Letters to the Editor


ST segment depression on the 12-lead ECG: high risk patients and high risk electrocardiography

The purpose of this correspondence is three-fold. First, the article from Belfast and Jordanstown on body surface potential mapping for the early detection of myocardial infarction (MI) when the presentation is with ST segment depression only[1], is a refreshing innovation. Commendations are due as it is in fact comparable in status to another landmark Irish accomplishment, that of Pantridge and Geddes[2], who, in 1967, utilized electrocardiography (ECG) to monitor community electrical resuscitation from ventricular fibrillation in the setting of a mobile CCU ambulance.

Second, I share with your readership the archetype of a high risk ECG peculiar to the high risk ‘ST segment depression’ population (Fig. 1). The common denominator of this ECG pattern is diffuse ischaemia of the subendocardial myocardium which equates electrocardiographically with ≥2 mm segment depression in one or more leads, an upright T wave reflecting a normal sequence of repolarization and a gradual decrease of R-wave amplitude. As Menown and colleagues[3] state and as graphed with the culprit left main coronary artery in Fig. 1, there may be diffuse narrowing present, but not necessarily coronary arterial obstruction in these patients. Electrocardiographers will recognize two ECG signatures of an evolving anterior wall infarction i.e. the negative ‘U’ wave in lead V5 and the early tall symmetric T waves, without ST segment elevation, in lead V6. A negative ‘U’ wave may be the earliest and only marker of a developing MI[3]. The configuration of TV5, without a major change in the terminal position of the QRS complex historically antedated ‘transmural’ Q injury[4]. However, recently we have been elegantly reminded by Phibbs[5] that 50% of subendocardial infarcts also produce Q waves. Finally, the left heart ‘strain’ pattern in Fig. 1 is a reminder of the ominous prognosis of this ECG pattern, rightfully triggering consideration of prompt intervention.

Third, it is envisaged by the undersigned, for possible future consideration, that the graphing of posterior ECG leads V7, V8, and V9 in conjunction with true posterior body surface mapping may well further augment the diagnostic sensitivity for non-ST elevation MI in this cardiac projection. Further, posterior leads validate the infarct-related-artery[6], differentiating left circumflex from right coronary artery insufficiency, thus establishing the guarded prognosis of the former[7] and ensuring definitive hospital allocation for reperfusion therapy. Finally, a ‘pure’ posterior (postero basal) pattern of injury is uncommon, usually being accompanied by an inferior or lateral infarct. As such, serial standard 12 lead ECGs accompanied by posterior leads V7, V8, and V9 provide a fuller description of the extent of myocardial injury and necrosis and, as such, expand the selection of candidates for reperfusion treatment[8].

J. MORPHE T
Department of Medicine,
McMaster University,
Hamilton, Ontario, Canada

References