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Defining acute myocardial infarction by ST segment deviation

See page 275 for the article to which this Editorial refers

The advent of thrombolytic therapy ushered in a new era in the management — and diagnosis — of acute myocardial infarction. Although traditional diagnosis and classification has revolved around the presence or absence of Q waves, such a diagnosis is usually made 1–2 days after the patient presents to the hospital. Since thrombolytic therapy needs to be administered as quickly as possible, rapid diagnosis of acute myocardial infarction is needed. Although some early thrombolytic trials allowed patients to be enrolled on the basis of a clinical diagnosis of suspected acute myocardial infarction, regardless of what ECG changes were present, it was subsequently found that the only patients who benefited from thrombolysis were those with ST segment elevation or bundle branch block[1].

In contrast, despite the fact that patients with ST segment depression had a much higher risk of death than those with ST elevation, thrombolytic therapy made their mortality higher[1]. This lack of benefit of thrombolytic therapy was confirmed in the TIMI III trial of patients with unstable angina and non-ST elevation myocardial infarction[2]. The proposed mechanism for this difference in outcome is related to the pathophysiology of these two types of myocardial infarction: ST elevation is an excellent marker of acute coronary occlusion, in which reperfusion therapy is needed. In contrast, patients with non-ST elevation myocardial infarction and unstable angina have a thrombotic stenosis in the culprit artery, but the artery is usually patent[2]. Since thrombolysis is known to be prothrombotic, these forces can lead to progression of the thrombus to 100% occlusion, thereby creating a myocardial infarction, as was observed in TIMI IIIB[2]. In contrast, in ST elevation myocardial infarction, the artery is occluded at baseline, and thus the flow cannot worsen, it can only improve.

Accordingly, the spectrum of patients with acute coronary syndromes is no longer best divided into those with acute myocardial infarction vs unstable angina, but rather into those with ST elevation myocardial infarction vs non-ST elevation acute coronary syndromes. With this new nomenclature, acute therapy can be triaged: with thrombolytic therapy or 'primary' percutaneous coronary intervention for those with ST elevation myocardial infarction, whereas 'primary' therapy for those with non-ST elevation acute coronary syndromes is antithrombotic therapy (aspirin, heparin or low molecular weight heparin and/or glycoprotein IIb/IIIa inhibitors).

Given the importance of ST elevation in discriminating acute myocardial infarction, Menown and colleagues conducted a comprehensive study of the exact sensitivities and specificities of various degrees and definitions of ST elevation and/or ST depression for diagnosis of myocardial infarction[3]. They used univariate and multivariate models to predict development of myocardial infarction. They observed that models with either ≥ 1 mm ST elevation or ≥ 2 mm in precordial leads V_1–V_4 had a sensitivity for myocardial infarction of 55·8% and a specificity of 94%. When requiring at least two contiguous leads to demonstrate ST elevation (as used in most clinical trials), specificity for myocardial infarction rose and sensitivity fell slightly. Interestingly, the addition of variables of abnormal QRST features (e.g. T wave inversion) did not improve the model's performance — indicating the importance of careful examination of the ST segments on the ECG. For patients without ST elevation on the ECG, they observed that the presence of ST depression in ≥ 2 leads was very sensitive for the diagnosis of myocardial infarction (80%), but not specific. On the other hand, if ≥ 6 leads show ST depression, the specificity for myocardial infarction is 96·5%[3].

There are two other related areas where ST segments are (re)emerging as a clinical tool of great importance. The first is in evaluating the response to thrombolytic therapy (or primary percutaneous coronary intervention). Muller, Maroko and Braunwald identified in 1975 that early resolution of ST segment elevation is a useful means of assessing reperfusion[4]. Schroeder extended these observations using a 12 lead electrocardiogram at baseline and at 3 h post thrombolysis, and found that complete ($\geq 70\%$) resolution of ST segment elevation from baseline was an excellent marker of a good prognosis[5]. In other recent studies, resolution of ST segment elevation on a 12-lead electrocardiogram performed 90 min following thrombolysis has been found to be not only an excellent marker of coronary reperfusion but also of better myocardial tissue perfusion. Most importantly, it was observed in the Thrombolysis in Myocardial

Infarction (TIMI) 14 trial that addition of the IIb/IIIa inhibitor to reduced dose thrombolytic therapy significantly improved myocardial perfusion as assessed by ST segment resolution[6]. Thus, this simple tool (that all clinicians can use by simply obtaining a 12-lead ECG 90 min after the start of thrombolysis — and comparing it to the baseline ECG), allows determination of several key aspects of response to therapy and assessment of prognosis.

The second area of importance of the ST segments is in assessing prognosis. This is true for patients with ST elevation myocardial infarction (with either the baseline ECG, or as noted above, using the change from baseline to 90 or 180 min), and for those with non-ST elevation acute coronary syndromes[7]. In the latter case, presence of just $\geq 0{\cdot}5$ mm ST segment depression has been found to confer as bad prognostic significance as the more traditional ≥ 1 mm ST depression. It should be noted that for ST elevation myocardial infarction, the significance of 0·5 mm ST elevation has not been evaluated.

For me, the take home message is: Pay close attention to the ST segments! They are helpful in (1) the initial diagnosis of myocardial infarction, (2) evaluating the response to therapy, and (3) predicting future prognosis. If we carefully scrutinize the 12-lead electrocardiogram, it should assist us in targeting appropriate therapies for the broad group of patients with acute myocardial infarction and acute coronary syndromes.

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Dilated cardiomyopathy, are a few drinks allowed?

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The incidence of alcohol as the cause of dilated cardiomyopathy has been reported at between 20% and 45%[1,2]. These data, however, originate from observational studies and do not allow for discrimination between alcohol as the primary cause and alcohol as a contributing or aggravating factor for heart failure in dilated cardiomyopathy. The latter distinction may become important in view of the increasing awareness of inherited gene defects as important causes of dilated cardiomyopathy. Recently the frequency of familial dilated cardiomyopathy has been reported as up to 35% (instead of the formerly assumed 10%)[3]. Therefore, some cases of dilated cardiomyopathy identified as alcohol-induced may have been caused by inherited defects. In such cases excessive alcohol consumption may have promoted dilatation of the heart chambers rather than have caused it. Alcohol may also have contributed to the development of heart failure in patients with asymptomatic dilated cardiomyopathy. The larger than expected genetic origin of dilated cardiomyopathy might also explain the reported individual variations in the amounts of alcohol necessary to induce cardiomyopathy.

Data on the exact minimal amount and duration of alcohol consumption to cause cardiomyopathy are