Electrocardiographic Distinction of Culprit Artery in Patients With Acute Inferior ST Segment Elevation Myocardial Infarction

Ahmed Mohammed Aly Ahmed, Ahmed Abd Elfattah Al –Zayat, Magdy Mohammed Abdelsamei, and Mohamed Mohsen Mohamed

Cardiovascular Department, Faculty of Medicine, Zagazig University, Egypt.

Corresponding author: **Ahmed Mohammed Aly Ahmed**

Email: drabdrabo2017@gmail.com

Article History: Received: 26.06.2023 **Revised:**04.07.2023 **Accepted:** 22.07.2023

Abstract: ECG changes has been introduced in previous studies and showed good sensitivity and specificity in detection of culprit artery in acute inferior STEMI.

Keywords: ECG**,** STEMI, Culprit**.**

DOI: 10.48047/ecb/2023.12.10.1000

Introduction:

The ECG being a non-invasive and inexpensive tool ready available at bedside it would be most favorable if the initial ECG could reveal information that would help the clinician to tailor the most optimal treatment strategy for the individual patient. Maybe the ECG can help identify patients who will benefit greatly from reperfusion therapy versus patients with modest effect. Additionally, the ECG may identify patients who will have a better prognosis with pPCI versus patients who will obtain similar outcome with fibrinolysis. Accordingly, costs of pPCI could be saved and some patients protected from the stress of being sent to a distant hospital for an acute intervention **(1).**

The normal ECG

The standard 12-lead ECG records differences in electrical potential between defined sites on the body surface **(2).**

More specifically, it reflects the progression of the trans-membrane difference in action potential of the heart muscle cells over the cardiac cycle and the ECG thus describes the electrical events of cardiac depolarization and repolarization. The variations of potential are caused mainly by the movement of K+ , Na+ and Ca++ across the cell membrane **(3).**

This cycle of inward and outward movement of electrolytes is in part dependent on adenosine triphosphate. The signals recorded from the electrodes are then filtered to reduce disturbances, e.g. from respiration, muscle artifacts, and movement. The result is a graphical presentation, consisting of a series of complexes depicting the electrical activity in the heart viewed from twelve different sites**(4)**.

The different positive and negative deflections are noted with the letters PQRST .A wave of depolarization moving toward, or a wave of repolarization moving away from the positive electrode will generate a positive

wave on the ECG, and the magnitude of the wave depends on the mass of cardiac tissue undergoing depolarization or repolarization**(5).**

ECG waveforms

Waveform changes caused with ischemia reflect its presence, location, extent, severity and timing. Presence, location, and extent of ischemia are indicated by changes in the ST-segment, while severity is indicated by distortion of the QRS complex, and timing by the occurrence of tall T-waves versus abnormal Qwaves in leads with ST-segment changes **(6).**

In a normal ECG the ST-segment is isoelectric or nearly isoelectric, but in the presence of an injury current generated by the difference in gradients across the boundary between normal and transmurally ischemic myocardium, the ST-segment will move towards the involved myocardial region. Consequently, the direction of the ST-segment changes will depend on the orientation of the affected myocardial region in relation to each individual lead. In the conventional ECG, involvement of anterior or inferior regions of the myocardium will then be recognized as ST-segment elevation. In contrast, posterior-lateral involvement (caused by occlusion of the left circumflex artery) will produce ST-segment depression and thereby never meet STEMI criteria, which suggests that a diagnosis of ischemia/infarction is indicated when the ST-segment reaches a predetermined threshold value in 2 or more anatomically contiguous ECG leads **(7).**

Not only the extent of the ischemia, but also other variables such as the distance between the heart and chest wall and the width of the chest wall may influence the magnitude of ST-segment elevation. Additionally, ST-segment elevation may also be caused by other abnormalities e.g.: acute pericarditis, elevated potassium levels, left ventricular hypertrophy, right or left bundle branch block (RBBB, LBBB), Brugada syndrome, acute myocarditis, cardiac tumors, and the normal variant "benign early repolarization" **(8).**

ST-segment elevation is associated with reciprocal ST-segment depression in leads in which the positive electrode is directed in the opposite direction (≈180° away from). For example, STsegment elevation in lead III (positive electrode is pointed rightward and inferiorly) is associated with ST-segment depression in lead aVL (the positive electrode is pointed leftward and superiorly) and vice versa **(9).**

Changes in the QRS complex are seen with severe ischemia and infarction**.** When the ischemia is severe because of poorly protected myocardium, the QRS complex is directed towards the ischemic region, but then shifts away from the region revealing abnormal Q-waves as infarction develops. The presence of abnormal Q-waves is usually pathognomonic of a prior myocardial infarction (MI) **(10).**

They represent loss of electrical activity from necrotic cells and are therefore a sign of cell death. Tall T-waves, directed toward the epicardial surface in the center of the ischemic area, are seen in the early stages

of ischemia. The mechanism behind tall T-waves is not fully clarified but may be associated with an increase in intercellular potassium, which then shorten the action potential duration in the ischemic zone and causes early repolarization **(11).**

In contrast, late repolarization causes negative T-waves seen later in the ischemic process as an indication of successfully reperfusion of the myocardium **(12).**

Based on the mentioned changes in ECG waveforms, 4 phases of serial ECG changes during acute coronary occlusion have been described: Hyperacute, Acute, Sub-acute, and Chronic **(13).**

The time courses of the phases differ in each individual, and will be delayed during gradual coronary occlusion, but accelerated with prompt occlusion. In general the ischemic process is potentially reversible in the hyperacute phase, while progressive infarction occurs throughout the acute phase. Consequently, jeopardized myocardium can potentially be salvaged from undergoing infarction in the earliest phases of coronary occlusion, while no significant salvage will occur during the subsequent phases. The benefit of initiating reperfusion therapy during the later phases is thus prevention of infarct extension, and enhancement of the healing process **(14).**

Acute Myocardial Infarction

Both collaterals interconnecting epicardial arteries and preconditioning may develop in various degrees depending on prior ischemic episodes in the individual**(15).**

Accordingly, the degree of ST-segment elevation has been shown to be markedly reduced in hearts preconditioned with ischemia and/or in hearts with rich collateral arterial flow **(16).**

In addition marked prolongation of the QRS complex seen with severe ischemia are decreased in preconditioned hearts**(17).**

The mechanisms of metabolic preconditioning are not fully clarified, but may be a humoral and/or neural response to the ischemic state which then increases the myocardium's tolerance to ischemia at later episodes. Interestingly, preconditioning has been shown to produce pronounced action potential duration shortening **(18).**

Thereby tall Twaves may be a result of preconditioning, especially when present after a prolonged period of ischemia. Even in the presence of the mentioned cardiac protective factors jeopardized myocardial tissue will undergo infarction if adequate reperfusion treatment is not established in a timely manner**(19).**

The significance of Q-waves and pathologic R-wave progression

the presence of Q-waves on the presenting ECG is associated with a poor prognosis **(20).**

STEMI-patients presenting with Q-waves, the risk of death within 30 days was about twice as high compared to patients without Q-waves **(21).**

The presence of Q-waves is also associated with infarct size There is a possibility that Q-waves can regress after reperfusion and over time **(22)**.

patients with persistent Q-waves had an almost five-fold increased risk of heart failure

or death within one year compared to patients without Q-waves **(23).**

Pathologic R-wave progression signifies a decreased magnitude of the R-wave in the precordial leads, and has been associated with anterior myocardial infarctions. It has also been associated with heart failure, and patients with prior anterior AMI who have a pathologic R-wave progression have a lower left ventricular ejection fraction (LVEF) on echocardiography than patients with normal R-wave progression **(24).**

Identification of certain electrocardiographic confounders

ECG confounding factors included: LBBB, RBBB, left anterior/posterior fascicular block, left/right ventricular hypertrophy (LVH/RVH), ventricular rhythm, Wolff-Parkinson-White syndrome, Brugada syndrome, poor quality ECG e.g. unstable baseline and lead reversal, prior MI with persistent ST-segment elevation, benign early repolarisation, intraventricular conduction abnormalities, acute pericarditis, ischemic dilated cardiomypatia, left ventricular aneurism, and pacemaker rhythm **(25).**

Distinguish between STEMI, benign early repolarization, and acute pericarditis. Employment of such an algorithm may assist paramedics in differentiating among these three conditions. As a general rule it has been shown that in STEMI ST-segment elevation is localized to a portion of the ECG leads, and is often accompanied by ST-segment depression in other leads. In contrary, STsegment elevation in acute pericarditis is diffuse with minimal difference between minimal and maximal amplitudes. Diffuse ST segment elevation is also seen in benign early repolarization, but the amplitudes are higher in all leads except in lead V1 and III. Tall T-wave amplitudes in all leads but V1 and III are also significant for benign early repolarization when compared to acute pericarditis and early AMI **(25)**.

The presence of LBBB may hide the traditional changes of STEMI, and without a previous ECG it can be very difficult to determine if a patient with chest pain has STEMI **(26).**

Consequently, these patients are often treated insufficiently with both delayed diagnosis and treatment.

Sgarbossa have proposed 3 electrocardiographic criteria with high specificity but low sensitivity for diagnosing STEMI in patients with LBBB **(27).**

The criteria are:

1) STsegment elevation of ≥1 mm concordant with the QRS complex;
2) ST-segment depression of ≥1 mm in lead V1, V2 or V3; and
3) ST-segment elevation of ≥5 mm discordant with the QRS complex.

The confounding factors LVH, LBBB, and benign early repolarization accounted for the majority of the cases with ST-segment elevation but not STEMI **(28).**

Consequently, ST-segment elevation alone lacks the PPV necessary for reliable prehospital STEMI diagnosis. By inclusion of reciprocal ECG changes the PPV of prehospital AMI criteria increased to more than 90%, suggesting that ST-segment elevation criteria in combination with

reciprocal changes can identify patients most likely to benefit from early interventional strategies **(29)**.

Similarly found that consideration of both ST-segment elevation and depression significantly increased the sensitivity from 50% to 84% for detection of STEMI with only a slight decrease in specificity from 97% to 93% **(30).**

Myocardial salvage

Myocardial salvage is a term used for the amount of myocardium at risk that does not undergo infarction due to e.g. spontaneous reperfusion or initiation of reperfusion therapy **(31)**.

The amount of myocardium salvaged by initiation of reperfusion therapy may then be a measure for reperfusion success. A salvage index was first proposed by Clemmensen**(32).**

Subtraction the final estimated infarct size from the initially predicted area at risk for infarction. Myocardium at risk can be predicted by the Aldrich score, which is based on ST-segment elevation on the initial ECG**(33)**.

The score is a measure of the initially predicted myocardial infarct size as a percentage of the left ventricle if no reperfusion treatment is initiated. The original formula has been validated for anterior AMI but changed for inferior AMI **(34)**.

The final infarct size can be estimated on the predischarge ECG by use of the Selvester QRS score **(35)**.

This score contains 50 criteria considering Q- and R-wave durations, and relative Q-, R- and Swave amplitudes. It awards a maximum of 31 points, each representing approximately 3% infarction of the left ventricle. A high QRS score implies more extensive transmural myocardial damage. This scoring system was originally developed from anatomic studies of anterior and inferior infarcts, and has since been validated using single photon emission computed tomography (SPECT) **(36)** and delayed enhancement cardiac magnetic resonance imaging (MRI**) (37).**

In conclusion, the acute changes in the ECG waveforms can provide clinicians with essential information when evaluating patients presenting with chest pain **(38).**

The 12-lead ECG may therefore be very useful as decision support and help optimize treatment in this large group of patients **(39).**

ST-SEGMENT ELEVATION

The presence of ST-segment elevation is the foundation for initiating reperfusion therapy in patients presenting with symptoms suggesting ACS. Furthermore, several studies have demonstrated that the degree of ST-segment elevation on the admission ECG implies the extent of myocardium at risk of infarction, final infarct size, and prognosis**(40).**

Thus, the degree of ST-segment elevation remains pivotal for early risk stratification and initiation of treatment, including the preferred reperfusion strategy**(41).**

ST-segment elevation and outcome

Patients with most ST-segment elevation (≥ 20 mm) had significantly larger

infarcts and higher hospital mortality rates compared with those with less ST-segment elevation. the sum of absolute ST-segment deviation was one of the strongest predictors of mortality **(42).**

However the manitude of ST-segment elevation was no longer an independent predictor of mortality when ECG "Grades of ischemia" (GI) was included in the logistic regression analysis **(43)**.

GI consists of three grades:

1) Grade 1: tall upright T-waves;
2) Grade 2: ST-segment elevation in ≥2 adjacent leads without terminal QRS distortion, and
3) Grade 3: ST-segment elevation in ≥2 adjacent leads with terminal QRS distortion. This suggest that characterization of the quantitative changes occurring in the STsegment and terminal portion of the QRS complex during the early stages of STEMI represents another and potentially better method for risk stratification in STEMI patients soon after presentation **(6).** Only patients in the highest ST-segment elevation quartile showed a tendency towards better survival with pPCI, while the remaining patients had similar mortality rates regardless of treatment therapy **(44).**

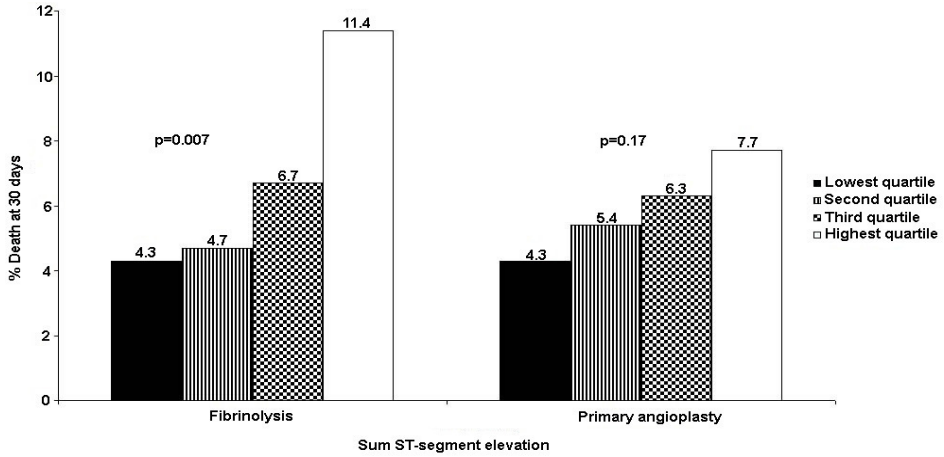

Figure 1: Mortality rates at 30-days for patients randomized to primary percutaneous coronary intervention or fibrinolysis. Patients are divided into quartiles of ST-segment elevation at baseline: First quartile: ≤6.5 mm; Second quartile: 7.0-9.5 mm; Third quartile: 10.0-14.5 mm; Fourth quartile: ≥15.0 mm **45**.

After fibrinolysis a residual stenotic lesion remains in 70-80% of patients **(46)** and 5-32% will reocclude **(47).**

In contrast, the presence of a residual stenotic lesion and the incidence of reocclussion after successful PCI are minimized with the routine use of stens and adjunctive pharmacological therapy **(48).**

In concordance with these findings it is not surprising that patients treated with

fibrinolysis had significant more reinfarctions than patients treated with pPCI regardless of ST-segment elevation. More reinfarctions were seen in patients with increasing ST-segment elevation in both treatment groups. This may partly be explained by anterior infarct location producing more ST-segment elevation compared to non-anterior infarcts, and that patients with anterior infarction have a higher reinfarction rate at late follow-up**(49)**.

Also reinfarction was associated with small reference diameter of the target vessel and small minimal luminal diameter, which are both characteristics of the left anterior descending artery **(50).**

The ECG for monitoring and optimal prehospital care

Once the diagnosis of STEMI is established on a prehospital ECG, ECG monitoring and/or serial 12-lead ECG recordings are necessary for further observation of the patient. Any ECG changes will play an important role in the decision support for minimizing morbidity and mortality during transport to the receiving hospital. Accordingly, all Danish ambulances are capable of establishing continuous 12-lead ECG monitoring in addition to measuring saturation and blood pressure **(11).**

ST-segment resolution analysis

ST-segment resolution starts occurring immediately after reestablishment of nutrient coronary blood flow to the ischemic area. STsegment resolution is not only considered a marker of epicardial blood flow but also microvascular perfusion, and persistent STsegment elevation, despite TIMI flow grade 3 in the coronaryartery, is associated with an increased risk of death **(51).**

Complete ST-segment resolution after reperfusion therapy is then a result of successful reperfusion at both the epicardial and microvascular level. Conversely, persistent ST-segment elevation appears to be indicative of either an occluded IRA or a patent IRA with failure of myocardial perfusion. The 2007 STEMI focused update uses the binary cut point of 50% ST-segment resolution as a marker for successful reperfusion **(52).** However, division into 3 groups is also an establish method for estimating reperfusion success **(53)**.

The 3 groups are classified as:

1) Complete resolution: >70% ST-segment elevation resolution,
2) Partial resolution: ≥30 to ≤70% ST-segment elevation resolution, and
3) No resolution: : <30% ST-segment elevation resolution **(54).**

ST-segment resolution and outcome

Regardless of treatment strategy all patients with complete ST-segment resolution tended to have the lowest mortality rate, but this difference only reached statistical significance in patients treated with fibrinolysis. Accordingly, STsegment resolution after thrombolysis has been proved to be a potent predictor of mortality, left ventricular function, final infarct size, and congestive heart failure**(55).**

In conclusion, ECG timing and the method for determining STsegment deviation are essential to obtain prognostic value when ST-segment measures are used

as a surrogate endpoint in large randomized trials **(56).**

It may be speculated whether the optimal time point for ST-segment measurements is individual. If this is the case continuous ST-segment monitoring would be optimal. This would also allow for early detection of recurrent ST-segment elevation caused by reocclusion as seen in acute stent thrombosis. relative ST-segment resolution in ECG recorded 90 minutes or later after PCI may not be a good surrogate endpoint for outcome in clinical trials. Interestingly, we found that patients with compete ST-segment resolution treated with fibrinolysis had the highest risk of reinfarction which emphasizes the need for considering PCI in all patients even after successful fibrinolysis **(57).**

Complete ST-segment resolution

Fibrinolyzed patients with complete ST-segment resolution seemed to have a much lower mortality rate compared to patients receiving pPCI both with and without ST-segment resolution **(58).**

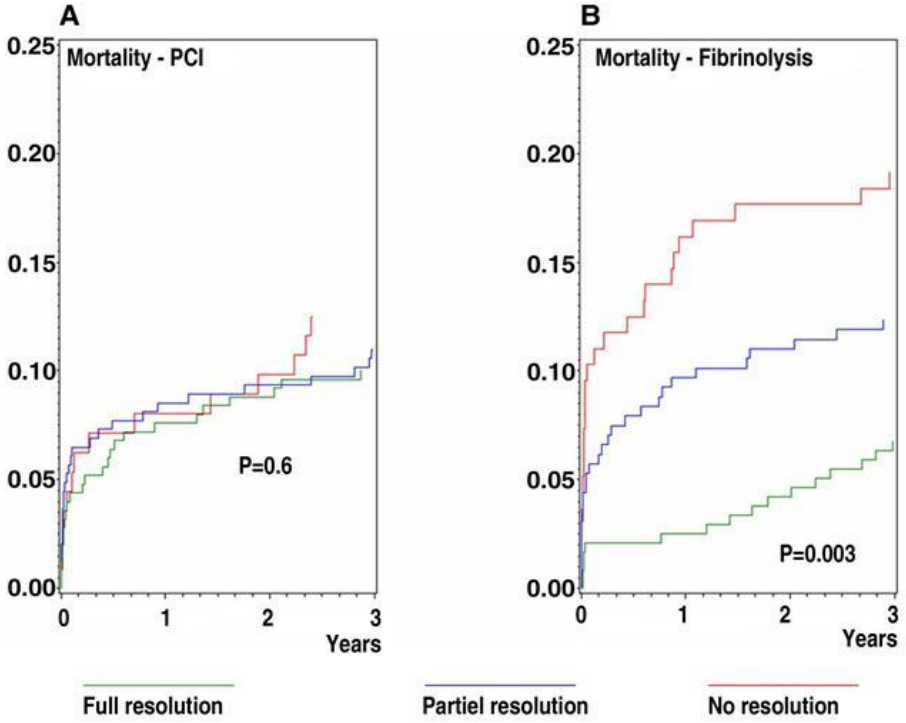

Figure 2: Kaplan-Meier curves illustrating mortality rates in primary percutaneous coronary intervention (pPCI) and fibrinolysis. The green line indicates full ST-segment resolution, the blue line indicates partial ST-segment resolution, and the red line indicates no ST-segmentresolution **59.**

However, this mortality benefit by fibrinolysis seemed to be counterbalanced by an increased risk of reinfarction in patients with complete ST-segment resolution **(60)**.

This relationship of complete ST-segment resolution with a high reinfarction rate may be explained by a difference in aetiology for patients with and without complete ST-segment resolution. Patients with complete ST-segment resolution may be experiencing a large acute occlusion which when removed by fibrinolysis is leaving a vulnerable plaque prone to reinfarct later. In contrast, patients with no

ST-segment resolution have no or only minor viable myocardium exposed for later reinfarction. Based on these finding all fibrinolyzed patients irrespective of the degree of ST-segment resolution seem to have a better prognosis with subsequent PCI. Accordingly, PCI in patients with failed fibrinolysis has been shown to improve outcome, including a reduction in reinfarction rate **(61).** this approach is supported by recent guidelines recommending transfer of all fibrinolyzed high risk patients to a PCIcapable facility, and consideration of transfer even if patients are not at high risk, especially if symptoms persist and failure to reperfusion is suspected.

ECG IN inferior STEMI

A cute myocardial infarction (AMI) of the inferoposterior wall is reflected on an electrocardiogram as ST elevation in the inferior leads accompanied by ST changes in other leads that may be a consequence of concomitant ischemia of other zones or a reciprocal image**(62).**

The prediction of the infarct-related artery on the basis of the admission electrocardiogram (ECG) in ST-segment elevation myocardial infarction (STEMI) is relevant and critical because it can anticipate specific mechanical complications, impending hemodynamic derangement, shock, and death **(63).**

However, after primary percutaneous coronary intervention (pPCI) became widely available and the preferred strategy for revascularization, many patients with STEMI are now being transferred to the catheterization laboratory before their admission ECG is examined to identify the possible culprit lesion. One unfortunate and common scenario is that wherein both the right coronary artery (RCA) and circumflex coronary artery (Cx) may show potentially culprit lesions in a patient brought to the catheterization laboratory because of inferior STEMI. Targeting the real culprit lesion is of utmost importance; however, sometimes, the angiographic appearance may be insufficient for guiding the interventionalist in this regard **(40).**

Accordingly, many algorithms have been developed to identify the infarct-related artery and the occlusion site, especially in cases of inferior STEMI **(64).**

These algorithms are based on the fact that in cases of AMI of the inferoposterior wall due to occlusion of the right coronary artery (RCA), the vector of injury is directed more downward than backward and more to the right than to the left ,in contrast, in cases of occlusion of the left circumflex (LCx) coronary artery, the vector of injury points downward, predominantly posteriorly, and more to the left than to the right **(65).**

The culprit artery in inferior wall acute myocardial infarction (AMI) is usually the right coronary artery (RCA), less often the left circumflex coronary artery (LCX), and rarely the left anterior descending artery (LAD).

Approximately 50% of patients with inferior wall AMI have significant bradycardia or hypotension, usually as a result of total occlusion of the proximal RCA **(2).**

ECG changes when culprit is RCA versus LCX

Early recognition of whether the culprit artery is the RCA or the LCX may facilitate management and, in some instances, may allow particular complications to be avoided.

Difficulty in differentiating between LCX and RCA occlusion in inferior wall AMI is a common clinical problem **(66).**

With regard to the electrocardiogram (ECG), the lateral limb leads are highly significant in inferior wall AMI, the aVL lead faces the high lateral segment of the left ventricular wall and is the only lead that is truly reciprocal to the inferior wall. The reciprocal image of the changes should be a decrease in the amplitude of the R wave and an increase in the negative wave (Q and S) amplitude. When there is no additional ischaemia of the lateral segment, as seen in RCA AMI, the aVL lead should depict a decrease in R wave amplitude and an increase in S wave amplitude**(67).**

The standard lead III is oriented to the right inferior segment, whereas lead II is oriented principally to the left inferior segment and also to the inferior region of the left lateral part of the superior wall of the ventricle. The axes of leads III and aVF are directed inferiorly, and therefore, STE is more prominently seen in these leads in RCA occlusions**(68).**

To understand the ECG changes in acute inferior wall MI, we need to understand the anatomy of coronary arteries, in most cases, the RCA terminates into a posterior descending artery and a few posterolateral branches to supply the inferior myocardium and the inferior part of the inferoposterior wall, respectively; whereas the LCX gives off a number of obtuse marginal branches to supply the posterior part of the inferoposterior as well as posterolateral wall. As a result, the vector of injury current is directed more to the right and inferior in RCA occlusions, and more to the left and posterior in LCX occlusions **(69).**

This minor difference in vector direction forms the basis of electrocardiographic differentiation between LCX and RCA occlusions.3-6 Lead III and aVF are pointing more to inferior and thereby ST elevation is found more prominent in these leads in RCA Occlusions, On the other hand, lead I and aVL are oriented more to lateral, and ST depression is less prominent or even ST elevation is present in these leads in LCX occlusions**(70).**

Precordial leads (V1-3) ST depression is common in inferior myocardial infraction, and explained either by reciprocal changes or various degrees of inferoposterior ischaemia **(71).**

The relation of vector direction between anterior and posterior wall is more strongly opposed than that between anterior and inferior wall.4 Therefore, LCX occlusions produce more prominent precordial ST depression than distal or proximal RCA occlusions **(70).**

The latter is known to be associated with precordial ST elevation due to

inadvertent detection of right ventricular infarction by the precordial leads **(11).**

In the context of inferior myocardial infarction, ST changes in precordial leads are vector summation of right ventricular as well as the inferoposterior ischaemia. Lead V1 (equivalent to lead V2R) is closest to the right ventricle and more sensitive in picking up right ventricular infarction **(72).**

SO Inferior wall AMI due to RCA occlusion frequently presents with ST elevation in leads II, III and aVF, and reciprocal ST depression in leads I and aVL.

The ECG changes in LCX occlusions are highly variable. Approximately 30 – 50% of patients present with ST elevation, usually in the inferior leads II, III and aVF. Others show ST depression in leads V1 to V4, or occasionally a tall R wave in lead V1. In up to 38% of patients there is no discernable ST elevation **(73).**

In the past few years, several investigators have proposed various criteria for identifying the infarct-related artery (RCA or LCX) in patients presenting with inferior wall AMI **(74).**

We found that ST segment elevation in lead III exceeding that in lead II, that is a ratio of elevation in lead III/elevation in lead II > 1 (criterion B), had a high specificity and sensitivity for RCA occlusion with sensitivity of 86%, a specificity of 94%, a positive predictive value of 95% and a negative predictive value of 56% for RCA involvement.

The ratio of ST elevation in lead III/elevation in lead II > 1 is an important predictor of RCA occlusion.

Higher ST segment elevation in lead III than in lead II was only seen in RCA occlusion, ST segment elevation in lead III greater than that in lead II was valuable in predicting RCA occlusion.

In addition S/R wave ratio > 0.33 plus ST depression > 1 mm in lead aVL (criterion A) was of value in predicting RCA occlusion.

Criterion a has sensitivity of 92%, a specificity of 94%, a positive predictive value of 97% and a negative predictive value of 65% for RCA involvement.

ST segment depression in lead I and aVL was only observed during RCA occlusion.

ST segment depression in lead aVL was a sensitive early ECG sign of RCA occlusion **(75).**

In reality, there are great variations in coronary anatomy amongst individuals, such as the relative size of vessels and the degree of dominance. Acute occlusion in a segment of a coronary artery may not produce the "expected" changes in a particular lead because of other anatomical opposing factors.

Most studies carried out so far have considered only one ECG criterion. By combining several leads, the error caused by variations in a single lead may be plausibly minimised.

The value of combining criterion A (an S/R wave ratio > 0.33 plus ST depression > 1 mm in lead aVL) and criterion B (ST segment elevation in lead III exceeding that in lead II) for predicting RCA involvement,

as well as their value when both negative for predicting LCX involvement.

The use of combined criteria gave a high specificity and sensitivity for both RCA and LCX occlusions **(76).**

Both criterion A and criterion B were positive in (83%) with RCA occlusion but no patients with LCX had this finding, giving a sensitivity of 83%, a specificity of 100%, a positive predictive value of 100% and a negative predictive value of 41% for RCA involvement. Both criteria were negative in (90%) with LCX occlusion but no patients with RCA had this finding, giving a sensitivity of 87% and a specificity of 100% for LCX involvement.

In conclusion, a ratio of ST elevation in lead III/elevation in lead II > 1, and an S/R wave ratio > 0.33 plus ST depression > 1 mm in lead aVL, are specific and sensitive markers of RCA occlusion.

If these changes are not detected in inferior wall AMI patients, LCX involvement is likely**(77).**

1) It is possible to predict the culprit coronary artery in inferior wall acute myocardial infarction by using the readily obtainable measures on the admission electrocardiography.
2) A higher ST segment elevation in lead III than in lead II, and deeper ST segment depression in AVL than in lead I are sensitive and specific markers for right coronary artery related acute inferior wall myocardial infarction.
3) The presence of ST segment depression in lead V2 and V3 is suggestive of left circumflex artery rather than right coronary artery related acute inferior wall myocardial infarction.
4) The amplitude of ST segment elevation and the proximity of culprit lesion along the infarct related right coronary artery were found to be closely related.
5) We recommend analysis of electrocardiography in patients with myocardial infarction especially for interventional cardiologist to predict the culprit coronary artery and its proximity, for more proximal occlusions are likely to cause greater myocardial damage and early intervention is recommended **(78).**

Using these ECG criteria shouldnot replace angiography, but it would provide earlier identification of the culprit artery **(79).**

References:

1. **Harjola, V. P., Parissis, J., Brunner-La Rocca, H. P., Čelutkienė, J., Chioncel, O., Collins, S. P., . . . Hill, L. (2018).** Comprehensive in-hospital monitoring in acute heart failure: applications for clinical practice and future directions for research. A statement from the Acute Heart Failure Committee of the Heart Failure Association (HFA) of the European Society of Cardiology (ESC). *European journal of heart failure, 20*(7), 1081-1099.
2. **Liang, H., Wu, L., Li, Y., Zeng, Y., Hu, Z., Li, X., . . . Zhou, X. (2018).** Electrocardiogram criteria of limb leads predicting right coronary artery as

culprit artery in inferior wall myocardial infarction: A meta-analysis. *Medicine, 97*(24).

3. **Xu, J., Dehaghani, A. R., Gao, F., & Wang, L. (2014).** Noninvasive transmural electrophysiological imaging based on minimization of total-variation functional. *IEEE transactions on medical imaging, 33*(9), 1860-1874.
4. **Van Leuteren, R., Hutten, G., de Waal, C., Dixon, P., van Kaam, A., & de Jongh, F. (2019).** Processing transcutaneous electromyography measurements of respiratory muscles, a review of analysis techniques. *Journal of electromyography and kinesiology, 48*, 176-186.
5. **Klabunde, R. E. (2017).** Cardiac electrophysiology: normal and ischemic ionic currents and the ECG. *Advances in physiology education, 41*(1), 29-37.
6. **Yamagishi, M., Tamaki, N., Akasaka, T., Ikeda, T., Ueshima, K., Uemura, S., . . . Kimura, T. (2021).** JCS 2018 guideline on diagnosis of chronic coronary heart diseases. *Circulation Journal, 85*(4), 402-572.
7. **Kumar, A., & Singh, M. (2016).** Ischemia detection using isoelectric energy function. *Computers in biology and medicine, 68*, 76-83.
8. **Members, A. T. F., Hamm, C. W., Bassand, J.-P., Agewall, S., Bax, J., Boersma, E., . . . Gielen, S. (2011).** ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: The Task Force for the management of acute coronary syndromes (ACS) in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). *European heart journal, 32*(23), 2999-3054.
9. **Apu, S. (2017).** *ECG for Medical Diagnosis*: JP Medical Ltd.
10. **Guidelines, E. C. f. P., Bax, J. J., Baumgartner, H., Ceconi, C., Dean, V., Deaton, C., . . . Hoes, A. (2012).** Third universal definition of myocardial infarction. *Journal of the American College of Cardiology, 60*(16), 1581-1598.
11. **Thygesen, K., Alpert, J. S., Jaffe, A. S., Chaitman, B. R., Bax, J. J., Morrow, D. A., . . . Infarction, E. G. o. b. o. t. J. E. S. o. C. A. C. o. C. A. H. A. W. H. F. T. F. f. t. U. D. o. M. (2018).** Fourth universal definition of myocardial infarction (2018). *Journal of the American College of Cardiology, 72*(18), 2231-2264.
12. **Fakhri, Y., Schoos, M. M., Clemmensen, P., & Sejersten, M. (2014).** Clinical use of the combined Sclarovsky Birnbaum Severity and Anderson Wilkins Acuteness scores from the pre-hospital ECG in ST-segment elevation myocardial infarction. *Journal of Electrocardiology, 47*(4), 566-570.
13. **Braschi, A., Frasheri, A., Lombardo, R. M., Abrignani, M. G., Lo Presti, R., Vinci, D., & Traina, M. (2021).** Association between Tpeak-Tend/QT and major adverse cardiovascular events

in patients with Takotsubo syndrome. *Acta Cardiologica*, *76*(7), 732-738.

14. **Campbell, B. C., De Silva, D. A., Macleod, M. R., Coutts, S. B., Schwamm, L. H., Davis, S. M., & Donnan, G. A. (2019).** Ischaemic stroke. *Nature Reviews Disease Primers, 5*(1), 1-22.
15. **Zhang, H., & Faber, J. E. (2015).** De-novo collateral formation following acute myocardial infarction: dependence on CCR2+ bone marrow cells. *Journal of molecular and cellular cardiology*, *87*, 4-16.
16. **Kleinbongard, P., & Heusch, G. (2022).** A fresh look at coronary microembolization. *Nature Reviews Cardiology, 19*(4), 265-280.
17. **Jennings, R. B., & Wagner, G. S. (2014).** Roles of collateral arterial flow and ischemic preconditioning in protection of acutely ischemic myocardium. *Journal of Electrocardiology, 47*(4), 491-499.
18. **Lejay, A., Fang, F., John, R., Van, J. A., Barr, M., Thaveau, F., . . . Scholey, J. W. (2016).** Ischemia reperfusion injury, ischemic conditioning and diabetes mellitus. *Journal of molecular and cellular cardiology*, *91*, 11-22.
19. **Ramkumar, R. (2015).** *A Study on Thrombolytic Effect of Streptokinase Infusion between Diabetic and Non-Diabetic Myocardial Infarction Patients with ECG as a Tool*. Madurai Medical College, Madurai.
20. **Koivula, K., Nikus, K., Viikilä, J., Lilleberg, J., Huhtala, H., Birnbaum, Y., & Eskola, M. (2019).** Comparison of the prognostic role of Q waves and inverted T waves in the presenting ECG of STEMI patients. *Annals of Noninvasive Electrocardiology*, *24*(1), e12585.
21. **Kholaif, N., Zheng, Y., Jagasia, P., Himmelmann, A., James, S. K., Steg, P. G., . . . Armstrong, P. W. (2015).** Baseline Q waves and time from symptom onset to ST-segment elevation myocardial infarction: insights from PLATO on the influence of sex. *The American Journal of Medicine*, *128*(8), 914. e911-914. e919.
22. **Tiller, C., Reindl, M., Holzknecht, M., Innerhofer, L., Wagner, M., Lechner, I., . . . Metzler, B. (2019).** Relationship between admission Q waves and microvascular injury in patients with ST-elevation myocardial infarction treated with primary percutaneous coronary intervention. *International Journal of Cardiology*, *297*, 1-7.
23. **Merghani, A., Malhotra, A., & Sharma, S. (2016).** The U-shaped relationship between exercise and cardiac morbidity. *Trends in cardiovascular medicine, 26*(3), 232-240.
24. **Schröder, L. C., Holkeri, A., Eranti, A., Haukilahti, M. A. E., Kerola, T., Kenttä, T. V., . . . Heliövaara, M. (2022).** Poor R-wave progression as a predictor of sudden cardiac death in the

general population and subjects with coronary artery disease. *Heart Rhythm*.

25. **Ripa, M. S. (2011).** *The ECG as decision support in STEMI*: Maria Sejersten Ripa.
26. **Birnbaum, Y., Fiol, M., Nikus, K., Niebla, J. G., Bacharova, L., Dubner, S., . . . Cygankiewicz, I. (2020).** A counterpoint paper: Comments on the electrocardiographic part of the 2018 Fourth Universal Definition of Myocardial Infarction. *Journal of Electrocardiology, 60*, 142-147.
27. **Segal, E., Ross, D., Proulx, M.-H., Xue, X., & Vacon, C. (2020).** Derivation and validation of the Montreal prehospital ST-elevation myocardial infarction activation rule. *Journal of Electrocardiology, 59*, 10-16.
28. **Guo, H., Zhang, W., Zhang, J., Ni, C., Cai, Z., Chen, S., & Huang, X. (2021).** Risk Factors for Nonischemic St-segment Elevation in Patients With Electrocardiographic Left Ventricular Hypertrophy.
29. **Lindow, T., Engblom, H., Pahlm, O., Carlsson, M., Lassen, A. T., Brabrand, M., . . . Ekelund, U. (2021).** Low diagnostic yield of ST elevation myocardial infarction amplitude criteria in chest pain patients at the emergency department. *Scandinavian Cardiovascular Journal, 55*(3), 145-152.
30. **Ayer, A., & Terkelsen, C. J. (2014).** Difficult ECGs in STEMI:: Lessons learned from serial sampling of pre-and in-hospital ECGs. *Journal of Electrocardiology, 47*(4), 448-458.
31. **Ibáñez, B., Heusch, G., Ovize, M., & Van de Werf, F. (2015).** Evolving therapies for myocardial ischemia/reperfusion injury. *Journal of the American College of Cardiology, 65*(14), 1454-1471.
32. **Sinning, C., Westermann, D., & Clemmensen, P. (2017).** Oxidative stress in ischemia and reperfusion: current concepts, novel ideas and future perspectives. *Biomarkers in Medicine, 11*(11), 11031-11040.
33. **Guo, Z., & Yang, X. (2019).** Does pre-angiography Total ST-segment resolution reliably predict spontaneous reperfusion of the infarct-related artery in patients with acute myocardial infarction? *BMC Cardiovascular Disorders, 19*(1), 1-8.
34. **Sejersten, M., Ripa, R. S., Maynard, C., Grande, P., Andersen, H. R., Wagner, G. S., & Clemmensen, P. (2007).** Timing of ischemic onset estimated from the electrocardiogram is better than historical timing for predicting outcome after reperfusion therapy for acute anterior myocardial infarction: a DANish trial in Acute Myocardial Infarction 2 (DANAMI-2) substudy. *American Heart Journal, 154*(1), 61. e61-61. e68.
35. **Loring, Z., Chelliah, S., Selvester, R. H., Wagner, G., & Strauss, D. G. (2011).** A detailed guide for quantification of myocardial scar with the Selvester QRS score in the presence

of electrocardiogram confounders. *Journal of electrocardiology, 44*(5), 544-554.

36. **Narous, M. (2016).** *Electrocardiographic Parameters in the Assessment of Myocardial Fibrosis and Left Ventricular Systolic Function.* Graduate Studies.
37. **Mello, R. P. d., Szarf, G., Schvartzman, P. R., Nakano, E. M., Espinosa, M. M., Szejnfeld, D., . . . De Paola, A. A. (2012).** Delayed enhancement cardiac magnetic resonance imaging can identify the risk for ventricular tachycardia in chronic Chagas' heart disease. *Arquivos brasileiros de cardiologia, 98*, 421-430.
38. **Harjola, V. P., Parissis, J., Bauersachs, J., Brunner-La Rocca, H. P., Bueno, H., Čelutkienė, J., . . . de Boer, R. A. (2020).** Acute coronary syndromes and acute heart failure: a diagnostic dilemma and high-risk combination. A statement from the Acute Heart Failure Committee of the Heart Failure Association of the European Society of Cardiology. *European journal of heart failure, 22*(8), 1298-1314.
39. **Al-Zaiti, S., Besomi, L., Bouzid, Z., Faramand, Z., Frisch, S., Martin-Gill, C., . . . Sejdić, E. (2020).** Machine learning-based prediction of acute coronary syndrome using only the pre-hospital 12-lead electrocardiogram. *Nature communications, 11*(1), 1-10.
40. **Vogel, B., Claessen, B. E., Arnold, S. V., Chan, D., Cohen, D. J., Giannitsis, E., . . . Kerneis, M. (2019).** ST-segment elevation myocardial infarction. *Nature reviews Disease primers, 5*(1), 1-20.
41. **Ibanez, B., James, S., Agewall, S., Antunes, M. J., Bucciarelli-Ducci, C., Bueno, H., . . . Halvorsen, S. (2018).** 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). *European heart journal, 39*(2), 119-177.
42. **Hashimoto, T., Ako, J., Nakao, K., Ozaki, Y., Kimura, K., Noguchi, T., . . . Nakama, Y. (2018).** Pre-procedural thrombolysis in myocardial infarction flow in patients with ST-segment elevation myocardial infarction a j-minuet substudy. *International Heart Journal, 59*(5), 920-925.
43. **Fabiszak, T., Kasprzak, M., Koziński, M., & Kubica, J. (2021).** Assessment of selected baseline and post-PCI electrocardiographic parameters as predictors of left ventricular systolic dysfunction after a first ST-segment elevation myocardial infarction. *Journal of Clinical Medicine, 10*(22), 5445.
44. **Shen, Y.-C., Krumholz, H., & Hsia, R. Y. (2021).** Association of cardiac care regionalization with access, treatment, and mortality among patients with ST-segment elevation myocardial infarction. *Circulation: Cardiovascular Quality and Outcomes, 14*(3), e007195.

45. **Engelberger, R. P., Fahrni, J., Willenberg, T., Baumann, F., Spirk, D., Diehm, N., . . . Kucher, N. (2014).** Fixed low-dose ultrasound-assisted catheter-directed thrombolysis followed by routine stenting of residual stenosis for acute ilio-femoral deep-vein thrombosis. *Thrombosis and haemostasis*, *111*(06), 1153-1160.
46. **Heisel, C., & Breusch, S. J. (2005).** The important role and choice of cement restrictor *The Well-Cemented Total Hip Arthroplasty* (pp. 150-154): Springer.
47. **Pradhan, A., Bhandari, M., Vishwakarma, P., & Sethi, R. (2021).** Deferred stenting for heavy thrombus burden during percutaneous coronary intervention for ST-elevation MI. *European Cardiology Review*, *16*.
48. **Zandecki, Ł., Sadowski, M., Janion, M., Kurzawski, J., Gierlotka, M., Poloński, L., & Gąsior, M. (2019).** Survival benefit from recent changes in management of men and women with ST-segment elevation myocardial infarction treated with percutaneous coronary interventions. *Cardiology Journal*, *26*(5), 459-468.
49. **Li, C., Ding, X., Wang, L., Li, K., Yang, X., Liu, L., & Xu, L. (2022).** Feasibility and Safety of Drug-Coated Balloon-Only Angioplasty for De Novo Ostial Lesions of the Left Anterior Descending Artery: Two-Center Retrospective Study. *Frontiers in cardiovascular medicine*, *9*.
50. **Mastrocola, L. E., Amorim, B. J., Vitola, J. V., Brandão, S. C. S., Grossman, G. B., Lima, R. d. S. L., . . . Araújo, J. R. N. d. (2020).** Update of the Brazilian guideline on nuclear cardiology-2020. *Arquivos Brasileiros de Cardiologia*, *114*, 325-429.
51. **Durante, A., & Camici, P. G. (2015).** Novel insights into an "old" phenomenon: the no reflow. *International journal of cardiology*, *187*, 273-280.
52. **Kunichika, H., Ben-Yehuda, O., Lafitte, S., Kunichika, N., Peters, B., & DeMaria, A. N. (2004).** Effects of glycoprotein IIb/IIIa inhibition on microvascular flow after coronary reperfusion: a quantitative myocardial contrast echocardiography study. *Journal of the American College of Cardiology*, *43*(2), 276-283.
53. **Ndrepepa, G., Alger, P., Kufner, S., Mehilli, J., Schömig, A., & Kastrati, A. (2012).** ST-segment resolution after primary percutaneous coronary intervention in patients with acute ST-segment elevation myocardial infarction. *Cardiology journal*, *19*(1), 61-69.
54. **Henry, T. (2016).** *Controversies in the Management of STEMI, An Issue of the Interventional Cardiology Clinics* (Vol. 5): Elsevier Health Sciences.
55. **Jabbar, A. (2021).** *Thyroxine in Acute Myocardial Infarction (ThyrAMI)*. Newcastle University.
56. **Nguyen, T. N., Gibson, C. M., Petrovski, B., Makam, P., Cong, N.**

D., & Khai, P. G. (2007). ST-Elevation Acute Myocardial Infarction. *THE EVIDENCE-BASED MEDICINE APPROACH*, 19.

57. **Pandit, B. N., Shrivastava, A., Nath, R. K., Kuber, D., Sinha, S. K., & Aggarwal, P. (2021)**. Impact of COVID-19 on thrombus burden and outcome in acute myocardial infarction. *Cureus, 13*(8).
58. **Mastoris, I., Giustino, G., Sartori, S., Baber, U., Mehran, R., Kini, A. S., . . . Dangas, G. D. (2016).** Efficacy and safety of routine thrombus aspiration in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention: An updated systematic review and meta-analysis of randomized controlled trials. *Catheterization and Cardiovascular Interventions, 87*(4), 650-660.
59. **Eitel, I., de Waha, S., Wöhrle, J., Fuernau, G., Lurz, P., Pauschinger, M., . . . Thiele, H. (2014).** Comprehensive prognosis assessment by CMR imaging after ST-segment elevation myocardial infarction. *Journal of the American College of Cardiology, 64*(12), 1217-1226.
60. **Mitchell[1], S. H., Hudson, K. B., & Brady, W. J. (2023).** Atrioventricular Conduction Block. *The Electrocardiogram in Emergency and Acute Care*, 54.
61. **Frampton, J., Devries, J. T., Welch, T. D., & Gersh, B. J. (2020).** Modern management of ST-segment elevation myocardial infarction. *Current problems in cardiology, 45*(3), 100393.
62. **Huang, X., Ramdhany, S. K., Zhang, Y., Yuan, Z., Mintz, G. S., & Guo, N. (2016).** New ST-segment algorithms to determine culprit artery location in acute inferior myocardial infarction. *The American Journal of Emergency Medicine, 34*(9), 1772-1778.
63. **Kamal, A. M., Soltan, G. M., & Ali, M. A. (2018).** Electrocardiographic prediction of culprit artery in acute ST-segment elevation myocardial infarction. *Menoufia Medical Journal, 31*(4), 1463.
64. **Shama, M. A., Hussaien, M. N., ELsaiedy, M. A., & Elsheikh, R. G. (2019).** Usefulness of the Electrocardiogram as a Tool to Localize Culprit Vessel Occlusion Site in Acute ST-Segment Elevation Myocardial Infarction. *Int. J. Curr. Microbiol. App. Sci, 8*(6), 1988-1995.
65. **Phalen, T., & Aehlert, B. J. (2018).** *The 12-lead ECG in acute coronary syndromes*: Elsevier Health Sciences.
66. **Roshdy, H. S., El-Dosouky, I. I., & Soliman, M. H. (2018).** High-risk inferior myocardial infarction: Can speckle tracking predict proximal right coronary lesions? *Clinical cardiology, 41*(1), 104-110.
67. **Alves, M. R. S., Momoli, J., Pilato, E. L., Marques, G. L., Schetz, M. R., & Pilato Sr, E. (2022).** Origin, Course, and Angulation of Coronary Artery Anomaly-A Case Report. *Cureus, 14*(9).

68. **Jim, M. H., Tsui, K. L., Yiu, K. H., Cheung, G. S., Siu, C. W., Ho, H. H., . . . Li, S. K. (2012).** Jeopardised inferior myocardium (JIM) score: an arithmetic electrocardiographic score to predict the infarct-related artery in inferior myocardial infarction. *Annals of the Academy of Medicine-Singapore, 41*(7), 300.
69. **Sattar, Y., & Chhabra, L. (2022).** Electrocardiogram *StatPearls [Internet]*: StatPearls Publishing.
70. **Meyers, H. P., Bracey, A., Lee, D., Lichtenheld, A., Li, W. J., Singer, D. D., . . . Meyers, K. E. (2021).** Ischemic ST-Segment Depression Maximal in V1–V4 (Versus V5–V6) of Any Amplitude Is Specific for Occlusion Myocardial Infarction (Versus Nonocclusive Ischemia). *Journal of the American Heart Association, 10*(23), e022866.
71. **Saad, A. A., Abbas, T., Khakwani, S. H. K., & Amin, M. (2018).** *Clinical Significance of ST Segment Depression in Lead aVR on ECG as a Predictor of Left Circumflex (LCx) Artery Involvement in Patients of Acute Inferior Wall Myocardial Infarction.* Paper presented at the Med. Forum.
72. **Tong-Wen, S., Wang, L.-X., & Zhang, Y.-Z. (2007).** The value of ECG lead aVR in the differential diagnosis of acute inferior wall myocardial infarction. *Internal Medicine, 46*(12), 795-799.
73. **Bayram, E., & Atalay, C. (2004).** Identification of the culprit artery involved in inferior wall acute myocardial infarction using electrocardiographic criteria. *Journal of international medical research, 32*(1), 39-44.
74. **Mohanty, A., & Saran, R. (2016).** Assessment of validity of the 'Culprit Score'for predicting the culprit lesion in patients with acute inferior wall myocardial infarction. *Indian Heart Journal, 68*(6), 776-779.
75. **Gregg, R. E., & Babaeizadeh, S. (2014).** Detection of culprit coronary lesion location in pre-hospital 12-lead ECG. *Journal of electrocardiology, 47*(6), 890-894.
76. **Kadoglou, N. P., Papadopoulos, C. H., Papadopoulos, K. G., Karagiannis, S., Karabinos, I., Loizos, S., . . . Klettas, D. (2022).** Updated knowledge and practical implementations of stress echocardiography in ischemic and non-ischemic cardiac diseases: An expert consensus of the Working Group of Echocardiography of the Hellenic Society of Cardiology. *Hellenic Journal of Cardiology, 64*, 30-57.
77. **Tzimas, G., Antiochos, P., Monney, P., Eeckhout, E., Meier, D., Fournier, S., . . . Schläpfer, J. (2019).** Atypical electrocardiographic presentations in need of primary percutaneous coronary intervention. *The American journal of cardiology, 124*(8), 1305-1314.