Primary ST changes
Diagnostic aid in paced patients with acute myocardial infarction

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In 34 out of 36 patients with apical right ventricular endocardial pacing, primary ischaemic ST alterations were observed during the early stage of acute myocardial infarction. These ST changes, indicating acute injury, were detected in the paced beats in inferior and in anterior infarct. The primary ST changes were consistent only during the early stages of acute myocardial infarction and were not detected when the electrode tip was not in the apex of the right ventricle. It is suggested that the primary ST changes should be used to diagnose acute myocardial infarction in paced patients.

The diagnosis of acute myocardial infarction in patients with implanted pacemakers is difficult and has remained an electrocardiographic diagnostic problem. Previous attempts to resolve this difficulty were based on the following concepts:

(1) Analysis of escape beats or of the intrinsic rhythm by means of continuous monitoring or by various electrical interventions in pacemaker function (Center et al., 1971; Batten and Abrahamsen, 1973).

(2) Applying the same criteria used to diagnose myocardial infarction in the presence of complete left bundle-branch block (Sodi-Pallares et al., 1963) to the paced QRS complexes (Cardenas et al., 1972; Castellanos et al., 1973). However, it was found that the changes in the paced QRS were not consistent, especially in cases of inferior myocardial infarction (Barold et al., 1976).

Rothfeld et al. (1973) described ischaemic ST-T alterations in paced days in whom myocardial infarction was induced by ligation of coronary arteries. They noticed large ST vectors oriented toward the site of infarction, reflecting an injury pattern similar to those seen in non-paced patients with or without intraventricular conduction defects. These ST-T changes were present in all paced dogs with infarction.

The purpose of this study was to measure the incidence of the ischaemic ST alterations in paced patients during the acute phase of myocardial infarction, in order to assess their reliability as a method of diagnosing acute myocardial infarction in patients with permanent pacing.

Subjects and methods
Studies were made on 45 patients with acute myocardial infarction who were admitted to the coronary care unit. The diagnosis of acute myocardial infarction was confirmed in all patients by clinical, electrocardiographic, and enzymatic criteria. A temporary pacemaker was inserted in all these patients because of the development of 2nd and 3rd degree atrioventricular block or trifascicular block during the acute phase. The transvenous bipolar electrode was introduced into the right ventricle under fluoroscopic control through the antecubital or jugular veins. The external pacemaker used was Medronic model No. 5880. In 41 patients the tip of the electrode was placed in the apex of the right ventricle, in 2 patients it was found to be in a mid-ventricular position, and in another 2 patients in the outflow tract. The site of infarction was as follows: anterior wall—15 patients, inferior wall—16, inferior and true posterior—12, anterior and inferior—2 patients. In cases with complete atrioventricular block, the electrocardiographic diagnosis of acute myocardial infarction was always made before the development of this conduction disturbance. Thus, in all the patients the informa-
tion about the phase and the site of infarction was obtained by the analysis of conducted sinus beats. A routine 12 lead electrocardiogram was obtained twice daily during pacing, and during interruption of pacing, unless contraindicated.

Results

Ischaemic ST-T changes were considered to be diagnostic if ST elevation was at least 2 mm with a convex shape. Such transient changes during the acute phase of myocardial infarction were found in 34 patients out of the total group of 45 (Fig. 1 and 2).

During the study we realised that patients in phase III of the electrocardiographic evolution of acute myocardial infarction, or with the electrode tip not situated at the apex (Fig. 3) did not show these typical ischaemic ST-T alterations. Nine such patients (7 in phase III and 2 with the electrode tip not at the apex) were excluded from the total number of patients. In order to determine the phase of the electrocardiographic evolution of acute myocardial infarction, we have used the following accepted classification which differentiates four phases (Rushmer, 1955): Phase I is characterised by a prominent ST elevation and a positive or biphasic T wave and phase II by a pathological q wave, a less prominent ST elevation, and an inverted T wave. In phase III, the ST segment is isoelectric, the T wave is inverted and the q wave is more pronounced. The electrocardiographic evolution might end at this phase or can proceed to phase IV, in which the T wave returns to normal. The time interval from phase I to phase II ranges between a few minutes and several hours and from phase II to phase III between a few hours and several days. Thus, in 34 of the remaining 36 patients (94.4%) ischaemic ST-T changes were diagnostic of acute myocardial infarction during pacing. In only 2 patients were these changes not observed though the tip of the electrode was at the apex and the electrocardiographic phase was I or II, and should, therefore, be considered false negatives. The ischaemic ST-T alterations during pacing were always located in the same leads in which they appear on conducted sinus beats (Fig. 1 and 2).

Discussion

Sodi-Pallares et al. (1963) were the first to describe the patterns of myocardial infarction in the presence of complete right or left bundle-branch block. By applying the same criteria used by Sodi-Pallares et al. (1963) several electrocardiographic patterns were recently described as diagnostic of myocardial infarction in the presence of right ventricular pacing, based on the similarity between the morphology of complete left bundle-branch block and the paced QRS. Cardenas et al. (1972) described an initial R in aVR and V1 for inferior and true posterior myocardial infarction, qR in V5 and V6 for antero-septal myocardial infarction, and RS in V5 and V6 for lateral myocardial infarction. Castellanos et al. (1973) described a Sr-qR pattern in lead I, aVL, V5, V6 for anteroseptal myocardial infarction and pointed out that these changes in QRS might be overlooked or even absent when unipolar stimulation is used. The distortion that large unipolar spikes can produce might make it difficult to determine whether the finding is an artefact or a true reflection of an alteration in ventricular activation. Barold et al. (1976) did not agree with the specificity of Cardenas’s criteria for inferior myocardial infarction and suggested that a qR pattern in the inferior leads is a more specific though rare sign of inferior myocardial infarction during right ventricular pacing. In summary, the patterns described are inconsistent and their significance is controversial.

Rothfeld et al. (1973) showed recently that in all paced dogs with experimental acute myocardial infarction, large ST vectors oriented toward the site of infarction were observed even during pacemaker rhythm. In the present study we have shown that primary ischaemic ST elevation can be also seen in a high percentage of paced patients during the acute phase of infarction. During right ventricular endocardial pacing as in complete left bundle-branch block, it is the septal depolarisation that determines the direction of the AQRS and therefore also the direction of the AST and AT. Thus, the ST changes seen during right ventricular pacing and in complete left bundle-branch block are of the secondary type, reflecting a disturbed depolarisation. In the presence of acute myocardial infarction, it is the site of the injured area (epicardial or endocardial) that determines the direction of the ST vector and its shape. If both ST vectors (primary and secondary) coincide in direction, there will be exaggeration of the ST displacement. On the other hand, if these vectors are opposed, the final ST displacement will be minimal or even absent. For example, in paced patients with inferior acute myocardial infarction, both ST vectors are oriented toward the same direction, causing exaggeration of the convex ST elevation in leads II, III, and aVF (Fig. 1). The same findings can be observed in leads V1 to V4 in anteroseptal acute myocardial infarction (Fig. 2). However, in anterolateral acute myocardial infarction there is an opposition between
Before pacing

During pacing

Fig. 1 Acute inferior myocardial infarction. Ischaemic ST elevation is present in the same leads, before and during pacing.

Fig. 2 Acute anterior myocardial infarction. Ischaemic ST elevation is present in conducted sinus beats and in the paced beats. The diagnostic QRS criteria are also present (q wave in lead I, aVL, V4 to V6).

Fig. 3 Acute inferior infarction. The electrode tip was transferred from the right ventricular apex to the mid-ventricle and outflow tract regions. During apical pacing, primary ST elevation is detected in leads II and III, but at both other tip positions it is masked by the shift to the right of the AQRS.
the 2 vectors and, therefore, the typical diagnostic ST elevation can be detected only during the initial stage of electrocardiographic evolution, when the disturbed electrophysiological activity of the injured area is still predominant.

The AQRS, and therefore the AST, is also determined by the site of the electrical stimulation at the right ventricle. When the electrode tip is at the apex, the AQRS is usually −60° or more to the left. However, if the tip is located at mid-ventricular region or at the outflow tract, the AQRS shifts to the right. This causes a secondary displacement of the ST segment which is opposed to the direction of the primary ST vector, and, therefore, may mask the typical injury pattern (Fig. 3).

In summary, we found in this study that primary ST changes, suggesting acute injury, are present during the early stage of infarction in almost all patients undergoing right ventricular apical pacing. We suggest the use of these ST changes as a diagnostic aid for detection of the acute event in paced patients.

References


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