Precordial ST Segment Mapping
A Sensitive Technique for the Evaluation of Myocardial Injury?

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Myocardial ischemic injury was created acutely in pigs by a closed-chest technique utilizing an intracoronary balloon occluder for the interruption of flow in the left anterior descending coronary artery and ST-segment elevation was followed over a two-hour period using an 18 lead precordial map. In an experimental group of 10 animals, occlusion was carried out within the left anterior descending coronary artery 8.3 ± 0.5 cm distal to the origin of the main left coronary. Mean ST segment elevation (ST) showed a peak rise of 0.16 mV 10 minutes after occlusion. The balloon was moved proximally 1.6 ± 0.2 cm giving a significant secondary rise of 0.16 mV within 5 minutes, despite indications of a generally small area of additional myocardial involvement, as judged from anatomic distribution of additional vessels occluded as well as a lack of significant change in hemodynamic parameters. In a control group of 5 additional pigs, a single distal occlusion at 6.4 ± 0.9 cm from the origin of the main left coronary was produced by an identical technique. The ST rose to a peak of 0.20 mV at 15 minutes and was followed by a steady decline. Unlike the experimental group, no additional rise in ST was seen. The technique of precordial mapping thus appears to be a sensitive index of myocardial injury. In addition, it appears from this study that the magnitude of ST elevation is a direct reflection of the extent of myocardial injury.

Methods

Farm-bred pigs, weighing between 91 and 128 lb, were anesthetized with sodium pentobarbital, 15 mg per pound IV

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determination of cardiac output, either as the site of injection for cardio-green dye, or as a sampling site for mixed venous blood in a Fick determination. In the former, blood was withdrawn using standard techniques from the femoral artery and outputs calculated by a cardiac output computer (Lexington Instruments); in the latter, mixed venous and arterial bloods were withdrawn simultaneously with expired air collected in Douglas bags. Arterial blood gases were monitored to assess the adequacy of ventilation. Animals were rejected from the study if initial $P_{O_2}$ was less than 80 mm Hg. If evidence of hypoxia developed during a study, supplemental oxygen was given to maintain the level of oxygenation between 80 and 90 mm Hg.

**Occlusion Technique**

The right internal carotid artery was isolated and an 8F thin-walled Lehman catheter (U.S. Catheter & Instrument Company No. 5400), shortened to 50 cm, was introduced and guided fluoroscopically into the left anterior descending coronary artery (LAD). Dilute x-ray contrast material was injected to verify its position. A 3F embolectomy catheter (Edwards Laboratories) was then passed through the 8F catheter and positioned in the distal LAD, (left anterior descending coronary artery). The larger 8F catheter was then withdrawn into the aorta, the balloon on the embolectomy catheter inflated with radiographic contrast material, and its expansion visualized fluoroscopically. Balloon expansion was maintained thereafter by closing the catheter lumen.

**Protocol**

Fifteen animals were studied. Control measurements of the precordial map, arterial blood gases, cardiac output, as well as calculation of heart rate and blood pressure were made. Following the occlusion, these were repeated at 5, 10, 15, 30, and 45 minutes, with cardiac output repeated only at 10 and 45 minutes.

At the end of the first hour following occlusion, measurements including cardiac output were again taken. The animals were divided into two groups: Group A consisting of five animals, which received no additional intervention; and Group B consisting of ten animals, in which a small extension of the initial myocardial injury was created by moving the occluder to a more proximal position in the LAD. A sequence of measurements identical to those made during the first hour were again taken during this second hour.

All animals received heparin, 0.3 mg/kg IV at the time of introduction of the carotid catheter. To prevent serious ventricular arrhythmias, each animal was given lidocaine, 100 mg IV by bolus, prior to the positioning of the occluding catheter, followed by a constant infusion (2 to 4 mg/min) for the duration of the study.

At the end of the second hour, the animal was sacrificed, the chest opened via a left lateral thoracotomy, and the position of the inflated balloon-occluder marked on the myocardium. The balloon was then deflated and removed, the heart excised, and the appearance of the area subserved by the occluded vessel noted. At this point, the left coronary artery was opened longitudinally beginning at its ostium and continuing into the left anterior descending as far as possible. The ostia of all branches of the LAD were noted, as well as their distribution (septal vs diagonal) and size. The distance of the final proximal occlusion, as observed from the ostium of the main left coronary artery, was measured.

The anatomic location of the distal occlusion was determined by marking the position of the occluding balloon on the fluoroscope screen just prior to and immediately after its withdrawal to a proximal position. This distance was measured and the fluoroscopic magnification calculated at the end of each study. The distance between the two occluding positions could then be calculated, and by direct measurement from the known proximal occlusion site, the position of the initial, more distal site determined.

**RESULTS**

Data concerning the animal and type of occlusion is summarized in Table 1. For Group B the location of both occluders, determined as previously described, is indicated for each animal; the mean distance of the initial occlusion from the ostium of the main left coronary artery was 8.3 ± 0.5 cm, and the mean interocclusion or pullback distance 1.6 ± 0.2 cm. When the hearts were examined in situ at the termination of the study, the exocardial surface was seen to be normal in color and appearance. The location of the balloon-occluder was within the main LAD in all animals with the exception of no. 8, in which the occluder was located in a major diagonal branch. Subsequent examination of the coronary artery distribution and correlation with the calculated pullback distance indicated

<table>
<thead>
<tr>
<th>Vessel Location Distance Occluded</th>
<th>Group A (n = 5) Single Occlusion</th>
<th>Group B (n = 10) Occlusion with Extension</th>
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<tr>
<td>Vessels (cm) by Pullback</td>
<td>Mean (of 5) ± SEM (4.0-8.5)</td>
<td>Mean ± SEM (of 4.0-8.5)</td>
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<tr>
<td>Animal</td>
<td>Distal Location Pullback</td>
<td>Vessels Occluded</td>
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<td>Group A</td>
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<tr>
<td>LAD</td>
<td>6.4</td>
<td>± 0.9</td>
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<tr>
<td>Range</td>
<td>6.4 - 8.5</td>
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Table 1—Summary of Anatomic Data on Pigs Studied

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that, with the exception of animal no. 8, the initial distal occlusion occurred in the main LAD. The septal and diagonal branches located between the two occlusions are listed in Table 1. Except for animals nos. 3 and 10, these branches were small in size and number.

The precordial map for animal no. 9 is presented as a typical example of the information obtained (Fig 1). Following the initial occlusion at a distance of 9.5 cm from the left main coronary ostium, ST rises 0.20 mV to a peak at 15 minutes after occlusion, and then falls 0.08 mV by the end of the first hour. Following a pullback of 1.1 cm, ST rises 0.19 mV, peaks at 10 minutes, and falls 0.13 mV by the end of the hour following pullback.

For Group B, the mean distance of the initial occlusion from the ostium of the main left coronary artery was 8.3 ± 0.5 (Table 1). ST elevation for this entire group (Fig 2) rises 0.16 mV within 10 minutes to a peak and falls 0.07 mV by the end of the first hour; after the pullback, ST rises 0.16 mV to a peak at 5 minutes and falls 0.14 mV one hour after pullback. The rise from control is statistically significant (p < .01) for the entire initial occlusion. Following the pullback occlusion, the secondary rise in ST was significantly elevated (p < .01) for the next 30 minutes.

In Group A (Table 1), the mean distance of the occlusion from the ostium of the main left coronary artery was 6.4 cm. This resulted in a significant rise of ST (Fig 3) of 0.21 mV to the peak, occurring at 15 minutes. This rise is greater than that seen during the experimental group occlusions, and is consistent with the more proximal location of the occlusion when compared to the initial occlusion of the Group B. Following the peak, there is a downward trend of ST.

Hemodynamic parameters followed in the control and experimental groups are indicated in Figure 4. Tests for significance were carried out comparing each observation with the preceding control.

In order to allow comparisons between Groups A and B, those observations in Group A made during the second hour use the one-hour value for comparison. In the extension group (Group B), there appears to be an upward trend for mean heart rate and a downward trend for mean arterial pres-
sure. There are, however, no significant differences demonstrated for the heart rate rise; arterial pressure is significantly lower \((p<.05)\) only at 105 minutes \((92 \pm 11\) compared to \(114 \pm 9\) at 60 min), well after the peak of the ST rise and at a time when the ST is not significantly different from 60 minute value. Cardiac output gradually fell during the study, although no significant differences can be shown when compared to the control prior to the more proximal occlusion \((p>.05)\).

In Group A, no significance could be demonstrated with regard to heart rate or arterial pressure. Cardiac output was not significantly depressed \((p>.05)\) during the second hour after occlusion, when compared to the 60 minute value.

**DISCUSSION**

Previous studies using a mapping technique for the evaluation of ST elevation at multiple precordial recording sites during myocardial ischemic injury have correlated the sum of the ST elevation \((\Sigma ST)\) with the extent of myocardial damage.\(^1\(^4\)\) The studies have demonstrated that hypotension as well as agents known to increase cardiac work and consequently myocardial oxygen need, may increase \(\Sigma ST\); agents known to decrease cardiac work decrease \(\Sigma ST.\(^1\(^3\)\)\) A serial examination of 14 patients with acute myocardial infarction, using similar techniques, has shown a subsequent rise of \(\Sigma ST\) to correlate with biochemical evidence of extension of the infarction in 8.\(^4\) Nevertheless, the actual sensitivity of this new, noninvasive index of myocardial injury remains unproved. This study was therefore devised to demonstrate the sensitivity of the technique by examining the effect on the precordial map of a small controlled extension of myocardial infarction in the closed-chest animal.

Pigs were chosen as the experimental animal because of the similarity of their coronary supply to the left ventricle, the atrioventricular node, and the similarity of the size and type of collateral circulation to man.

Inflation of the balloon occluding catheter with radiographic contrast material makes both catheter and balloon easily seen fluoroscopically, permitting the degree of balloon expansion to be easily controlled, and providing accurate localization on the fluoroscopic screen of the two points of occlusion.
Since the course of the left anterior descending coronary in its mid- to distal portion produces little if any foreshortening when the supine animal is viewed fluoroscopically in the PA projection, accurate assessment of the distance between the two occlusion sites is possible. When combined with post-mortem examination, the anatomic site of occlusion and the caliber and distribution of the vessels serving the area undergoing infarct extension can be readily determined.

The actual distances on the LAD between distal and proximal occlusions varied little in the animals studied and, with the exception of animals nos. 3 and 10, include a few small septal and diagonal branches (Table 1). Nevertheless, a distinct and abrupt rise of ST could be found shortly after the creation of the more proximal occlusion. The new ST elevation was statistically significant, remaining so for the next 30 minutes (Fig 2). While this second rise resembled, in general, that seen after the initial occlusion, it reached peak sooner (16.5 ± 3.9 min) than the initial peak (10.0 ± 2.5 min, P<.01), perhaps reflecting accelerated injury to myocardium already rendered ischemic by the initial occlusion.

Heart rate and arterial pressure have been demonstrated to affect the level of ST elevation during epicardial mapping.6 In this study, these parameters have not varied significantly during the period of infarct extension. While there does appear to have been a gradual fall in mean arterial pressure during this extension period, this does not appear to have adversely influenced the ST since it was associated with the return of ST to baseline, to a level which, at 45 minutes, is not significantly different from that observed before the infarct extension. Cardiac output, while dropping significantly during the initial occlusion, likewise falls to fall significantly during the period of infarct extension. It therefore seems unlikely that changes in heart rate, arterial pressure, or cardiac output are responsible in themselves for the ST elevations appearing during the infarct extension period.

A correlation has been shown between depressed levels of myocardial creatine phosphokinase and the degree of ST segment elevation over the epicardial surface of those areas.8 Precordial mapping, however, has not been shown to define clearly a similar area of cellular injury. This report, along with previous epicardial mapping studies (see references), does support the inference that a larger area of cellular injury is reflected by a greater degree of ST segment elevation. In this study, eight of the ten animals in Group B had distal occlusions with small pullback distances. The LAD branches between the two occlusions in this subgroup were small in number and size. The mean highest ST elevation at any one observation period for this group following reocclusion, was 0.34 ± 0.04 mV. The remaining two animals, nos. 3 and 10, had extensions which from an anatomic analysis were larger in size: animal 3, having many branches arising from a short but proximal pullback distance; and animal 10, having a long pullback area, including a major septal branch. Peak ST elevations of 0.61 and 0.57 mV for animals nos. 3 and 10 respectively, were clearly outside the range of the other eight. There does, then, seem to be a correlation between the size of the area of extension and the magnitude of the ST elevation, paralleling observations made during other, epicardial mapping studies.

Quantification of the actual mass of myocardium involved in the extension, while of major interest, was not within the scope of this study. However, the lack of significant hemodynamic changes, the anatomically distal location of the occlusions, along with the small size and numbers of vessels further occluded in order to create the extension, all indicate that the infarct extension does indeed involve a small mass of myocardium.

CONCLUSION

That this small area of additional infarction results in a major elevation of the mean ST segment, statistically different from a control group of animals having no extension, is indicative of the sensitive nature of this technique. While it examines the effect of two episodes of myocardial injury closely related in time, it is in basic agreement with the clinical study of P. R. Reid and associates,4 which indicated that precordial mapping in humans sustaining an acute myocardial infarction was sufficiently sensitive to detect an extension of the original infarction which did not affect immediate prognosis. It is, therefore, quite possible that precordial mapping can detect minimal myocardial ischemic injury of interest for statistical and research purposes, but which is below the threshold of clinical usefulness. Additional studies correlating the magnitude of the precordial ST segment elevations with the actual mass of injured myocardium would provide important clinical and investigative information.

ACKNOWLEDGMENT: We acknowledge the technical assistance of Carl Bruno, Wade Cooke, and Thomas DeVona, as well as the skilled secretarial help of Carol White and Kathleen Mahoney.

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