# An Overview about Electrocardiogram Role in Acute Myocardial Infarction

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## Abstract

**Background**: Electrocardiogram (ECG) is based on changes of electrical currents of the heart (measured in millivolts). Standard calibration of the ECG is 10mm/mV. Therefore 0.1 mV equals to 1 mm square on the vertical axis. For simplicity, ECG deviations are expressed in mm following the standard calibration. It is recommended to initiate ECG monitoring as soon as possible in all patients with suspected STEMI in order to detect life threatening arrhythmias and allow prompt defibrillation if indicated and when a STEMI is suspected, a 12-lead ECG must be acquired and interpreted as soon as possible at the time of first medical contact to facilitate early STEMI diagnosis and triage. **Objective:** To give an overview about Electrocardiogram role in acute myocardial infarction. **Conclusion:** Acute myocardial ischemia is often associated with dynamic changes in ECG waveform and serial ECG acquisition can provide critical information, particularly if the ECG at initial presentation is non-diagnostic. Recording several standard ECGs with fixed electrode positions at 15 – 30 min intervals for the initial 1 – 2 h, or the use of continuous computer-assisted 12-lead ECG recording (if available) to detect dynamic ECG changes, is reasonable for patients with persistent or recurrent symptoms or an initial non-diagnostic ECG. ECG manifestations suggestive of acute myocardial ischemia: New ST-elevation at the J-point in two contiguous leads with the cut-point: ≥ 1 mm in all leads other than leads V2–V3 where the following cut-points apply: ≥ 2mm in men ≥ 40 years; ≥ 2.5 mm in men < 40 years, or ≥ 1.5 mm in women regardless of age. New horizontal or down-sloping ST-depression ≥ 0.5 mm in two contiguous leads and/or T inversion > 1 mm in two contiguous leads with prominent R wave or R/S ratio > 1.

**Keywords:** Electrocardiogram, Acute Myocardial Infarction

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## Introduction

Electrocardiogram (ECG) is based on changes of electrical currents of the heart (measured in millivolts). Standard calibration of the ECG is 10mm/mV. Therefore 0.1 mV equals to 1 mm square on the vertical axis. For simplicity, ECG deviations are expressed in mm following the standard calibration. **(1)**

ECG uses sensors attached to the skin (electrodes) to record the electrical activity of the heart. A 12-lead ECG system includes 3 limb leads (bipolar), 3 augmented limb leads (unipolar) and 6 precordial leads (unipolar). With regard to the bipolar leads, two electrodes (+ and –) are placed equidistant from the heart, and electricity flow from the negative to the positive electrode is recorded. In the case of the unipolar leads, electricity flow from the center of the heart towards the positive electrode is recorded. The different segments of an ECG trace indicate electrical events during the cardiac cycle. Each lead generates a certain pattern in the trace, corresponding to the electrical activity in the area of the myocardium that is captured by the lead.

Specific changes in certain segments of the ECG trace (indicated by arrows), especially the ST segment, can be documented over time in patients with STEMI. AV, atrioventricular; aVF, augmented vector foot; aVL, augmented vector left; aVR, augmented vector right. **(2)**

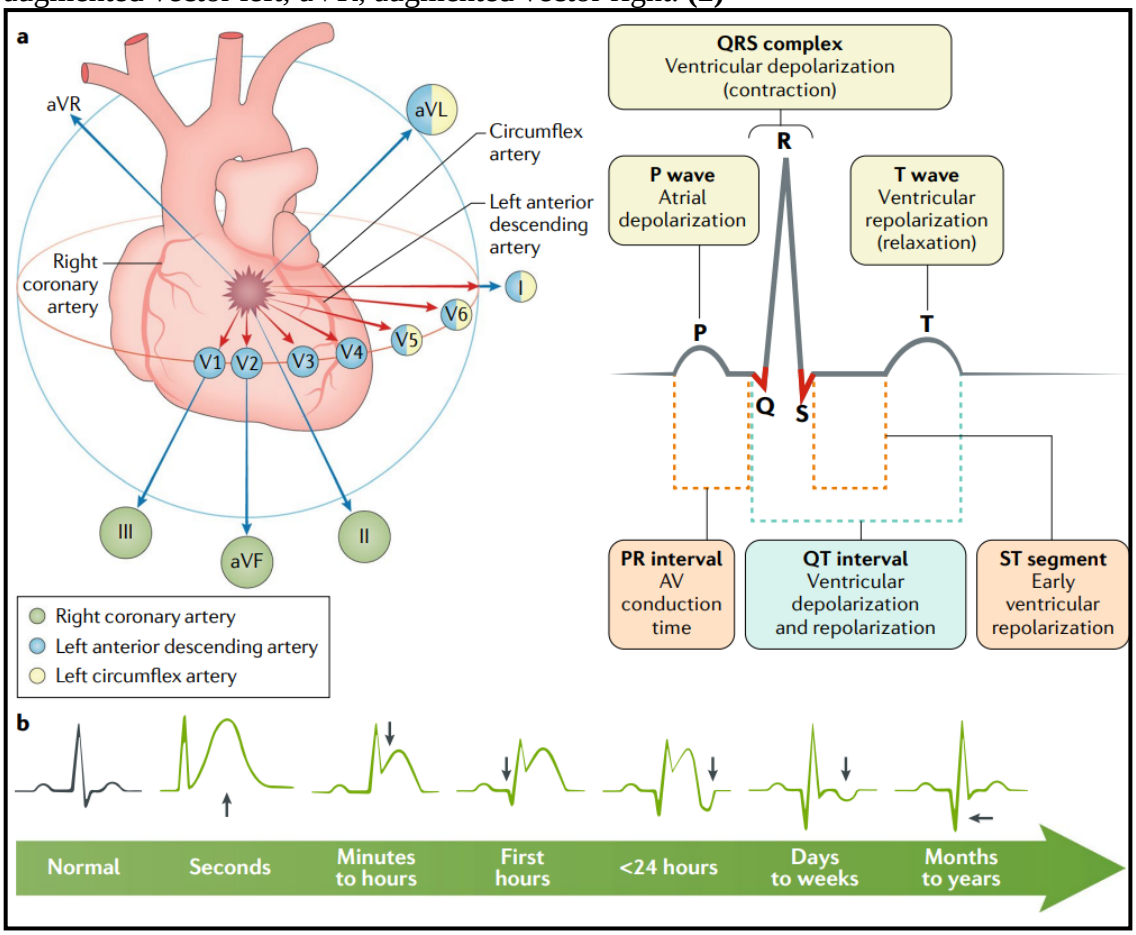


**Figure 1 ;** Healthy and STEMI ECG traces. **(3)**

ECG is the most widely used imaging tool helping in diagnosis and initial management of patients presenting with symptoms compatible with acute coronary syndrome, acute ischemia affects the configuration of the QRS complexes, the ST segments and the T waves. The ECG should be read along with the clinical assessment of the patient. ST segment elevation (and ST depression in leads V1–V3) in patients with active symptoms usually indicates acute occlusion of an epicedial artery with ongoing trans-mural ischemia. Knowing the clinical scenario, comparison to previous ECG and subsequent ECGs (in cases that there are changes in the quality or severity of symptoms) may add in the diagnosis and interpretation in difficult cases. **(4)**

The electrical activity of the heart, as depicted by the surface ECG is affected by ischemia, reperfusion and presence of necrosis or scar. Therefore, the ECG has remained the most widely utilized ancillary tool, along with focused history taking and physical examination for the initial assessment of patients presenting with symptoms compatible with acute coronary syndromes. **(4)**

It is recommended to initiate ECG monitoring as soon as possible in all patients with suspected STEMI in order to detect life threatening arrhythmias and allow prompt defibrillation if indicated and when a STEMI is suspected, a 12-lead ECG must be acquired and interpreted as soon as possible at the time of first medical contact to facilitate early STEMI diagnosis and triage**. (5)**

Acute myocardial ischemia is often associated with dynamic changes in ECG waveform and serial ECG acquisition can provide critical information, particularly if the ECG at initial presentation is non-diagnostic. Recording several standard ECGs with fixed electrode positions at 15 – 30 min intervals for the initial 1 – 2 h, or the use of continuous computer-assisted 12-lead ECG recording (if available) to detect dynamic ECG

changes, is reasonable for patients with persistent or recurrent symptoms or an initial non-diagnostic ECG. **(5)**

**ST-segment criteria suggestive of MI**

**Table (1)** ECG manifestations suggestive of acute myocardial ischemia.

| **ST-elevation** |
|---|
| New ST-elevation at the J-point in two contiguous leads with the cut-point: ≥ 1 mm in all leads other than leads V2–V3 where the following cut-points apply: ≥ 2mm in men ≥ 40 years; ≥ 2.5 mm in men < 40 years, or ≥ 1.5 mm in women regardless of age.[a] |
| **ST-depression and T wave changes** |
| New horizontal or down-sloping ST-depression ≥ 0.5 mm in two contiguous leads and/or T inversion > 1 mm in two contiguous leads with prominent R wave or R/S ratio > 1. |

[a]When the magnitudes of J-point elevation in leads V2 and V3 are registered from a prior electrocardiogram, new J-point elevation >_ 1 mm (as compared with the earlier ECG) should be considered an ischaemic response. For bundle branch block, see section below. Considered an ischemic response. For bundle branch block, see section below. **(6)**

Lists ST-segment–T wave criteria suggestive of acute myocardial ischemia that may lead to MI, the J-point (junction between QRS termination and ST-segment onset) is used to determine the magnitude of the ST-segment shift with the onset of the QRS serving as the reference point. In patients with a stable baseline, the TP segment (isoelectric interval) is a more accurate method to assess the magnitude of ST-segment shift, and in distinguishing pericarditis (PTa depression) from acute myocardial ischemia. Tachycardia and baseline shift are common in the acute setting and can make this determination difficult. Therefore, QRS onset is recommended as the reference point for J-point determination.

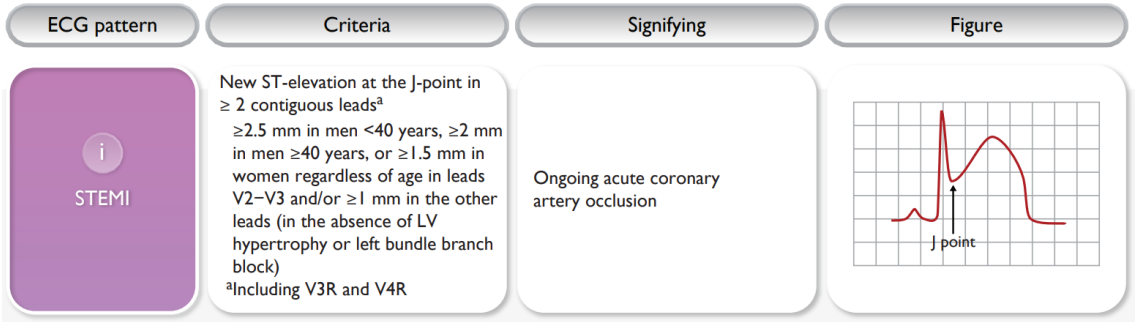


**Figure 2;** the reference point for J-point determination. **(6)**

New, or presumed new, J-point elevation ≥ 1 mm (1 mm = 0.1 mV) is required in all leads other than V2 and V3 as an ischaemic response. In healthy men under age 40, J-point elevation can be as much as 2.5 mm in leads V2 or V3, but it decreases with increasing age. Sex differences require different cut-off points for women, since J-point elevation in healthy women in leads V2 and V3 is less than in men. **(6)**

The criteria in require that the ST shift be present in two or more contiguous leads. For example, ≥ 2 mm of ST-elevation in lead V2 and ≥ 1 mm in lead V1 would meet the criteria of two abnormal contiguous leads in a man ≥ 40 years old. However, ≥ 1 mm and < 2 mm of ST-elevation, seen only in leads V2 –V3 in men (or < 1.5 mm in women), may represent a normal finding. It should be noted that lesser degrees of ST

displacement or T wave inversion than those described can also represent an acute myocardial ischaemic response.

Absence of ST-elevation in the precordial leads, tall, prominent, symmetrical T waves in the precordial leads, up sloping ST-segment depression > 1 mm at the J-point in the precordial leads, and in most cases ST-segment elevation (> 1 mm) in lead aVR or the symmetrical, often deep (> 2 mm), T wave inversions in the anterior precordial leads are associated with significant left anterior descending artery (LAD) occlusion. **(7)**

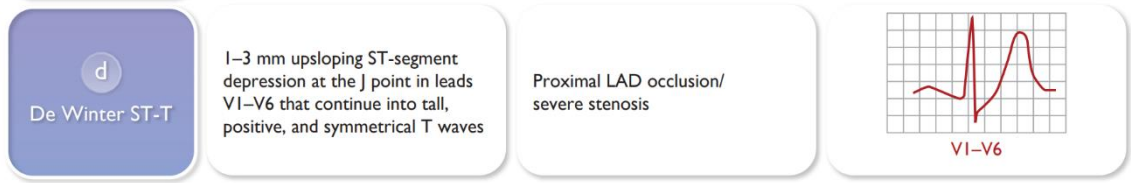


**Figure 3;** De Winter ST-T. **(7)**

ST-elevation in lead aVR > 1 mm may accompany anterior or inferior STEMI, and is associated with increased 30 day mortality in patients with acute MI. Supplemental leads, as well as serial ECG recordings, should be deployed with a very low threshold in patients who present with ischemic chest pain and a non-diagnostic initial ECG**. (8)**

ECG evidence of myocardial ischaemia in the distribution of a left circumflex artery is often overlooked. Isolated ST-segment depression ≥ 0.5 mm in leads V1–V3 may indicate left circumflex occlusion and can best be captured using posterior leads at the fifth intercostal space (V7 at the left posterior axillary line, V8 at the left mid-scapular line, and V9 at the left para-spinal border). Recording of these leads is strongly recommended in patients with high clinical suspicion of acute circumflex occlusion (e.g. initial ECG non-diagnostic or ST segment depression in leads V1–V3). **(8)**

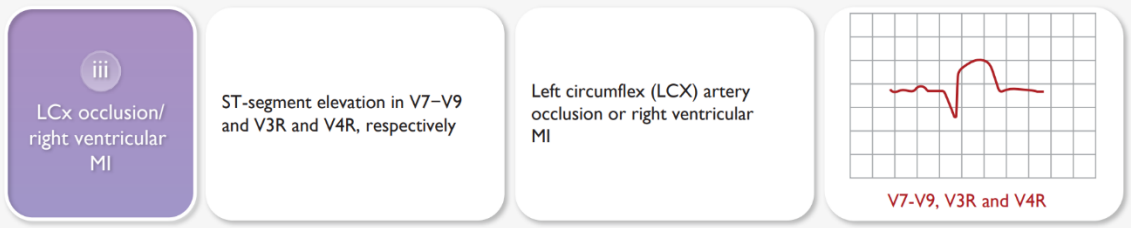


**Figure 4**; LCx occlusion/ right ventricular MI**. (6)**

A cut-off point of 0.5 mm ST segment elevation is recommended in leads V7–V9; specificity is increased at a cut-off point ≥ 1 mm ST-elevation and this cut-off point should be used in men < 40 years old. ST-segment depression in leads V1–V3 may be suggestive of infero-basal myocardial ischaemia (previously termed posterior infarction), especially when the terminal T wave is positive (ST-elevation equivalent); however, this is non-specific. **(9)**

In patients with inferior and suspected right ventricular infarction, leads aVR or V1 may exhibit ST-segment elevation ≥ 1mm.The early recording of right precordial leads V3R andV4R should be performed, since ST-elevation ≥ 0.5 mm (≥ 1 mm in men < 30 years old) provides supportive criteria for the diagnosis. **(10**)

**Contiguous Leads**

Contiguous leads are leads that represent arterial territories that are next to each other in general. **(11)**

- For the precordial leads, the neighboring leads (V3 and V4, for example).
- II, III and AVF are contiguous leads for the inferior portion of the myocardium.
- I and AVL are contiguous leads for the high lateral portion of the heart.
- For the posterior leads, V7, V8 and V9 are contiguous leads.

**Evolution of ECG Changes in STEMI**

The ECG in STEMI changes over time, as when the diagnosis is unclear, repeating ECGs can be helpful in making the diagnosis. **(11)**

In general progression of the ECG changes in a STEMI follows:

- Acute**,** hyper-acute T waves form. These forms in the first minutes to hours of a STEMI. A hyper-acute T wave is characterized by an increase in the amplitude and the width of the T wave.
- Next, ST elevations will develop.

- Followed by the development of Q waves**.**
- Later, the T wave will invert**.**

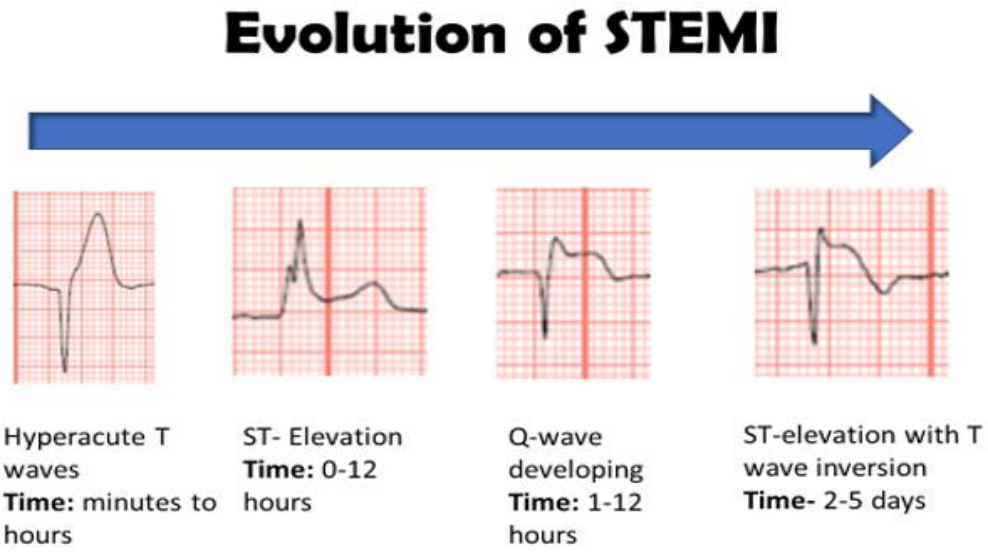


**Figure 5;** *Evolution of ECG Changes in STEMI****.(Sekhon)***

**Reciprocal Changes**

Reciprocal changes in STEMIs are the ST depressions found on the ECG that are on the opposite side of the heart of the myocardial infarction. It is important to note that the absence of reciprocal changes does not preclude the presence of a STEMI**,** but the presence of reciprocal changes makes it more likely. **(12)**

**Table (2)** Reciprocal changes in STEMI.

| **Reciprocal Changes in STEMI** | | |
|---|---|---|
| **STEMI Territory** | **Leads with ST Elevations** | **Leads with Reciprocal ST Depressions** |
| Anterior/Septal | V1-V4 | II, III and AVF |
| Lateral | V5-6, I and AVL | II, III and AVF |
| Inferior Leads | II, III And AVF | I and AVL |
| Posterior | V7, V8 and V9 | V1-V4 |

**Table (3)** STEMI territories and their vasculature. **(6)**

| **STEMI Territories** | | |
|---|---|---|
| **Territory** | **Leads with STEMI** | **Occluded Coronary Artery** |
| Anterior/Septal | V1-V4 | LAD |
| Lateral | V5-6, I and AVL | Left Circumflex or Diagonal Branch of the LAD |
| Inferior Leads | II, III And AVF | RCA and/or Left Circumflex |
| Posterior | V7, V8 and V9 | Posterior Descending Artery |

**Anterior STEMI**

Anterior STEMIs generally result from the occlusion of the left anterior descending coronary artery, and carries the worst prognosis of all STEMIs given the larger infarct size. Anterior STEMIs can be diagnosed by recognizing ST elevations in the anterior leads (V1-V4) and reciprocal changes in the inferior leads (II, III and AVF). **(13)**

**Lateral STEMI**

Lateral STEMIs generally result from the occlusion of the left circumflex coronary artery or the diagonal branch of the left anterior descending. On ECG, this is recognized by ST elevations in the lateral leads (V5-6) or the high lateral leads (I and aVL). It is important to remember that I and aVL are contiguous leads and ST elevations in these leads can signify a high-lateral STEMI. **(14)**

**Inferior STEMI**

In general, inferior STEMIs result from the occlusion of the Right Coronary Artery (70%) or the Left Circumflex. On ECG, this can be recognized with ST elevations in the inferior leads (II, III and AVF) with reciprocal changes in the high lateral leads (I and aVL). With these leads in particular, complete coronary occlusion can occur with less than 1mm in ST elevation in the anterior leads, so a high index of suspicion should be maintained. **(14)**

A subset of inferior STEMIs is right ventricular infarcts, which are preload dependent. This is important to recognize because if they become hypotensive, fluids can be helpful. In addition, right ventricular infarcts should not be given nitroglycerin as it can precipitously drop their blood pressure. A right ventricular infarct in the context of an inferior STEMI can be discovered by the following ECG findings:

- The degree of ST-elevation on Lead III is greater than the degree of ST-elevation on Lead II.
- ST-elevation on Lead V4r on a right sided ECG (performed by flipping the precordial leads onto the right side of the chest **so,** In patients with inferior MI, it is recommended to record right precordial leads ($V_3R$ and $V_4R$) seeking ST-segment elevation, to identify concomitant right ventricular (RV) infarction**. (11)**

**Posterior STEMI**

Acute coronary artery occlusions can present without ST elevations on the 12 lead ECG. This occurs because the normal 12 lead ECG looks at the anterior part of the heart, and only the reciprocal changes of a posterior can be seen on the 12 lead ECG so posterior STEMIs are caused by an occlusion of posterior descending coronary artery. On a normal 12 lead ECG, it can be recognized by: ST depression in the anterior leads, Prominent R waves in leads V2-3 and Upright anterior T waves. **(15)**

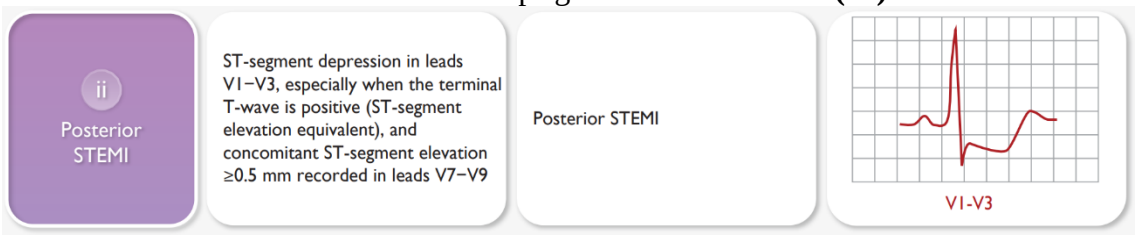


**Figure 6;** Posterior STEM**. (6)**

To help confirm your diagnosis, you can also perform a posterior ECG. This can be performed by placing the leads on the patient's back. The leads should be placed:

- **V7**- At the level of V6, place at the posterior axillary line
- **V8**- At the tip of the scapula at the level of V6
- V**9**- Left para-spinous region at the level of V6.

So, ST-segment depression in leads $V_1$–$V_3$ suggests myocardial ischaemia, especially when the terminal T-wave is positive (STEMI equivalent), and confirmation by concomitant ST-segment elevation ≥ 0.5 mm recorded in leads $V_7$–$V_9$ should be considered as a means to identify posterior MI. Isolated posterior MI in Acute Myocardial Infarction of the inferior and basal portion of the heart, often corresponding to the left circumflex territory, isolated ST-segment depression 0.5mm in leads V1–V3 represents the dominant finding; These should be managed as a STEMI as the use of additional posterior chest wall leads [elevation V7–V9

0.5mm (1mm in men, 40 years old)] is recommended to detect ST-segment elevation consistent with inferior and basal MI**. (16)**

**STEMI Equivalents**

There are ECG morphologies that do not meet the traditional ST elevation criteria but nevertheless may suggest acute coronary occlusion that would benefit from emergent revascularization. As reported by recognition of these STEMI equivalents is crucial to avoid delayed reperfusion. Three such patterns have been increasingly identified as "STEMI equivalents" that require prompt intervention. De Winter T waves indicate proximal occlusion of the left anterior descending (LAD) artery and are distinguished by tall symmetric T waves with associated ST depression in leads V1-V4. Elevation of the ST segment in lead aVR has been associated with left main coronary artery occlusion. Diffuse ST depression with simultaneous ST elevation in lead aVR is the hallmark of the aVR sign. ST elevation can also be present in V1. The higher the ST elevation, the more severe the prognosis. **(17)**

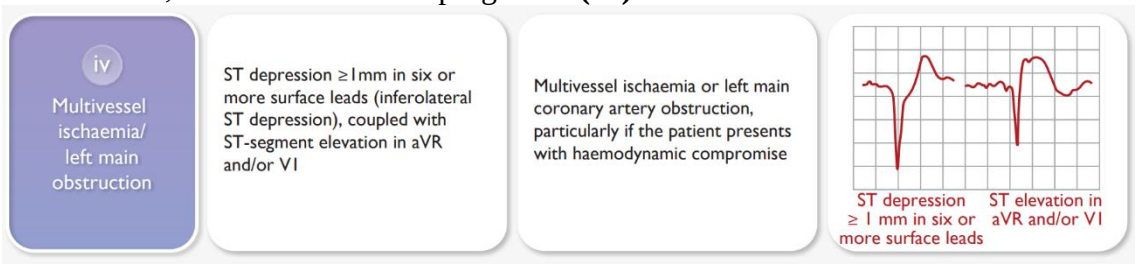


**Figure 7;** left main occlusion/equivalent **(6)**

Wellen's syndrome has been described in two forms, both of which suggest critical LAD stenosis. Type A is identified by biphasic T waves in leads V2-V3, whereas Type B is characterized by deep T waves in V2-V3. **(18)**

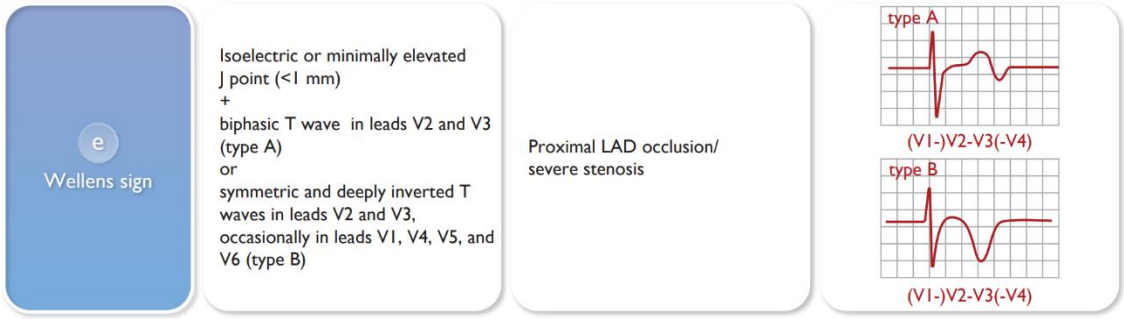


**Figure 8;** Wellens signs. **(6)**

**Left Bundle Branch Block with Sgarbossa Criteria**

The ECG diagnosis may be more difficult in some cases, which nevertheless deserve prompt management and triage. Among these: Bundle branch block. In the presence of LBBB, the ECG diagnosis of AMI is difficult but often possible if marked ST-segment abnormalities are present. Somewhat complex algorithms have been offered to assist the diagnosis**.** The presence of concordant ST-segment elevation (i.e. in leads with positive QRS deflections) appears to be one of the best indicators of ongoing MI with an occluded infarct artery Concordant ST segment changes include: ST segment elevation with a positive QRS amplitude and ST segment depression with a negative QRS amplitude. Discordant ST changes are changes when the amplitude of the QRS segment is opposite to the deviation of the ST segment. For example, discordant ST segment changes include; ST segment elevation with a negative QRS amplitude and ST segment depression with a positive QRS amplitude. **(19)**

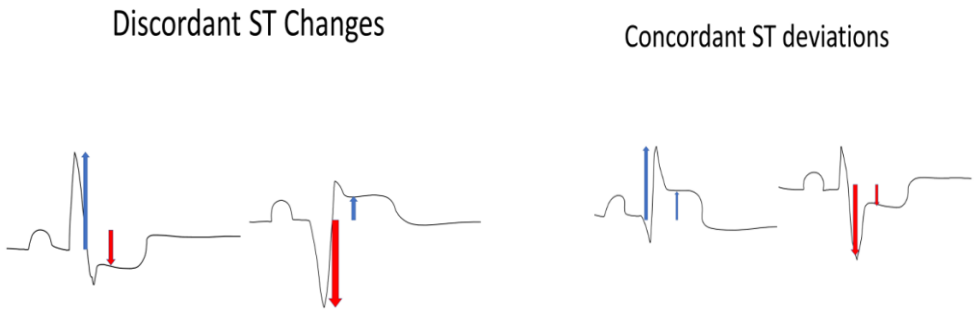


**Figure 9;** Sgarbossa Criteria (Image courtesy of Navdeep Sekhon, MD*).*

Patients with MI and right bundle branch block (RBBB) have a poor prognosis. It may be difficult to detect transmural ischaemia in patients with chest pain and RBBB. Therefore, a primary PCI strategy (emergent coronary angiography and PCI if indicated) should be considered when persistent ischaemic symptoms occur in the presence of RBBB. **(20)**

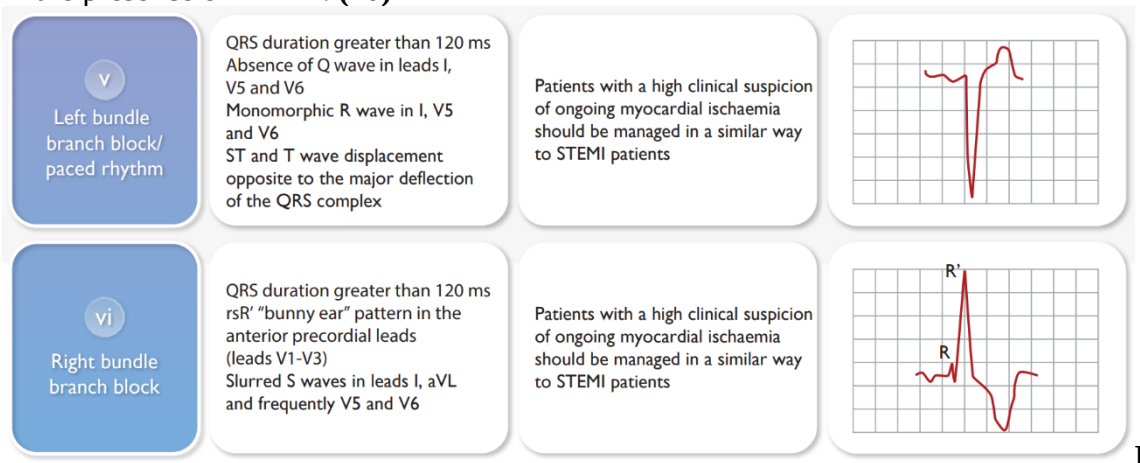


**Figure 10;** Left and right Bundle Branch Block. **(6)**

**Table (4)** lists the atypical ECG presentations that should prompt a pPCI strategy in patients with symptoms consistent with myocardial ischemia;

| |
|---|
| **Bundle branch block**<br>Criteria that can be used to improve the diagnostic accuracy of STEMI in LBBB[50]:<br>• Concordant ST-segment elevation ≥1 mm in leads with a positive QRS complex<br>• Concordant ST-segment depression ≥1 mm in $V_1$–$V_3$<br>• Discordant ST-segment elevation ≥5 mm in leads with a negative QRS complex<br>The presence of RBBB may confound the diagnosis of STEMI |
| **Ventricular paced rhythm**<br>During RV pacing, the ECG also shows LBBB and the above rules also apply for the diagnosis of myocardial infarction during pacing; however, they are less specific |

| |
|---|
| **Isolated posterior myocardial infarction**<br>Isolated ST depression ≥0.5 mm in leads $V_1$–$V_3$ and ST-segment elevation (≥0.5 mm) in posterior chest wall leads $V_7$–$V_9$ |
| **Ischaemia due to left main coronary artery occlusion or multivessel disease**<br>ST depression ≥I mm in eight or more surface leads, coupled with ST-segment elevation in aVR and/or $V_1$, suggests left main-, or left main equivalent- coronary obstruction, or severe three vessel ischaemia |

**ESC Guidelines (6)**ECG = electrocardiogram; LBBB = left bundle branch block; RBBB = right bundle branch block; RV = right ventricular; STEMI = ST-segment elevation myocardial infarction

**Fragmented QRS complexes**

Fragmented QRS is defined as QRS complexes with the presence of an additional R wave (R') or notching in the nadir of the R wave or the S wave, or the presence of >1 R' (fragmentation) in 2 contiguous leads, corresponding to a major coronary territory. Typical bundle branch block (BBB) pattern (QRS ≥ 120 ms) and incomplete right BBB were excluded from the original definition. **(21)**

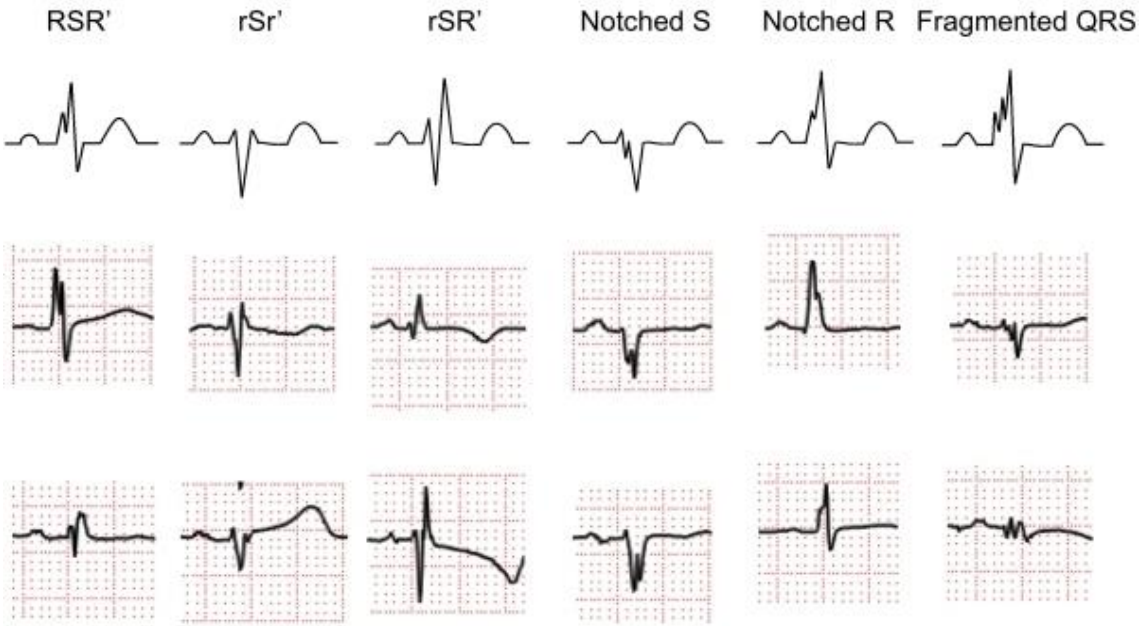


**Figure 11;** Classification of fragmented QRS (various RSR' patterns). Various RSR' patterns are present in the mid precordial lead or infesrior lead. **(6)**

It has been show in some studies that a subtle abnormality within the QRS complex can represent conduction disturbance and myocardial scar. A notch in the QRS complex in patients with left ventricular hypertrophy has been suggested to be a result of an intraventricular conduction defect. Fragmentation originates from injured tissue around an infarct scar where ventricular activation is delayed and asynchronous, resulting in the RSR' pattern of the QRS complex in 12-lead electrocardiography. Myocardial single photon emission tomography (SPECT) can identify regional perfusion abnormalities from a scar by a prior myocardial infarction, Studies in which the diagnostic values of Q wave and fQRS for a myocardial scar detected by SPECT were compared showed that fQRS was associated with significantly greater perfusion and functional abnormalities than was the Q wave. **(22)**

The presence of fQRS in anterior leads (V1-5) predicts myocardial scar in the anterior myocardial segment or in the left anterior descending territory. The presence of fQRS in lateral leads (I, aVL, and V6) predicts myocardial scar in the lateral myocardial segment or left circumflex territory myocardial scar. The presence of fQRS in inferior leads (II, III, and aVF) predicts myocardial scar in the inferior myocardial segment or in the right coronary artery territory. **(21)**

**Prognostic value of fQRS in coronary artery disease**

Since fQRS represents myocardial scar, fQRS may be associated with heart failure and ventricular tachyarrhythmia. Some studies have shown a relationship between existence of fQRS in patients with CAD and prognosis. In patients having narrow QRS complexes (< 120 ms), all-cause mortality and cardiac event rate were higher in patients with fQRS than in patients without fQRS and the presence of fQRS in 2 or more contiguous leads, they showed that the presence of fQRS in 3 or more leads was the most useful for distinguishing between patients with and without risk for cardiac death or hospitalization. An increase in the number of leads with fQRS would represent a wide scar area, which would result in an adverse outcome. The presence of fragmented QRS is associated with increased risk of in-hospital mortality, long-term mortality, and MACE in patients with AMI, while transient fQRS cannot be ignored. Moreover, it is related to an increased risk of in-hospital heart failure and VT/VF events. In the presence of fQRS, the OR for in-hospital mortality is higher than that for long-term mortality.**(22)**

fQRS on 12-lead ECG developed in 55% of patients with ST elevation myocardial infarction (STEMI) and in 50% of patients with non ST elevation myocardial infarction (NSTEMI), but in only 3.7% of patients with unstable angina pectoris (UAP). A new Q wave occurred in 44% of patients with STEMI, 23% of patients with NSTEMI and 0.4% of patients with UAP. Although the sensitivities of fQRS for STEMI and NSTEMI were 55% and 50% respectively, the specificity of fQRS for AMI was 96%. All-cause mortality of patients with fQRS was higher than that of patients without fQRS. In multivariate analysis, fQRS was an independent predictor for all-cause mortality.**(5)**

Therefore; fQRS is a useful marker of myocardial scar and can predict cardiac events and mortality in various heart diseases. However, some studies showed negative data of fQRS for diagnosis and prognosis of the diseases. For improvement of diagnosis and prediction of prognosis, qualitative analysis of fQRS might be required.**(5)**

**Pathologic Q waves**

Q wave criteria associated with MI and an increased relative risk of death are illustrated in the following table;

**Table (5)** Electrocardiographic changes associated with prior myocardial infarction (in the absence of left ventricular hypertrophy and LBBB)

| |
|---|
| Any Q wave in leads $V_2$–$V_3$ > 0.02 s or QS complex in leads $V_2$–$V_3$. |
| Q wave ≥ 0.03 s and ≥ 1 mm deep or QS complex in leads I, II, aVL, aVF or $V_4$–$V_6$ in any two leads of a contiguous lead grouping (I, aVL; $V_1$–$V_6$; II, III, aVF).[a] |
| R wave > 0.04 s in $V_1$–$V_2$ and R/S > 1 with a concordant positive T wave in absence of conduction defect. |

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[a]The same criteria are used for supplemental leads $V_7$–$V_9$. s = seconds.

The specificity of the ECG diagnosis for MI is greatest when Q waves occur in several leads or lead groupings, or are > 0.04 s, when the Q waves are associated with ST deviations or T wave changes in the same leads, the likelihood of MI is increased; for example, minor Q waves >_ 0.02 s and < 0.03 s that are >_ 1 mm deep are suggestive of prior MI if accompanied by inverted T waves in the same lead group and non-invasive imaging techniques also provide important supportive evidence of prior MI. In the absence of non-ischaemic causes, regional myocardial thinning, scar or reduced wall motion shown by echocardiography, myocardial perfusion scintigraphy (MPS) with single photon emission computed tomography (SPECT) or positron emission tomography (PET), or magnetic resonance imaging provide strong evidence for prior MI, particularly when ECG criteria are equivocal.**(6)**

The presence of Q waves on the presenting ECG is believed to indicate more extensive myocardial involvement and has been shown to be an independent predictor of adverse clinical outcomes after STEMI; however, baseline predictors of the propensity to develop Q waves are incompletely characterized, and it is currently unknown whether the association between Q waves on the presenting ECG and mortality is predominantly influenced by baseline patient characteristics or the rapidity of mechanical revascularization based on the current guideline-driven metric of door-to-balloon time (DTB**).** Pathologic Q waves increase the prognostic risk associated with acute myocardial ischaemia include cardiac arrhythmias, intraventricular bundle branch blocks, atrioventricular conduction delays, and loss of precordial R wave amplitude, a less specific finding. The ECG by itself is often insufficient to diagnose acute myocardial ischaemia or infarction, since ST deviation may be observed in other conditions, such as acute pericarditis, LV hypertrophy (LVH), left bundle branch block (LBBB), Brugada syndrome, takotsubo syndrome (TTS), and early repolarization patterns. **(23)**

A prior ECG is often helpful in distinguishing a new from a chronic finding, but should not delay the decision for treatment. Prolonged new convex ST-segment elevation, particularly when associated with reciprocal ST-segment depression, usually reflects acute coronary occlusion and results in myocardial injury with necrosis. Reciprocal changes can help to differentiate STEMI from pericarditis or early repolarization changes. As in cardiomyopathy, Q waves may also occur due to myocardial fibrosis in the absence of CAD. Some of the earlier manifestations of myocardial ischemia are typical T wave and ST-segment changes. Increased hyperacute T wave amplitude, with prominent symmetrical T waves in at least two contiguous leads, is an early sign that may precede the elevation of the ST-segment. In general, the development of new Q waves indicates myocardial necrosis, which starts minutes/hours after the myocardial insult. Transient Q waves may be observed during an episode of acute ischemia or (rarely) during acute MI with successful reperfusion.**(4)**

**T-wave**

**Normal T-wave Etiology**

Normally, the T wave is formed at the end of the last phase of ventricular repolarization. Ventricular repolarization is the process by which the ventricular myocytes return to their negative resting potential so they can depolarize again. While this phase of the cardiac cycle is rapid, an upright low amplitude broad hump following the QRS complex is seen in normal T wave morphology. **(23)**

Normal T waves are upright in leads I, II, and V3-V6, inverted in AVR. Less than five mm in limb leads, less than ten mm in precordial leads, and variable presentations in III, AVL, AVF, and V1-V2. This graphical depiction on ECG is associated with lead placement and the electrical pathways of the heart. **(23)**

**Abnormal T-wave Pathophysiology**

T wave changes are secondary to electrolyte abnormalities in the myocardium since the ECG is representative of the electricity of the heart. The outflow of potassium from the myocyte during repolarization is necessary to restore resting membrane potential. In disease states such as ischemia, the Na/K-ATPase cannot function to restore this gradient; when there is hyperkalemia, the electrochemical gradient for potassium to flow out of the cell is skewed, altering the repolarization phase. These changes during phase three of the action potential are reflected by abnormalities in the T wave on an ECG. **(24)**

The transition from ST segment to T-wave should be smooth. The amplitude of the T-wave is rarely >6 mm in the limb leads. In the chest leads the amplitude is highest in V2–V3; males may display up to 10 mm T-wave amplitude in these leads, although most have <6 mm in V2–V3. T-wave amplitude is on average 3 mm in V2–V3 in females and it rarely exceeds 8 mm. Note that the amplitude of the T-wave is related to the amplitude of the QRS complex (large QRS amplitudes yield large T-wave amplitudes, and vice versa). The normal T-wave is slightly asymmetric; the slope of the descending limb is slightly steeper than the ascending limb. **(24)**

**The inverted (negative) T-wave**

T-wave inversion means that the T-wave is negative. By definition, the T-wave is negative if the terminal portion of the T-wave is below the baseline. T-wave inversions are actually graded according to the amplitude (depth). Strictly speaking the term T-wave inversion refers to T-waves that are 1 to 5 mm negative (deep). The term deep T-wave inversion is applied to T-waves 5 to 10 mm deep. The term gigantic T-wave

inversion is used if the T-wave is deeper than 10 mm. myocardial ischemia may present with any degree of T-wave inversion. Myocardial ischemia may also present with flat T-waves, which are defined as T-waves with an amplitude between +1 and -1 mm. **(25)**

The following events is thought to occur with T wave changes:

- A sudden complete occlusion of the LAD causes a transient anterior STEMI, causing chest pain & diaphoresis. This stage may not be successfully captured on an ECG recording.
- Re-perfusion of the LAD (e.g. due to spontaneous clot lysis or prehospital aspirin) leads to resolution of chest pain. ST elevation improves and T waves become biphasic or inverted. The T wave morphology is identical to patients who reperfuse after a successful PCI.
- If the artery remains open, the T waves evolve over time from biphasic to deeply inverted.
- The coronary perfusion is unstable, however, and the LAD can re-occlude at any time. If this happens, the first sign on the ECG is an apparent normalization of the T waves so called ***"pseudo-normalization"***. The T waves switch from biphasic/inverted to upright and prominent. This is a sign of hyper-acute STEMI and is usually accompanied by recurrence of chest pain, although the ECG changes can precede the symptoms.
- If the artery remains occluded, the patient now develops an evolving anterior STEMI.
- Alternatively, a "stuttering" pattern may develop, with intermittent reperfusion and re-occlusion. This would manifest as alternating ECGs demonstrating Wellen's and pseudo-normalization STEMI patterns.

This sequence of events is not limited to the anterior leads as similar changes may be seen in the inferior or lateral leads, e.g. with RCA or circumflex occlusion also the inciting event does not necessarily have to be thrombus formation, Wellen's syndrome may also occur in normal coronary arteries following an episode of vasospasm, as in a case of *cocaine-induced vasospasm*. However, it is safer to assume the worst (i.e. critical LAD stenosis) and work the patient up for an angiogram.**(25)**

Ischemic T-wave inversions are symmetric (the normal T-wave is asymmetric) and maybe, but rarely are, deeper than 10 mm. ECG leads with the opposite angles of observation (opposite to leads with T-wave inversions) usually display positive T-waves. Post-ischemic T-waves may be accompanied by negative U-waves, which further increases the likelihood of ischemia as the underlying cause. (DiMino, Ivanov, Burke, & Kowey, 2006)

The T-wave inversions following myocardial infarction usually resolve within days or weeks, but they may become chronic (defined as persisting >1 year). Normalization of T-wave inversion after infarction indicates some recovery in the infarct area. **(25).**

**Conclusion:** Acute myocardial ischemia is often associated with dynamic changes in ECG waveform and serial ECG acquisition can provide critical information, particularly if the ECG at initial presentation is non-diagnostic. Recording several standard ECGs with fixed electrode positions at 15 – 30 min intervals for the initial 1 – 2 h, or the use of continuous computer-assisted 12-lead ECG recording (if available) to detect dynamic ECG changes, is reasonable for patients with persistent or recurrent symptoms or an initial non-diagnostic ECG. ECG manifestations suggestive of acute myocardial ischemia: New ST-elevation at the J-point in two contiguous leads with the cut-point: ≥ 1 mm in all leads other than leads V2–V3 where the following cut-points apply: ≥ 2mm in men ≥ 40 years; ≥ 2.5 mm in men < 40 years, or ≥ 1.5 mm in women regardless of age. New horizontal or down-sloping ST-depression ≥ 0.5 mm in two contiguous leads and/or T inversion > 1 mm in two contiguous leads with prominent R wave or R/S ratio > 1.

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