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Journal of Electrocardiology 38 (2005) 113–118

JOURNAL OF Electrocardiology

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Electrocardiogram of acute ST-elevation myocardial infarction: the significance of the various "scores"

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Abstract The Electrocardiogram has extensively been used for evaluation and triage of patients with acute chest pain. The clinician admitting a patient with ST elevation acute myocardial infarction should be able to estimate the size and location of the ischemic area at risk, how much of the ischemic myocardium has already undergone irreversible necrosis by the time of presentation, and the "severity of ischemia" (or what is the rate of progression of necrosis as long as ischemia continues). The electrocardiographic variables that are used to make these estimates are the initial portion of the QRS (Q and R waves), the terminal portion of the QRS (the S waves and the J-point), the ST segment, and the configuration of the T waves. This editorial discuss the ability to predict each of the "physiological" parameters using the above mentioned electrocardiographic variables.
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Keywords: Electrocardiogram; Infarct size; Ischemia; Collaterals; Q waves; ST segment; T waves; Prognosis

1. Introduction

The immediate and long-term outcomes after a myocardial infarction (MI) correlate with the amount of remaining viable and functioning myocardium, that is, the myocardial reserve. This is determined not only by the extent of acute necrosis, but also by the presence of old infarcts, the viable but "stunned" myocardium that recovers function after reperfusion, the amount of remote and chronically ischemic (or "hibernating") myocardium, and by the remote myocardium that is acutely ischemic either because collateral vessels to that area have occluded or because compensatory demands to that area for augmenting contractility for preservations of cardiac output induce ischemia. Whereas the myocardial reserve of patients with single-vessel disease and one MI is inversely correlated with the size of the ischemic area at risk, in those with multivessel disease and/or previous myocardial injury, a relatively small MI can cause significant decompensation. For example, occlusion of the distal posterior descending artery would by itself cause a small inferior MI, but the outcome could be much worse if the artery were also providing collaterals to myocardium perfused by an anterior descending artery that was also diseased.

Final infarct size is determined by the amount of tissue perfused by the occluded artery (the ischemic myocardium at risk), the duration of the occlusion, and by the "severity of ischemia" (or "rate of progression of necrosis"). The last of these is determined by the amount of anterograde flow through the culprit vessel, the perfusion by collaterals, and by metabolic factors such as ischemic preconditioning and the effect of various drugs.

Theoretically, the most accurate prognosis and best therapy could be determined by knowing the duration of ischemia, the severity of ischemia, the amount of ischemic myocardium at risk, and the amount of this area that had already infarcted. For example, one could predict that acute reperfusion would be most beneficial in patients with a large area at risk but a small amount of necrosis, as opposed to those with a small area at risk that was already dead. Reperfusion might improve remodeling and/or might prevent an arrhythmia in the latter group, but it would not preserve myocardial function.

Of course, time is also a major factor; however, in the clinical setting, most patients cannot recall the exact time when ischemia has started. In addition, because of the high

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0022-0736/$ – see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.jelectrocard.2005.01.003

Final infarct size= (the amount of already dead myocardium) + ("severity" X time X ischemic myocardium at risk)

Severity= severity of ischemia (the rate of progression of necrosis over time), determined by residual flow (antegrade or collaterals) and metabolic factors (ischemic preconditioning, medications, etc)

Time= time from presentation to reperfusion

Fig. 1. Prediction of final infarct size.

variability in the severity of ischemia (presence of collateral, incomplete occlusion of the culprit lesion, and/or presence of protective metabolic mechanisms) and the frequent occurrence of spontaneous intermittent reperfusion and reocclusion [1], the actual time that elapsed between the onset of ischemia to arrival may not correlate well with the progression of infarction or how much of the ischemic area at risk has already undergone irreversible damage. Theoretically, a more physiological term should be "time-severity integral"; however, in the clinical setting it cannot be measured.

In addition, opening the culprit epicardial artery will not always guaranty myocardial perfusion downstream [2,3], and any area that is reperfused will likely include both infarcted and stunned myocardium. Moreover, there is a lag period between the time that one makes the decision to proceed with reperfusion therapy and the time reperfusion actually occurs (usually 45-90 minutes with thrombolytic therapy; 1-2 hours with primary percutaneous intervention). Thus, final infarct size is actually the sum of the amount of myocardium already dead at presentation, plus the amount irreversibly damaged between presentation and reperfusion (Fig. 1).

Electrocardiogram (ECG) has helped physicians evaluate and triage patients with acute chest pain for a long time. Many studies have extended and refined its ability to estimate the size of the ischemic area at risk, the severity of ischemia, the amount of myocardium that has already infarcted, and the presence and quality of reperfusion. Arnold and Simoons [4] have integrated all these variables to define the "expected myocardial infarct size without reperfusion therapy." The ECG variables that change during ischemia and reperfusion are (1) the initial QRS (Q and R waves), (2) the terminal QRS (Sclarovsky-Birnbaum score) [5,6], (3) the ST segment, and (4) the T waves. Investigators have examined each variable alone, several variables using analysis of covariance or logistic regression, and have also incorporated changes in 2 or more variables to define clinically meaningful patterns, templates, or scores.

2. The initial portion of the QRS

Q waves are the traditional sign of myocardial necrosis, and thus they are thought to reflect the amount of infarcted myocardium (Fig. 1). One of the established methods of quantifying infarct size is the Selvester QRS score [7-11]. In the chronic phase of MI, it was found to correlate with left ventricular ejection fraction (LVEF) and infarct size in patients who did not receive reperfusion therapy; however, in patients who underwent reperfusion therapy the correlation is less strong [12]. As stated by Startt/Selvester et al [13], the presence of residual ST elevation may further cloud this QRS scoring system because only patients with isoelectric ST, TP, and PR segments were used to validate it. We have confirmed that, among patients with residual ST elevation on the predischarge ECG, the correlation between the QRS score and the infarct size (determined by predischarge technetium Tc 99m sestamibi scans) or LVEF is poor, whereas there is good correlation among patients with isoelectric ST segments [12].

In the study by Kalinauskiene et al [14] published in this issue of the *Journal of Electrocardiology*, the authors show that the QRS score decreases if the ECG evolves (T-wave inversion with or without ST resolution). Thus, in the early stages of an MI, the Q waves may not represent irreversible myocardial damage. This is consistent with an earlier study showing that 53% of the patients with an acute MI admitted within 1 hour of onset of symptoms had abnormal Q waves on presentation, before the start of reperfusion therapy [15]. Others have also suggested that Q waves appearing within 6 hours of symptom onset may not represent irreversible damage and therefore do not preclude myocardial salvage by reperfusion therapy [16]. This notion is supported by the finding that Q waves appearing early in the course of acute ischemia may be transient [17-19].

However, early Q waves may reflect both the amount of irreversible damage and a large ischemic zone, and thus portend a large infarct [15,16]. Such Q waves may result from a transient loss of electrophysiologic function due to intense ischemia ("myocardial concussion") in a region at risk that later becomes necrotic [17,20,21]. Some investigators have found that Q waves develop rapidly only after reperfusion [18,22-24], and it has been suggested that injury currents (ST deviation) can mask Q waves, as they sometimes appear only after the injury current has resolved [18]. These phenomena may result from the resolution of acute processes such as reperfusion injury, interstitial edema, and hemorrhage [22]. Therefore, the same amount of acute necrosis may or may not produce Q waves. If the amount of ischemic myocardium surrounding the necrotic zone is relatively small, Q waves

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