ST-segment mapping in the diagnosis of acute myocardial infarction: a new role for an old method

ST-segment precordial mapping for the diagnosis of repolarization abnormalities in experimental and clinical acute myocardial infarction was introduced in the 1970s[1,2]. This was followed by use of the much more extensive ‘total thoracic’ ST wave mapping using standard exercise protocols to improve sensitivity and specificity when diagnosing ischaemic heart disease[3,4]. As known from theoretical studies, the strength of the surface maps is the direct association of the areas of positive and negative surface potentials with major wavefronts of the heart[5], and — since electrocardiographic potentials are recorded from large areas of the chest — body surface potential mapping enables the detection of significant physiological and diagnostic information not transmitted to the left precordial zones that are usually studied.

Whereas mapping directly explores the torso, the standard 12-lead electrocardiogram relies on remote-field bipolar and unipolar limb leads to compensate for the limited thoracic sampling. Based on these concepts, body surface potential mapping has been applied successfully in the study of various abnormal clinical conditions, such as chronic myocardial infarction, exercise stress testing and the diagnosis of accessory atrioventricular bypass tracts[6]. However, the real clinical relevance of body surface potential mapping has remained rather limited, since the breakthrough of invasive catheter diagnostics and other imaging methods, such as echocardiography and nuclear imaging, which seemed to overcome the diagnostic problems.

The results of large population, evidence based studies, such as GUSTO-IIb, show that the electrocardiogram is still the most accessible and widely used diagnostic tool in patients arriving at an emergency department with symptoms suggestive of acute myocardial ischaemia[7]. According to the GUSTO-IIb study, the 30-day incidence of death or myocardial reinfarction was 10·5% in a total of 12 142 patients with ST-segment depression. A similar number of adverse events occurred in 12·4% of the patient group with ST-segment elevation and depression. The results of GUSTO-IIb has also strengthened the value of two diagnostic tools available in the emergency department: the ECG and creatine kinase determinations, which enable bedside risk stratification and prediction of cardiac events.

It has been known for many years that the ST segment is not electrically inactive, and ST segment elevation measured on the electrocardiogram has been considered to be a characteristic of acute ischaemia, and thus the subject of numerous studies since the first report of Pardee[8] in 1920 on ECG alterations during acute ischaemia. When the whole thoracic surface is explored, the ST segments of normal subjects are quite uniform, with some minor individual variations, and are characterized by a potential maximum in the left mid-sternal region and a posterior minimum[6]. This pattern is stable and increases in intensity during repolarization without any significant spatial displacement. The ST segment is, in fact, the early part of repolarization, during which the potential distributions may depend on both the distribution of cellular action potential shapes and the sequence of ventricular activation. In normal
subjects, the dipolar nature of the ST segment is related to the longer repolarization time of the endocardium compared to the epicardium. As the epicardium recovers earlier, the extracellular potentials at the epicardium are more positive and this transmural gradient persists throughout the recovery process\(^9\). In ischaemia, when abnormal ST-T patterns are observed, two major mechanisms are considered to underlie ST-segment displacements: (1) a localized shortening of action potential duration, or (2) a localized decrease in resting membrane potential. In experimental studies by Kléber et al.\(^{10}\), changes in the resting membrane potential predominated during the first minute of acute ischaemia, and the changes in action potential morphology and duration occurred only after some minutes of coronary artery occlusion.

It is interesting to note that body surface potential ST segment maps, observed during abrupt coronary occlusion (i.e. PTCA) differ from those recorded during exercise testing, or spontaneous angina in patients with similar coronary artery disease. ST-segment shifts after balloon inflation, as reported by Shenasa et al.\(^9\) showed a significant difference in the patterns of ST shift 40 ms after end-QRS in the three major epicardial coronary arteries. The group average map patterns showed a striking mound of positivity focused on the anterior midchest during left anterior descending occlusion, but negativity was similarly focused on the same site during right coronary artery occlusion, with new positivity all around the inferior chest. Circumflex coronary artery occlusion also produced a new anterior sink of negativity, but with an accompanying positivity concentrated over the left back rather than over the entire inferior torso. Most, but not all, of the subjects with single vessel coronary artery disease followed the expected group pattern. Individual variations might be related to the level and direction of functional collaterals\(^{10}\).

ST-segment maps recorded immediately after exercise show a minimum in the left anterior chest region for all patients, irrespective of the diseased coronary arteries. The differences observed in the ST-segment body surface potential maps for angioplasty and exercise testing may be related to factors such as geometry and the nature of the ischaemia. For PTCA, a sudden, well-circumscribed and transmural ischaemia orientates the ST-segment vector according to the occluded territory. For exercise testing, the diffuse nature of coronary atherosclerosis and the presence of endothelial dysfunction, which is thought to play an important role in non-occlusive ischaemia, changes into a precordial ST-segment depression, generally most pronounced in the precordial V\(_2\) lead\(^{10}\).

Despite the well defined evidence of ST-segment shifts during exercise and even in the course of artificial occlusion of a main epicardial coronary artery (PTCA) in single vessel disease\(^9\), there are relatively few data on suspected myocardial infarction patients whose presenting ECGs show ST depression as the predominant feature. The results of GISSI\(^{11}\) and ISIS-2 trials\(^{12}\) show that subgroups of patients with ST depression have a high mortality of 16–19%, which is not reduced by thrombolytic therapy. As well emphasized in the study commented on in this editorial\(^{13}\), the value of the ECG in the correct diagnosis of the cardiac condition and in the risk stratification of these patients is of paramount importance. It is also evident from the results of Menown et al.\(^{13}\), that severe ST depression is highly specific for the subsequent diagnosis of acute myocardial infarction. Special isointegral and isopotential variables, easy to integrate into the everyday diagnostics of myocardial infarction, are of high sensitivity (88%) and good specificity (75%) thus indicating their real clinical value in the early bedside diagnosis of acute myocardial infarction.

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References

Towards a more precise definition of heart failure aetiology

See page 228 for the article to which this Editorial refers

Heart failure is a multifaceted syndrome with a multiple aetiology. In some cases the aetiology remains hypothetical or undefined. Obviously this is not without consequences since, even if advanced heart failure therapy is independent of the disease that brought on the decompensation, it is also true that, especially during the initial or intermediate phases of heart failure, knowledge of the cause can be crucial to plan the therapy.

The epidemiological context

The available epidemiological data confirm the variability, and thus the indeterminateness, of the aetiology of heart failure in various settings. According to the available data, coronary artery disease and hypertension (either singly or together) seem to account for the great majority of cases of heart failure within the developed world, whereas rheumatic heart disease, infections, and nutritional diseases are more common causes in the developing world. Thus heart failure is more common in young–middle-age groups in under-developed countries, and in the elderly population in developed countries. Among the elderly, the senescent modifications of the myocardium together with degenerative alterations of the cardiac valves, in particular of the aortic valve, are cofactors of increasing importance.

Trends of heart failure causes have changed in recent decades[1]. In the Framingham study, coronary artery disease was the primary attributable cause of heart failure in 22% of patients in the 1950s, 36% in the 1960s, 53% in the 1970s, and 67% in the 1980s. In contrast, valvular heart disease has markedly declined as a causal factor in the last few decades in the western world. The data on the prevalence rate of hypertension in heart failure are rather discordant. In the Framingham population it was noted that 91% of patients with heart failure had a history of hypertension[2]. In contrast, an overview of 31 studies indicated that hypertension was the primary aetiological factor in only 4% of heart failure patients[3]. In hospitalized populations, hypertension was found as the primary factor in 15–17% of patients[4,5]. More recently, hypertension also appeared to play a diminished causal role in the Framingham population; prevalence decreased by about 10% in men and 30% in women[1]. The decreasing prevalence of left ventricular hypertrophy associated with the effective treatment of hypertension paralleled the declining incidence of heart failure in treatment trials and probably underlies the declining causal role of hypertension in heart failure[6]. In contrast, the prevalence of diabetes is rising in heart failure patients. In the Framingham population, the rate of increase was 20% per decade[1]. A reduced glucose tolerance, a marker of insulin resistance, can also be considered as an indicator of a metabolic pattern favouring heart failure. Atherosclerosis accounts for up to 60% of all diabetes-related deaths. Impaired glucose tolerance and diabetes mellitus type 2 are often linked to a metabolic syndrome (insulin resistance, hypertension, central upper body obesity, dyslipidaemia with or without hyperglycaemia) at high risk of macrovascular disease, whereas in type 1 diabetes microvascular complications are a predominant feature linked to hyperglycaemia. Overall these findings suggest that coronary artery disease, hypertension and diabetes are the leading causes of heart failure.

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