes in V1-V6 are positive) strongly suggests VT generated from the posterobasal LV but may occur with a posterior bypass tract. **Absence of RS-complexes** in all precordial leads, i.e. all QRS-complexes are monophasic R-waves or monophasic S-waves, suggests VT.
- **D for Dissociation: AV dissociation** manifested as P-waves not temporally associated with the QRS-complexes is diagnostic of VT. **Capture beats** (occasional small QRS-complexes) and **fusion beats** (half-widened QRS-complexes) occur when the ventricles are depolarized through the His-Purkinje system. Capture beats and fusion beats are signs of AV-dissociation and are pathognomonic for VT.
- **E for Early wide: RS-complex > 100 msec** (time from onset of the R-wave to nadir of the S-wave) in a precordial lead suggests VT (Brugada sign). **RWPT** (R-wave to Peak Time) is the time from the onset of the QRS to the first change in polarity (either peak of the R-wave or nadir of the Q-wave). **RWPT ≥ 50 msec in lead II** was associated with a LR+ 35 for VT according to one study [64].
- **F for Fragmentation**: **Josephson sign** (notch at the bottom of S-wave in V1 or V2) suggests VT.

	**Ventricular tachycardia with a capture/fusion beat (50 mm/s)**



Ventricular tachycardia with AV-dissociation

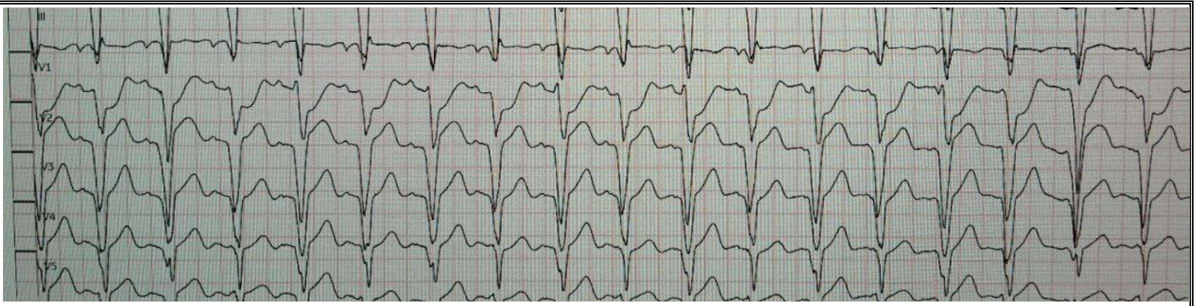




Patient presenting with a wide QRS, regular tachycardia. Prior EKG reveals pre-existing left BBB, suggesting a SVT with BBB instead of VT.

Accelerated Idioventricular Rhythm (AIVR) is a regular wide QRS rhythm with a rate of 50-120 bpm, i.e. slower than VT but faster than a ventricular escape rhythm. The QRS-complexes have neither RBBB nor LBBB morphology. Capture and fusion beats may be present. It is a sign of reperfusion after occlusion and it is one of the OMI (Occlusion Myocardial Infarction) criteria.

Isorhythmic Atrioventricular Dissociation is a cardiac arrhythmia where the atria and ventricles beat independently but at approximally the same rate, creating the illusation of synchronization. The atrial rhythm can be somewhat variable, affected by sympathetic and parasympathetic tone.



Accelerated Idioventricular Rhythm with Isorhythmic Atrioventricular Dissociation

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