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ECG in Acute Myocardial Infarction in the Reperfusion Era

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1. Introduction

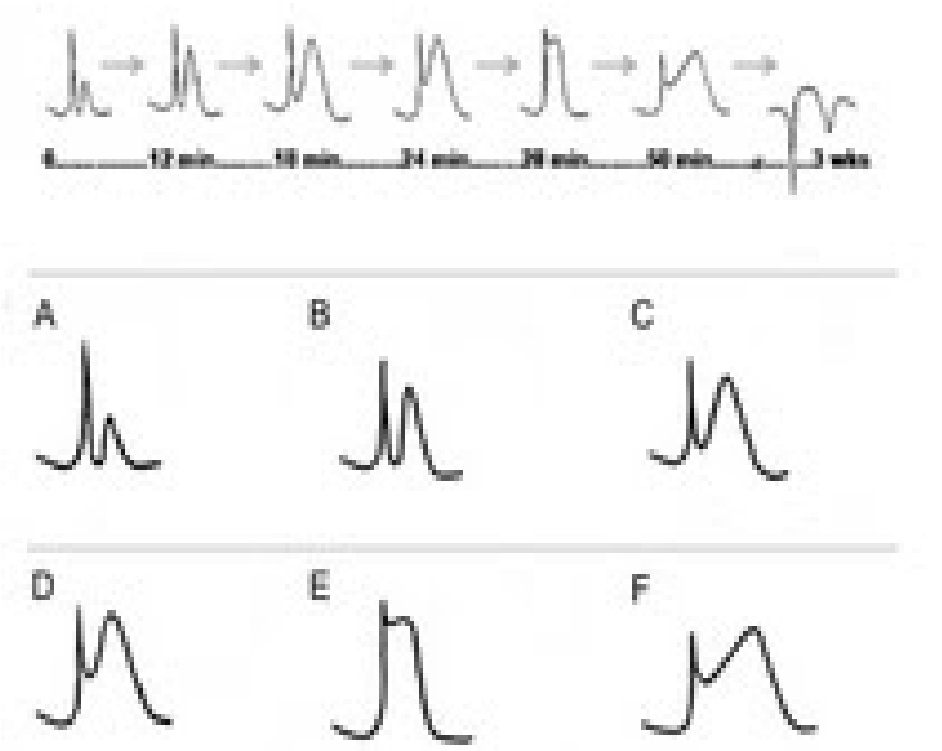
Acute myocardial infarction can be defined from a number of different perspectives related to clinical, electrocardiographic, biochemical and pathological characteristic. The electrocardiogram (ECG) is the most important diagnostic tool in the diagnosis of ST-segment elevation myocardial infarction (STEMI), and therefore it should be accomplished immediately at hospital admission. In fact, it represents an important step not only for STEMI diagnosis, but also and more importantly for the therapeutic plan. The present article pertains to electrocadiographic findings in patients affected by persistent STEMI. Moreover, it takes into account the clinical utility of ECG in the diagnosis and therapeutic decisions of evolving STEMI, as well as the prognostic implications of the ECG evolutions in the reperfusion era.

2. Evolving ECG changes occurring in the early phase of ST-elevation myocardial infarction

Typically, the ECG in the evolving STEMI shows five abnormalities, which develop in turn: hyperacute T waves, ST-segment elevation, abnormal Q waves, T-waves inversion, normalization of the ST-segment (Figure 1).

2.1 Hyperacute T waves

The T-waves represent the period of ventricular repolarization on the surface ECG. During the first minutes of coronary arterial occlusion (Dressler et al., 1947), the earliest ECG changes are represented by an increase in the amplitude of the T-wave, the so-called "Hyperacute T-waves" (Figure 1B,C). The morphologic characteristic of hyperacute T-wave are typical of ischemic event: they are asymmetric with a broad base and generally associated with reciprocal ST segment depression. In the evolving STEMI the hyperacute T-waves turn into giant R wave (Figure 1E). Hyperacute T-waves represent the electrocardiographic expression of ischemia before the beginning of necrosis; for this reason they are considered as the most significant phase during which the reperfusion therapy may achieve the greatest benefit in term of myocardial salvage (Lee et al., 1995). Prominent T-waves, however, are also associated with other diagnoses, including hyperkalemia, early repolarization end left ventricular hypertrophy (Somers et al., 2002). Thus in the differential diagnosis, the clinicians must consider additional features related to patient, including age, comorbidity and current medical status.



A Normal: Normal ST-segment and T-wave; **B Early, Hyper-acute T wave**: Development of Prominent T Wave; **C Hyper-acute T Wave**: Prominent T-wave with early ST-segment elevation; **D ST-segment elevation**: Progressive ST-segment elevation with persistent prominent T-wave; **E Giant R Wave**: ST - segment elevation continues with development of giant R-wave. **F ST-segment Elevation:** ST-segment elevation with oblique morphology.

Fig. 1. Evolving ECG changes occurring in the early phase of ST-elevation myocardial infarction

2.2 ST-segment elevation

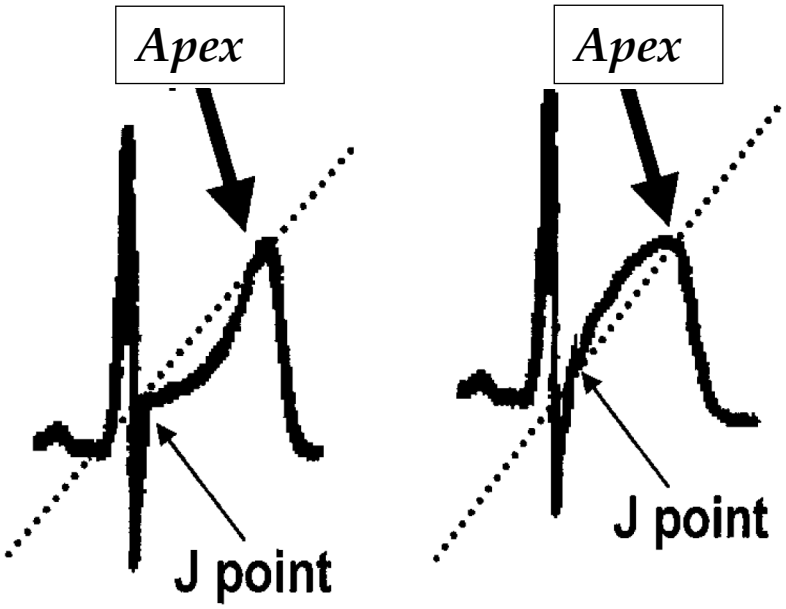
The ST-segment, defined as the segment beginning at the J point and ending at the apex of the T-wave, represents the electrocardiographic period between ventricular depolarization (QRS) and repolarization (T-wave) (Figure 1A). The ST-segment changes on the standard ECG that are associated with infarction are due to flow of current across the boundary between the ischemic and nonischemic zones. ST-segment elevation generally occurs with reciprocal ST depression in ECG leads in which the axis is opposite in direction from those with ST elevation (Figure 1D). The best criteria to classify abnormally elevated ST-segment are resumed in the Minnesota code 9-2 and are defined as ST-segment elevation of 1 mm in at least 1 peripheral lead, or 2 mm elevation in at least 1 precordial lead. These criteria have 94% of specificity for STEMI with a sensitivity of 56% in STEMI diagnosis (Menown et al., 2000). The threshold values results from recognition that some elevation of the junction of the QRS complex and the ST-segment (J-point) is a normal finding. Indeed, these are

dependent on gender, age, and ECG lead. Thus, the current thresholds recommended by the American Heart Association Electrocardiography and Arrhythmias, the Amrican College of Cardiology vary according to age, gender, and ECG lead (Table 1).

Men 40 years old of age and older	The threshold value for abnormal J-point elevation should be 0.2 mV (2 mm) in leads V_2 and V_3 and 0.1 mV (1 mm) in all other leads.
Men less than 40 years of age	The threshold value for abnormal J-point elevation in V_2 and V_3 should be 0.25 mV (2.5 mm).
Women of all ages	The threshold value for abnormal J-point elevation should be 0.15 mV (1.5 mm) in leads V_2 and V_3 and greater than 0.1 mV (1 mm) in all other leads.
Men and women of all ages	The threshold for abnormal J-point elevation V_3R and V_4R should be 0.05 mV (0.5 mm), except for males less than 30 years of age, for whom 0.1 mV (1 mm) is more appropriate.
Men and women of all ages	The threshold value for abnormal J-point elevation in V_7 through V_9 should be 0.05 mV (0.5 mm).
Men and women of all ages	The threshold value for abnormal J-point depression should be – 0.05 mV (-0.5 mm) in leads V_2 and V_3 and – 0.1 mV (- 1 mm) in all other leads.

Table 1. **Threshold values for ST-segment elevation according to age, gender, and ECG leads.** Adapted from AHA/ACCF/HRS (2009) Recommendations for standardization and interpretation of the electrocardiogram . *J Am Coll Cardiol*, Vol. 53, No. 11, pp. 1003-10011, ISSN 0735-1097/09/

However, ST-segment elevation can also attributed to other causes, different from acute myocardial infarction: a normal variant, frequently referred as *early repolarization,* commonly characterized by J-point elevation and rapidly upsloping or normal ST-segment; ventricular dyskinesis, often characterized by a small ST elevation; pericarditis, in which usually the ST elevation can be detected in more than one discrete region, as the inflammation involves a large portion of the epicardial surface, and reciprocal ST-depression is absent; elevated serum potassium; acute myocarditis; cardiac tumors or intra-thoracic mass. An additional ECG criteria in diagnosis of evolving STEMI is represented by the morphology of ST-segment elevation. In fact, two patterns of ST-segment morphology can be distinguish, according to the direction of the ST slope: a concave morphology and a convex morphology (Figure 2A,B). The concave morphology (Figure 2A) is hardly consistent with STEMI diagnosis, and rather related to other conditions, such as benign early repolarization, acute pericarditis. On the other hand, the convex morphology is usually associated with STEMI (Brady et al., 2001) (Figure 2B). The assessment of ST-segment elevation during STEMI is also useful to evaluate the extension of the myocardial at risk, and then the prognosis. In fact the number of leads with ST segment elevation and the sum of the total ST deviation have been related to the extension of area of myocardium at risk, defined as the extent of jeopardize ischemic myocardium, and consequently to the extent of necrotic area if reperfusion is not undertaken (Aldrich et al., 1988).



A Concave Morphology **B Convex Morphology**

A Concave Morphology: the concave morphology is characterized by downward ST slope; the ST slope remains below the virtual line drawn from the J-point to the apex of T-wave. **B Convex Morphology:** the convex morphology is characterized by upward ST-slope; the ST slope remains above the virtual line drawn from the J-point to the apex of T-wave

Fig. 2. Patterns of ST-segment elevation at ECG

Moreover , the analysis of the electrocardiographic leads revealing ST-segment elevation as well as of those showing ST depression, permits an almost accurate identification of the occluded coronary artery and also the proximal or distal location of the occlusion within that artery (Wagner et al., 2009). Anterior wall ischemia/infarction is invariably due to occlusion of the left anterior descending coronary artery and results in the spatial vector of the ST segment being directed to the left and laterally. This will be expressed as ST elevation in some or all of leads V1 through V6. The location of the occlusion within the left anterior descending coronary artery, that is, whether proximal or distal, is suggested by the chest leads in which the ST-segment elevation occurs and the presence of ST-segment elevation or depression in other leads. Occlusion of the proximal left anterior descending coronary artery above the first septal and first diagonal branches results in involvement of the basal portion of the left ventricle, as well as the anterior and lateral walls and the interventricular septum. This will result in the ST-segment spatial vector being directed superiorly and to the left and will be associated with ST-segment elevation in leads V1 through V4, I, aVL, and often aVR. It will also be associated with reciprocal ST-segment depression in the leads whose positive poles are positioned inferiorly, that is, leads II, III, aVF, and often V5 (Birnbaum et al., 1993). When the occlusion is located between the first septal and first diagonal branches, the basal interventricular septum will be spared, and the ST segment in lead V1 will not be elevated. In that situation, the ST-segment vector will be directed toward aVL, which will be elevated,

and away from the positive pole of lead III, which will show depression of the ST segment. When the occlusion is located more distally, that is, below both the first septal and first diagonal branches, the basal portion of the left ventricle will not be involved, and the ST-segment vector will be oriented more inferiorly. Thus, the ST segment will not be elevated in leads V1, aVR, or aVL, and the ST segment will not be depressed in leads II, III, or aVF. Indeed, because of the inferior orientation of the ST-segment vector, elevation of the ST segment in leads II, III, and aVF may occur. In addition, ST-segment elevation may be more prominent in leads V3 through V6 and less prominent in V2than in the more proximal occlusions (Engelen et al., 1999). Inferior wall infarction that results in ST-segment elevation in only leads II, III, and aVF may be the result of occlusion of either the right coronary artery or the left circumflex coronary artery, depending on which provides the posterior descending branch, that is, which is the dominant vessel. When the right coronary artery is occluded, the spatial vector of the ST segment will usually be directed more to the right than when the left circumflex is occluded. This will result in greater ST-segment elevation in lead III than in lead II and will often be associated with ST-segment depression in leads I and aVL, leads in which the positive poles are oriented to the left and superiorly. However, recently these criteria resulted less accurate in patients with electrocardiographic small inferior myocardial infarction (Verouden et al., 2009). Indeed, when the RCA is occluded in its proximal portion, ischemia/infarction of the right ventricle may occur, which causes the spatial vector of the ST-segment shift to be directed to the right and anteriorly, as well as inferiorly. This will result in ST-segment elevation in leads placed on the right anterior chest, in positions referred to as V3R and V4R, and often in lead V1 (Correale et al., 1999). Lead V4R is the most commonly used right-sided chest lead. It is of great value in diagnosing right ventricular involvement in the setting of an inferior wall infarction and in making the distinction between right coronary artery and left circumflex coronary artery occlusion and between proximal and distal right coronary artery occlusion. It is important to recognize that the ST elevation in the right-sided chest leads associated with right ventricular infarction persists for a much shorter period of time than the ST elevation connoting inferior wall infarction that occurs in the extremity leads. For this reason, leads V3R and V4R should be recorded as rapidly as possible after the onset of chest pain. ST-segment depression in leads V1, V2, and V3 that occurs in association with an inferior wall infarction may be caused by occlusion of either the right coronary or the left circumflex artery. This ECG pattern has been termed posterior or posterolateral ischemia since the early reports based on anatomic and pathological studies of ex vivo. However, recent in vivo imaging studies, including magnetic resonance imaging, have demonstrated that the region referred to as the posterior wall was lateral rather than posterior since the oblique position of the heart within the thorax: correlating the ECG patterns of healed myocardial infarctions to their anatomic location as determined by magnetic resonance imaging, the most frequent cause of abnormally tall and broad R waves in leads V1 and V2 was involvement of the lateral and not the posterior wall of the left ventricle (Bayes de Luna et al., 2006a). On these basis it has been proposed that the term posterior be replaced by the designation lateral (Cerqueira et al., 2002). Therefore, the terms posterior ischemia and posterior infarction be replaced by the terms lateral, inferolateral, or basal-lateral depending on the associated changes in II, III, aVF, V1, V5, and V6. Such terminology has been endorsed by the International Society for Holter and Noninvasive Electrocardiography (Bayes de Luna A et al., 2006b). It is not possible to determine whether the right coronary artery or left circumflex

vessel is occluded when changes of inferior wall ischemia/infarction are accompanied by depression of the ST-segment in leads V1, V2, and V3; however, the absence of such changes is more suggestive of right coronary than left circumflex artery occlusion. When the left circumflex is occluded, the spatial vector of the ST-segment in the frontal plane is more likely to be directed to the left. For this reason, the ST-segment may be elevated to a greater extent in lead II than in lead III and may be isoelectric or elevated in leads I and aVL (Bairey et al., 1987). Conversely, when a dominant right coronary artery is occluded proximally, left posterolateral and right ventricular wall involvement will be present, and the posteriorly directed ST-segment vector associated with this involvement may cancel the ST-segment elevation in lead V1 anticipated by right ventricular involvement and vice versa. The American College of Cardiology (ACC)/American Heart Association (AHA) guidelines for the management for patients with acute myocardial infarction (ACC/AHA, 2009) note the presence of electrocardiographic ST-segment elevation of greater than 0.1 mV in two anatomically contiguous leads; they suggest that such a finding is a Class I indication for urgent reperfusion therapy in the patient presumed to have STEMI. However, in few patients the presence of a left bundle branch block make the ECG less specific for the diagnosis of STEMI, because LBBB resembles STEMI changes. In this setting, the presence of suggestive symptoms and/or the certainty of the new-onset of conduction disorders may be helpful in diagnosis. Nevertheless, when these are not conclusive for diagnosis, the presence of some ECG criteria, pertaining the ST shift in relation to QRS vectors, may still indicate the diagnosis. To this regard, the ECG should be interpreted using the "rule of appropriate discordance", described by Sgarabossa and colleagues (Sgarbossa, 1996, 1998). They identified three independent electrocardiographic criteria suggesting for STEMI diagnosis in presence of LBBB: ST-segment elevation of at least 1mm that is concordant with the QRS complex; ST-segment depression of at least 1mm in leads V_2 and V_3; and ST-segment elevation of at least 5 mm that is discordant with the QRS complex. The Sgarbossa criteria provide a simple and practical diagnostic approach to identify STEMI in presence of LBBB, contributing to better address risk stratification and to optimize the risk-benefit ratio of reperfusion therapy in this challenging and high-risk population. In fact, the presence of LBBB in patients with acute myocardial infarction is usually related to large necrosis and consequently to high risk of complications and death. In fact, the new onset LBBB is related to the occlusion of the proximal left anterior descending artery and a large amount of jeopardized myocardium (Opolski et al., 1986). On the other hand, a pre-existing left bundle branch block is a powerful marker of depressed left ventricular systolic function, and any additional loss of myocardium is likely to result in large infarction and cardiogenic shock (Hamby et al., 1983)

2.3 Abnormal Q-wave

Q-wave are commonly present in normal ECG. Abnormal Q-wave suggesting myocardial necrosis have grater negative deflection and longer duration. Pathologic Q-wave typically appear within the first 9 hours of infarction, with a wide interval, ranging from few minutes to 24 hours (Perera, 2004; Goldberger, 1991). In particular in the evolution of non-reperfused myocardial infarction, Q-wave usually appear within 9 hours from coronary occlusion (Bär et al., 1996). However, it is not infrequent to observe Q-wave early after symptom onset. Abnormal Q-wave may be related to ischemia of the conduction system (Raitt at al., 1995; Smith & Whitwam, 2006). Thus, Q-wave should not be used exclusively as a marker of late

presentation of acute coronary occlusion, denying patients potentially beneficial reperfusion therapy. It is important to note that, in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial patients who did not develop Q-wave after fibrinolysis for STEMI had a lower mortality rate when compared to those who did develop Q wave at 30 days post infarction and 1 year post infarction (Bargelata et al., 1997). Thus, the absence of Q-wave after reperfusion therapy is a powerful marker of non-transmural necrosis and then of favorable prognosis.

2.4 T-wave inversion

In healthy patients, T-wave are normally upright in the left-sided leads (I, II, V3-V6). Within hours to days, an evolving STEMI will typically demonstrate T-wave inversion (Goldberger, 1991).The inverted T-wave appear generally in the same leads showing ST-segment elevation (Oliva et al., 1993). The morphology of inverted T-wave tends to be symmetric (Goldschlager & Goldman,1989). In the course of an evolving STEMI, T-wave inversion occurs when ischemia involves the epicardium. T-wave inversion is hypothesized by Mandel et al. to occur because of delayed depolarization in ischemic tissue (Mandel et al., 1968). In normal hearts, the epicardium is the first to depolarize, whereas the endocardium is the last. Delayed repolarization of the epicardium during ischemia reverses the direction of the ventricular repolarization current. With repolarization moving in the direction of endocardium to epicardium, the repolarization vector also reverses, causing a downward deflection of the T-wave (Smith & Whitwam, 2005). T-wave inversion occurs in approximately 3/4 of all patients with a completed myocardial necrosis (Goldschlager & Goldman,1989). Presence of T-wave inversion in precordial leads, of at least 2 mm, has a positive predictive value of 86% for left anterior descending artery stenosis (Haines et al., 1983). Indeed, a deepening T-wave soon after fibrinolysis may then determine successful reperfusion. However, normalization of T-waves may also predict a lower morbidity months after STEMI. One study by Tamura et al. found that patients with T-wave normalization within 6 months of infarction had higher left ventricular ejection fraction than those who did not, indicating that patients with normalization of inverted T waves had improved myocardial recovery (Tamura et al., 1999). The morphology of the T-wave inversion may help to differentiate between these other causes of T-wave inversion. Pacemaker T wave, in other words T wave inversion related to permanent ventricular pacemakers, tend to be broader than the narrower infarction T waves. A prolonged QTc distinguishes long QT syndrome. In mitral valve prolapse, T wave may be flattened or even inverted in inferior or lateral leads (Goldberger, 1991). In stroke, T waves tend to be very wide and the QT interval prolonged (Cropp & Manning, 1960).

2.5 Normalization of the ST segment

In not-reperfused STEMI, after a peak elevation approximately 1 hour after the onset of chest pain, the ST segment reaches a plateau at about 12 hours (Essen et al., 1979), and a complete resolution within 2 weeks in 95% of patients with inferior STEMI and 40% of patients with anterior STEMI (Mills et al., 1975). Even if the resolution of the ST-segment elevation may rarely occur from spontaneous reperfusion (Parikh & Shah, 1997), nowadays the normalization of the ST-segment can be observed in the majority of patients as result of successful reperfusion therapy. In fact, after successful fibrinolysis or mechanical reopening of infarct-related artery, abrupt changes occur in ECG as result of recovery in depolarization

currents across the myocites membrane. Thus a prompt decrease in ST-segment elevation is a powerful predictor of reperfusion (Richardson et al., 1988), whereas the persistence of ST-segment elevation represent a marker of unsuccessful reperfusion therapy and is an independent determinant of major adverse cardiac event (Claeys et al., 1999). Interestingly, a decrease in ST-segment elevation by at least 50% seems to be associated with 94% positive predictive value for complete reperfusion (Krucoff et al., 1993). Indeed, studies have also found that even after a complete and sustained patency of epicardial infarct-related artery obtained by pharmacological or mechanical recanalization, about one-third of patients still show a persistent ST- segment elevation, as result of unsuccessful reperfusion of the microvasculature (De Lemos & Brunwald, 2001). This condition is known as a no-reflow phenomenon, and has been related to a higher mortality and worse clinical outcome after myocardial infarction (Poli et al., 2002). Thus it is important to remark that normalization of the ST-segment indicates adequate perfusion throughout the myocardial microvasculature rather than epicardial coronary patency.

3. Choice of reperfusion strategy

Primary percutaneous coronary intervention and thrombolysis remain therapies of choice for patients presenting with evolving STEMI. However, clinical outcome after STEMI is mainly related to complete and sustained myocardial reperfusion, but strongly influenced by delay in achieving reperfusion. In fact, the extension of necrosis is time dependent, with a wave front developing from the subendocardium and extending transmurally to the epicardium over time. For every 30 minutes duration of ischemia, there is an 8-10% increase in mortality (Pinto et al., 2006). Reperfusion therapy, with dissolution or removal of the intracoronary thrombus, provides the best chance for mortality reduction. The Focused Update gives primary percutaneous coronary intervention (P-PCI) a Class IA recommendation for reperfusion, as long as it can be accomplished with a first medical contact to balloon inflation time of 90 minutes or less (Antman et al., 2008). Fibrinolysis, which is less effective than P-PCI in head-to-head trials, is given a Class IB rating as an alternative to P-PCI, as long as P-PCI can't be accomplished within 90 minutes. Although P-PCI is commonly more effective than thrombolytic therapy (TT) for the treatment of patients with STEMI, the mortality benefit of P-PCI over TT is risk and time-dependent (Antman et al., 2008; Keeley et al., 2003; Tarantini et al., 2005; Thune et al., 2005; Cannon et al., 2000; De Luca et al., 2003). As the time delay for performing P-PCI increases, the mortality benefit of P-PCI compared with fibrinolysis decreases. The P-PCI strategy may not reduce mortality when the delay is 60 min compared with immediate administration of a fibrin-specific lytic agent (Nallamothu & Bates, 2003). However, the value of 60 min is still controversial and should not be stated so categorically; other authors, for example, found that longer P-PCI-related delays do not negate the survival benefit of PPCI even when the delay is up to 3 h (Boersma et al., 2006; Stenestrand et al., 2006; Betriu & Masotti, 2005). Moreover, a recent evaluation of registry data has shown that the acceptable P-PCI-related delay depends upon the risk of the patient (Pinto et al., 2006). It has been explored the relationship between risk and P-PCI delay, adjusted for the delay at presentation, which leads to equivalent 30-day mortality between P-PCI and fibrin-specific thrombolytic therapy. Baseline mortality risk of STEMI patients is a major determinant of the acceptable time delay to choose the most appropriate therapy. Although a longer delay lowers the survival advantage of P-PCI, a longer P-PCI-related delay could be acceptable in high-risk STEMI patients (Tarantini et al.,

2010). Generally factors which preclude "waiting for PCI" include young age, anterior MI, and early (<3 hrs of pain) presentation. Factors which make delayed P-PCI the preferred strategy include contraindications to fibrinolysis, cardiogenic shock, advanced age, inferior MI, and delayed presentation (Antman et al., 2004). Whichever reperfusion strategy is chosen, it is important to maximize the effectiveness of that therapy by applying not only prompt, but also appropriate anti-platelet and anti-thrombin adjuncts. The recommendations for these therapies differ with the reperfusion method chosen. Appropriate protocol development demands maximization of the effectiveness of anti-platelet and anti-thrombin agents with each reperfusion choice.

4. Arrhythmias and conduction disturbances in the acute phase

Ventricular tachycardia (VT), Ventricular Fibrillation (VF) and complete atrio-ventricular (BAVC) block, may be the first manifestation of ischemia and requires immediate correction, since these arrhythmias may cause sudden cardiac death. VF and VT have been reported up to 20% of patients with STEMI (Henkel et al., 2006). Often arrhythmias are the manifestation of a serious underlying disorder, such as persisting ischemia, severe pump failure, or endogenous factors such as abnormal potassium levels, autonomic imbalances, hypoxia, and acid-base disturbances, that may require corrective measures. The need for arrhythmias treatment depends mainly upon the hemodynamic impact of the rhythm disorder.

4.1 Ventricular arrhythmias

VF occurring within 48 hours of the onset of STEMI has been related to higher in-hospital mortality, but not to long-term mortality. The major determinants of risk of sudden death are related more to the severity of the cardiac disease and less to frequency of classification of ventricular arrhythmias (Huikuri et al., 2001).

4.2 Ventricular ectopic rhythms

Ventricular ectopic beats are common during the initial phase of STEMI. Irrespective of their complexity (multiform QRS complex beats, short runs of ventricular beats or the R-on-T phenomenon) their value, as predictor of VF, is questionable.

4.3 Ventricular tachycardia and Ventricular fibrillation

Either not sustained VT (lasting <30s) not accelerated idioventricular rhythm (usually a harmless consequence of reperfusion with a ventricular rate<120beats), occurring in the setting of STEMI, serve as a reliably predictive marker of early VF. Sustained and/or haemodinamically compromising VT (occurring in 3%) requires suppressive therapy, and outlined in the guidelines for ventricular arrythmias (Zipes et al., 2006). Pulsless VT and VF should be managed according to the resuscitation guidelines.

4.4 Supraventricular arrhythmias

Atrial fibrillation (AF), which complicates 10-20% of STEMI is more prevalent in older patients and in those with severe LV damage and heart failure. AF is associated with increased in-hospital mortality (Fuster et al., 2006). In many cases this arrhythmia is well tolerated and no specific treatment is required. In other instances, the high ventricular response contributes to heart failure and prompt treatment is needed.

4.5 Sinus bradycardia and heart block

Sinus bradicardya: is common (9-25%) in the first hour, particularly in inferior infarction (Goldestein et al 2005). If associated with hemodynamic compromise it should be treated. *AV block:* Data from four large, randomized trials suggest that AV block occurs in almost 7% (Meine et al., 2005) and persistent LBBB in up to 5.3% of cases of STEMI (Newby et al., 1996). Patients with peri-infarction AV block have an higher in hospital mortality than those with preserved AV conduction (Meine et al., 2005). The increased mortality seems related to the extensive myocardial damage required to develop heart block rather than to heart block itself. AV block associated with inferior wall infarction is usually transient, whereas AV block related to anterior wall infarction is more often located below the AV node and associated with an unstable, wide QRS escape rhythm due to extensive myocardial necrosis. A new LBBB usually indicated extensive anterior infarction with high probability to develop complete AV block and pump failure. The preventive placement of a temporary pacing electrode may be warranted. Raccomandations for permanent cardiac pacing for persistent conduction disturbances (>14 days) due to STEMI are outlined in the ESC Guidelines for cardiac pacing.

5. ECG in pharmacological reperfusion - implications for adjunctive therapies

As a tool to identify epicardial reperfusion all methods of ST resolution, assessed by either continuous monitoring or static ECG recording, have the limitation that ST-segment changes integrate both epicardial and myocardial reperfusion. A resolution of ST-segment elevation of more than 70% of the initial value at 60 to 90 minutes after the initiation of therapy, is a powerful predictor of successful myocardial reperfusion and is therefore associated with enhanced recovery of LV function, reduced infarct size, and improved prognosis (de Lemos et al., 2000; Zeymer et al., 2001). Thus patients with complete ST-resolution at 90 minutes after fibrinolysis have a > 90% probability of a patent infarct-related artery associated with a successful reperfusion at the microvascular level. However, approximately 50% of patients with no ST-segment resolution after fibrinolysis still show a patent epicardial infarct artery. In fact in these patients the lack of ST resolution is caused by the failure of reperfusion at the level of microvasculature rather than at epicardial vessel. Thus, ST resolution represents a powerful predictor of infarct-related artery patency, but it is less accurate for predicting the persistence of epicardial vessel occlusion after fibrinolysis (Schröder et al., 2004). Therefore, in order to judge the need for adjunctive mechanical reopening of the infarct-related artery after failed fibrinolysis, by the so called "rescue angioplasty", it is important to integrate clinical and ECG data. According to the ACC/AHA guidelines, it is reasonable to monitor the pattern of ST-segment elevation, cardiac rhythm, and clinical symptoms during the 60 to 90 minutes after the initiation of fibrinolytic therapy. Non-invasive findings suggesting for a successful reperfusion include relief of symptoms, maintenance or restoration of hemodynamic and electrical stability, and a reduction of at least 50% in the initial ST-segment elevation. In this scenario, the presence of particular arrhythmias, such as not rapid ventricular tachycardia, idioventricular rhythm or not-sustained bradycardia, early after fibrinolytic administration, represents a highly specific marker of reperfusion. Otherwise, persistence of ischemic chest pain, absence of resolution of the qualifying ST-segment elevation, and hemodynamic or electrical instability are generally predictors of failed pharmacological reperfusion, needing rescue angioplasty.

6. ECG in mechanical reperfusion - implication for prognosis

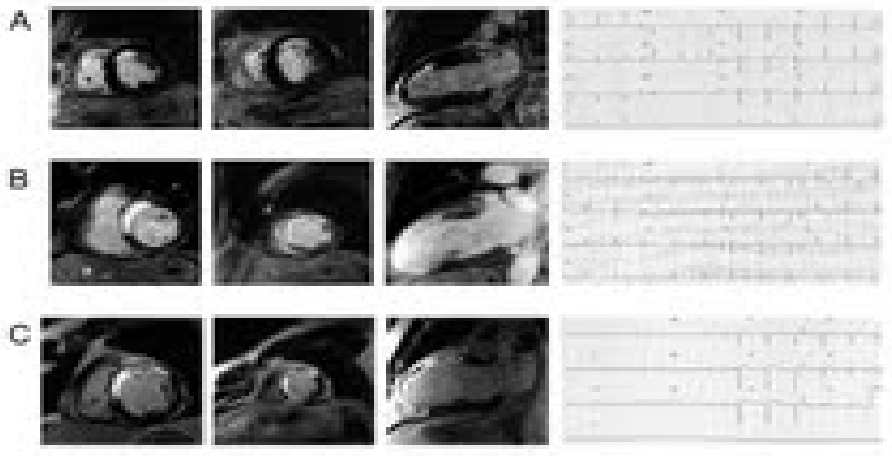
Many studies, evaluating the outcomes of primary angioplasty in STEMI, found that persistent ST-segment elevation after coronary flow restoration, is one of the independent determinant of adverse cardiac event (Schröder et al., 1994; de Lemos & Braunwald, 2001). In fact, patients with persistent ST-segment elevation, even after a successful restoration of normal antegrade coronary flow in the epicardial artery, show absent or inadequate flow at level of microvasculature (van't Hof et a., 1997). This phenomenon, known as a no-reflow phenomenon, has been described in animal and clinical studies, involving about one third of patients who underwent successful recanalization of the infarct-related artery. This condition, has been related to larger necrosis, adverse ventricular remodeling and higher morbidity/mortality at short and long-term follow-up. Otherwise, a resolution of ST-segment elevation by at least 50% is associated with a high positive predictive value for successful myocardial reperfusion. In this setting, the analysis of ST-segment evolution during and after coronary recanalization represents an useful tool to guide further pharmacological treatments, as well as more aggressive management of these patients. Different methods, cut-offs, and timing have been proposed to evaluate ST-segment resolution. In most studies, resolution of ST-segment elevation has been expressed as percentage of resolution of the sum of ST-segment elevation in all leads (van't Hof et a., 1997; Schröder et al., 1994; de Lemos JA & Braunwald, 2001; Zeymer et al., 2003). To this purpose, ST sum should take into account not only the ST shift in all leads showing ST elevation, but also the reciprocal ST deviation in leads showing ST depression. However, measuring ST resolution from all leads is time consuming and may be influenced by patient's position and by changes in position of lead electrodes. In order to simplify ST resolution assessment, other authors have proposed an alternative method based on measurement of ST resolution in only the single lead showing the maximum deviation before reperfusion: "the single lead ST resolution" (Schröder et al., 2001). In the single lead method, ST resolution is measured by comparing one ECG lead with the most prominent ST-segment shift at baseline and at a given time-point after reperfusion therapy, irrespective of the ECG lead measure at baseline. This method resulted as simple as accurate when compared to conventional model of sum ST resolution model. The optimal cut-off for defining reperfusion effectiveness and then mortality risk groups were assessed by statistical methods. Applying 2 cut-offs provides the most powerful stratification of high and low mortality risk group. To this purpose sum ST resolution is conventionally categorized as complete (≥ 70%), partial (<70% to 30%), and no (≤ 30%) ST-segment elevation resolution (Schroder et al., 1994). Although different time points have been reported across the studies evaluating the relationship between ST resolution and outcome, such as 30, 60, or even 90 minutes after reperfusion therapy, no significant differences between various time points were found, and the ideal time of measuring ST resolution remains unclear. However, these studies were mostly conducted in patients receiving fibrinolytic therapy, and may not directly applicable when assessing success of primary angioplasty. In fact, after fibrinolysis for STEMI the ECG for measuring ST resolution is usually taken 60 to 90 minutes after the onset of therapy, in order to detect by means of non-invasive tool the patency of epicardial vessel, rather than the reperfusion at microvascular level. In studies assessing the ST resolution on ECG, the timing of ECG after primary angioplasty was highly variable, ranging from 30 minutes after angioplasty to 60 minutes, or even several hours later. Strong evidences showed that ST resolution as evaluated 30

minutes after angioplasty correlated better with other markers of myocardial perfusion than ST resolution at 60 to 90 minutes. Indeed, recent evidences have shown that early complete ST recovery, as assessed immediately after last contrast injection in the catheterization laboratory, have a better preserved left ventricular ejection fraction and smaller infarct at magnetic resonance than patients showing ST resolution at 30 minutes or later (Haeck et al., 2011). These findings are not only consistent with the hypothesis that ST resolution implies effective microvascular and tissue reperfusion, but also relate the recovery of electrocardiografic changes to salvage of viable myocardium. Indeed, early assessment of ST recovery may represents the appropriate time to identify patients at higher risk of adverse events potentially benefit from additional novel therapies, ideally starting already at the catheterization laboratory.

7. ECG in stabilized myocardial infarction

The ECG in the stabilized phase of STEMI, after reperfusion therapy, represents a simply and universally applicable diagnostic tool to understand the prognosis and to guide further interventions. One method for determining the presence of pathological Q-waves related to myocardial infarction has been the Minnesota Code (Blackburn et a., 1960). This method was developed for diagnosis of infarction rather than the quantification of its size and correlates poorly with anatomically measured infarct size (Pahlm et al., 1998). An improved correlation of changes in the QRS complex with infarct size was the development of a QRS scoring system by Selvester et al. The Selvester QRS scoring system included 54 criteria from the QRS complexes in 10 of the standard leads, which totaled 32 points, each equivalent to approximately 3% of the left ventricular wall (Startt-Selvester et al., 1989). Recently, studies using cardiac magnetic resonance have show that Q-wave predict the location and size of myocardial infarction (Wu E et al., 2001.). Historically the presence of Q-wave on ECG after myocardial infarction has been used in clinical practice to stratify patients in Q-wave and non-Q-wave myocardial infarction, according to larger necrosis and worse outcome discovered in Q-wave infarctions (Stone PH et al., 1988). On these basis, for many years after the original report by Prinzmetal in animal model (Prinzmetal et al., 1954), the presence of Q-wave has been related to transmural infarction, whereas its absence was categorize as non-transmural infarction. Recently, studies based on cardiac magnetic resonance have clarified that, even if this distinction still appears useful to stratify the risk after myocardial infarction, the presence of Q-wave on surface ECG is determined by the total size of necrosis rather than transmural extent of underlying myocardial infarction (Moon et al., 2004). A relative small number of patients after myocardial infarction still show persistence of ST-segment elevation even days and month after the acute event. Historically, this late persistence of ST-segment elevation has been ascribed to left ventricular aneurysm or impending rupture of free wall or ventricular septum, identifying patients at very high risk for heart failure and death (Chon et al., 1967). However, this association is among the most controversial in electrocardiography, since previous studies, including echocardiography and angiography, clearly showed a more severe systolic dysfunction and wall motion abnormalities in patients with persistent STE, but failed to demonstrate a definite relationship between this electrocardiographic pattern and left ventricular aneurysm. Moreover, the explanation of the underlying mechanism of persistent STE and its pathological correlates are still unclear (Bar et al., 1984 & Lidsay J et al., 1984 ; Bhatnagar, 1994). Recently, using cardiac magnetic resonance, correlations between this ECG pattern

and type of myocardial damage have been reported. Particularly, the presence of persisting ST-elevation seems related to the presence of large microvascular damage in the context of transmural necrosis (Figure 3). These findings suggest that in this scenario late persistence of ST elevation indicates not only, as predictable, a greater extent of myocardial necrosis, but also, and more interestingly, the presence of severe microvascular damage as shown by cardiac magnetic resonance. Patients exhibiting persistent ST elevation showed more frequently left ventricular aneurysm, even though this difference did not achieve a statistical significance. Taking into account the findings of previous studies, these observations lead to the criticism about wall motion abnormalities as mechanism of electrocardiographic alterations. Recently, Li et al provided direct evidence in animals that opening of sarcolemmal KATP channels underlies ST elevation during ischemia (Li RA et al., 2000). It has also been demonstrated in a swine model that mechanical stimuli can induce marked ST elevation , by producing the stretching activation of KATP channel (Link et al., 1999). On these basis it has been hypothesized that outward bulging of myocardial necrotic wall, producing an abnormal stretch on the adjacent tissue, may alter cellular activity, generating injury currents at this level responsible for the ST elevation (Gussak et al., 2000). Thus patients exhibiting persistence of ST elevation had not only more severe myocardial



Panel A: ECG shows neither ST-segment elevation nor pathological Q-wave; the ce-MRI detects non-trasmural necrosis (middle and apical segments) of anterolateral wall, without either persistent microvascular obstruction or left ventricular aneurysm. **Panel B**: ECG shows pathological Q-wave in leads V4 to V6, with persistent ST elevation; the corresponding ce-MRI shows transmural necrosis of the septum, anterolateral wall (middle and apical segments), and apex, with evidence of persistent microvascular obstruction in the setting of necrotic core, without aneurysm. **Panel C :** ECG shows Q-wave in leads V1 through V6, DI, aVL, and STE in leads V1 through V6. The corresponding ce-MRI shows a large trasmural necrosis in the septum and anterolateral wall (middle and apical segments), and of the apical segments of inferior wall, with evidence of persistent microvascular obstruction in the necrotic core.

Fig. 3. Different patterns of myocardial structural abnormalities detected by contrast-enhanced magnetic resonance imaging (ce-MRI) and corresponding 12-leads electrocardiogram (ECG).

damage, but also more frequently coexistence of microvascular damage within it, that could account for diffuse alterations in myocardial skeletal favoring myocardial bulging and mechanical activation of KATP channels in the adjacent tissue. Finally, these findings may also explain the temporal discrepancy between developing of aneurysm and ECG alterations.

8. Conclusion

The ECG is the most important diagnostic tool in the diagnosis of evolving ST-segment elevation myocardial infarction, influencing therapeutic strategies and management. Moreover, ECG remains a simple but valuable method to estimate the risk of STEMI patients either before and after reperfusion therapy. Finally the value of ECG in the prognostic stratification after stabilized STEMI have still a role in current management of these patients.

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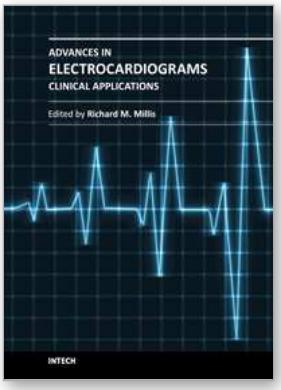

Advances in Electrocardiograms - Clinical Applications

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Electrocardiograms have become one of the most important, and widely used medical tools for diagnosing diseases such as cardiac arrhythmias, conduction disorders, electrolyte imbalances, hypertension, coronary artery disease and myocardial infarction. This book reviews recent advancements in electrocardiography. The four sections of this volume, Cardiac Arrhythmias, Myocardial Infarction, Autonomic Dysregulation and Cardiotoxicology, provide comprehensive reviews of advancements in the clinical applications of electrocardiograms. This book is replete with diagrams, recordings, flow diagrams and algorithms which demonstrate the possible future direction for applying electrocardiography to evaluating the development and progression of cardiac diseases. The chapters in this book describe a number of unique features of electrocardiograms in adult and pediatric patient populations with predilections for cardiac arrhythmias and other electrical abnormalities associated with hypertension, coronary artery disease, myocardial infarction, sleep apnea syndromes, pericarditides, cardiomyopathies and cardiotoxicities, as well as innovative interpretations of electrocardiograms during exercise testing and electrical pacing.

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