Changes in Intramyocardial ST Segment Voltage and Gas Tensions with Regional Myocardial Ischemia in the Dog

By Shukri F. Khuri, John T. Flaherty, John B. O’Riordan, Bertram Pitt, Robert K. Brawley, James S. Donahoo, and Vincent L. Gott

ABSTRACT

This study was designed to evaluate the sensitivity of changes in myocardial carbon dioxide and oxygen tensions as indicators of regional myocardial ischemia and also to determine to what extent these changes can be related to changes in intramyocardial ST segment voltage. Changes in ST segment voltage recorded in unipolar epicardial electrodes proved to be a less-sensitive indicator of underlying myocardial ischemia than were changes in ST segment voltage recorded in unipolar intramyocardial electrodes. In 9 dogs, regional ischemia was produced by placing a variable constrictor on the left circumflex coronary artery; circumflex flow was monitored. Myocardial carbon dioxide and oxygen tensions were measured using a mass spectrometer. Unipolar electrograms were recorded using a multicontact plunge electrode. With progressive degrees of proximal stenosis, ranging from a critical stenosis, which is associated with a decrease in mean flow of less than 15%, to a severe stenosis associated with an 80% decrease, ST voltage increased 21 mv and carbon dioxide tension increased 84 mm Hg, but oxygen tension decreased only 7 mm Hg. The study suggests that increases in intramyocardial ST segment voltage, an index of myocardial ischemia, are associated with parallel increases in myocardial carbon dioxide tension, each providing a more sensitive quantitative correlate of regional myocardial ischemia than do decreases in oxygen tension. The local accumulation of carbon dioxide may be an important pathophysiological mechanism in myocardial ischemia.

Recent studies by Maroko et al. (1, 2) have utilized epicardial and precordial ST segment changes to define the extent of myocardial ischemic injury and to assess the efficacy of various therapeutic interventions for reducing infarct size. However, the metabolic abnormalities responsible for the genesis of reversible ST segment changes seen in myocardial ischemia are not well understood. Scheuer and Brachfeld (3) have shown correlations between ischemic epicardial ST segment changes and (1) decreased total myocardial oxygen consumption, (2) increased myocardial oxygen extraction, and (3) excess lactate production, as measured in coronary sinus effluent. Determination of myocardial oxygen tension by the polarographic technique by Sayen et al. (4) has provided an indication of the balance between oxygen supply and consumption in the local region of myocardium surrounding the electrode tip. Intramyocardial oxygen tension falls following coronary artery stenosis and returns to base-line levels when the stenosis is removed. Following coronary artery ligation, border areas surrounding a central infarcted zone often fail to show ST segment changes in unipolar epicardial electrograms despite significant decreases in oxygen tension in the underlying myocardium. In contrast, ST segment elevation is consistently recorded in unipolar intramyocardial electrograms in these border areas, providing a better correlation with the changes measured in local myocardial oxygen tension. Durrer et al. (5) have also been able to consistently record ST segment elevations in unipolar intramyocardial electrodes following coronary artery occlusion.

The electrophysiology of ST segment elevation as recorded with standard a-c-coupled recording systems has been studied by Katcher et al. (6) utilizing a d-c-coupled system. In d-c-coupled electrograms, base-line (TP segment) depression is the dominant change during myocardial ischemia. True ST segment elevation occurs later and is of lesser magnitude in these d-c-coupled recordings. Brantigan et al. (7) in our laboratory have reported the use of a Teflon membrane-vacuum mass spectrometer system for the determination of
local myocardial gas tensions. Levels of intramyocardial oxygen tension obtained by this new method are similar to those measured by the polarographic method. The mass spectrometer, however, provides the unique capability of measuring intramyocardial carbon dioxide tension in addition to oxygen tension.

The present study attempted to support three hypotheses: (1) the mass spectrometer provides a useful new tool for the quantification of intramyocardial oxygen and carbon dioxide tensions under conditions of regional myocardial ischemia, (2) the changes in local intramyocardial gas tensions are quantitatively related to the changes in local intramyocardial ST segment voltage, and (3) the changes in intramyocardial carbon dioxide tension provide a more sensitive quantitative indicator of myocardial ischemia than do the changes in myocardial oxygen tension.

**Methods**

Twenty dogs weighing 20–25 kg were anesthetized with chloralose (60 mg/kg, iv), intubated, placed on a Harvard respirator, and ventilated with room air. Arterial blood pressure was continuously monitored via a catheter inserted into the femoral artery, utilizing a Statham P23DB transducer. A left thoracotomy extending across the sternum was then performed. The pericardium was incised, the left atrial appendage was retracted, and a 3-cm segment of the proximal circumflex coronary artery (CCA) was dissected free. An electromagnetic flow probe (Biotronex series 6000 with Biotronex model 610 Pulso logic flowmeter), a variable screw-type constrictor, and a snare were placed around the exposed proximal CCA (Fig. 1). Arterial gas tensions were monitored intermittently.

Mass spectrometer probes for the measurement of intramyocardial gas tensions were inserted into the anterior wall of the left ventricle, a region supplied by the unconstricted left anterior descending coronary artery, and into the postero lateral wall, a region supplied by the constricted CCA. The mass spectrometer probes were inserted by making a small nick in the epicardium and pushing them gently into the myocardial wall until the sensing surface of each probe was well within the myocardial wall and the longitudinal axis of the probe was approximately parallel with the epicardial surface. The probes were then secured with a single 2-0 silk mattress suture. The probes used for the measurement of intramyocardial gas tensions consisted of 22-gauge stainless steel tubing slotted in the distal 2 cm and covered with heat-shrinkable Teflon (Fig. 1). The Teflon was heat shrunk over all of the stainless steel tubing except for a 19-mm segment near the distal tip where it remained in the expanded state. A tight seal of the tip of the probe was achieved by shrinking the Teflon over a round-tipped piece of stainless steel wire 0.125 inches long. The proximal end of the probe was then connected to a vacuum mass spectrometer, which sampled the mixture of dissolved gases surrounding the probe tip at a rate of $5 \times 10^{-6}$ ml/sec.

The gas mixture was withdrawn across the Teflon membrane, through the slots in the stainless steel tubing, and into the mass spectrometer. The individual gases were withdrawn in quantities proportional to their partial pressures in the tissues and analyzed according to their molecular weights. The probes were calibrated before each experiment in a glass tonometer at 37°C with two known gas mixtures. Calibration of the Teflon probe–mass spectrometer system has been shown to be stable for the duration of these experiments with a drift in calibration of only 4% in 24 hours (7). The 1/e response time of the system was 1.5 minutes for the measurement of oxygen tension and 3 minutes for the measurement of carbon dioxide tension. The 99% response time was 5.1 minutes for oxygen tension and 9.9 minutes for carbon dioxide tension. To allow for this inherent time delay in gas tension determinations, measurements of the intramyocardial gas tensions were recorded for at least 10 minutes following a given ischemic intervention.

**Epidermal or intramyocardial electrocardiogram (ECG) electrodes were also placed in the posterolateral myocardial wall adjacent to the mass spectrometer probe. The electrodes were made of Teflon-coated silver wire 0.001 inches in diameter. The epicardial electrodes consisted of a 12-inch segment of wire, the tip of which was fixed to the center of a plastic disk 5 mm in diameter, with two holes near the periphery of the disk used for suturing the electrodes to the epicardial surface. The multicontact intramyocardial electrodes consisted of ten Teflon-coated wires 0.001 inches in diameter inserted into the shaft of a 22-gauge needle which had...**

*FIGURE 1 Schematic presentation of the experimental preparation. The insert in lower right is a cutaway view of the mass spectrometer probe used in the study. Ant. desc. cor. a. = anterior descending coronary artery, circumflex cor. a. = circumflex coronary artery, Ao = aorta, P. A. = pulmonary artery, R. A. = right atrium, R. V. = right ventricle, L. V. = left ventricle, and 22G. st. steel wire = 22-gauge stainless steel wire.*

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side holes drilled at 1-mm spacings. The tips of each of the ten wires were passed through side holes and fixed with epoxy cement, resulting in a multicontact electrode with ten recording points spaced at 1-mm intervals. Following insertion of the mass spectrometer and the ECG probes, approximately 1 hour was allowed for stabilization of gas tensions and ST segment voltages. One of two interventions was then performed.

**PERMANENT CORONARY ARTERY OCCLUSIONS**

Branches of the CCA supplying areas in which mass spectrometer probes and epicardial ECG electrodes had been placed were permanently ligated, and gas tension and ST segment changes were monitored for 120 minutes.

**GRADED PROXIMAL CORONARY ARTERY STENOSIS**

The proximal CCA was progressively stenosed utilizing the screw-type variable constrictor, and the degree of stenosis was assessed by recording with an electromagnetic flowmeter the reactive hyperemic response in mean circumflex coronary artery flow following a 10-second occlusion. As more constriction was applied, the reactive hyperemic response could be completely abolished without a significant reduction in mean flow (≤15%). This degree of constriction was termed a "critical stenosis," with further degrees of stenosis, a significant (>15%) reduction in mean flow could be produced. Previous studies in this laboratory have shown that a critical stenosis corresponds angiographically to a decrease in the coronary artery luminal diameter to approximately 25% of the initial value (8).

The positions of the mass spectrometer probes and the epicardial or intramyocardial ECG electrodes with respect to the anatomical distribution of the epicardial coronary vessels were documented with a Polaroid CU-5 Land camera. Following each experiment, the position of each mass spectrometer probe within the left ventricular wall was determined by dissection and then recorded photographically. X rays and photographs following dissection were also employed to document the position and the depth of the intramyocardial electrode contacts with respect to both the endocardial and epicardial surfaces and the nearby mass spectrometer probes.

**Results**

For the entire group of 20 dogs, the mean control carbon dioxide tension was 43.0 ± 2.0 mm Hg, the mean control oxygen tension was 23.3 ± 1.9 mm Hg, and the mean control ST segment voltage was 2.9 ± 1.0 mv.

**PERMANENT CORONARY ARTERY OCCLUSIONS**

Permanent ligation of branches of the CCA resulted in a regional increase in myocardial carbon dioxide tension and epicardial ST segment voltage and a decrease in myocardial oxygen tension. The results of six experiments, in which gas tensions and epicardial ST segment voltages were monitored for 2 hours following ligation, are presented in Figure 2. Epicardial ST segment voltage increased rapidly, reaching a maximum of 15 ± 4 mv above control voltage 15 minutes after ligation. Myocardial carbon dioxide tension reached its maximum value, 121 ± 26 mm Hg above control, 30 minutes after ligation. The fall in myocardial oxygen tension followed a time course similar to that seen for the rise in carbon dioxide tension and epicardial ST segment voltage, reaching a minimum of 15 ± 4 mm Hg below control 15 minutes after ligation. Over the 2 hours following ligation, epicardial ST segment voltage did not change significantly. Myocardial carbon dioxide tension showed a gradual decrease, reaching a level of 56 ± 17 mm Hg above
control 120 minutes after ligation. In contrast, myocardial oxygen tension did not change significantly; 2 hours after ligation, the oxygen tension remained at a level 11 ± 5 mm Hg below the control value.

**GRADED PROXIMAL CORONARY ARTERY STENOSES**

Changes in unipolar ST segment voltage recorded in epicardial electrodes were compared with changes in ST segment voltage recorded in intramyocardial electrodes. In seven dogs, application of a critical stenosis to the proximal CCA resulted in a decrease in myocardial oxygen tension from 24 ± 4 to 12 ± 3 mm Hg and an increase in carbon dioxide tension from 41 ± 3 to 74 ± 6 mm Hg. Epicardial ST segment voltage increased from 1 ± 1 to 3 ± 2 mv. In two of these seven studies, however, no change in ST segment voltage was noted in the unipolar epicardial electrograms which were recorded on a region of the epicardium overlying the mass spectrometer probe. In contrast, an increase in myocardial carbon dioxide tension and an increase in ST segment voltage measured in unipolar intramyocardial electrograms were noted in all seven studies. In those two studies in which no changes in epicardial ST segment voltage were apparent, increases of 1.4 and 4 mv were seen in the intramyocardial electrograms recorded at a depth within the myocardial wall comparable to the depth of the mass spectrometer probe.

Unipolar ST segment changes recorded by multicontact intramyocardial electrodes were therefore used in subsequent studies, and ST segment voltages recorded by these electrodes were compared with the changes in myocardial gas tensions. Twenty-four stenoses of variable severity were studied in nine dogs, and the stenoses were divided into five groups according to the associated percent decrease in mean circumflex flow compared with control flow. The resultant values for intramyocardial gas tensions and unipolar intramyocardial ST segment voltages are presented in Table 1 and summarized graphically in Figure 3. Prior to application of any stenosis, the mean control myocardial carbon dioxide tensions in these nine dogs was 43.3 ± 3.3 mm Hg, the mean control myocardial oxygen tension was 25.9 ± 2.8 mm Hg, and the base-line ST segment voltage was 3.3 ± 1.6 mv. Following application of a critical stenosis, there was an increase in myocardial carbon dioxide tension of 21 ± 4 mm Hg above control and an associated decrease in myocardial oxygen tension of 13 ± 4 mm Hg below control (Fig. 3). Intramyocardial ST segment voltage, recorded in an electrode comparable in depth to the nearby mass spectrometer probe, increased 3 ± 2 mv following application of the critical stenosis. A further increase in the degree of CCA stenosis resulted in increases in intramyocardial carbon dioxide tension and unipolar ST segment voltage. Although myocardial oxygen tension decreased 13 ± 4 mm Hg with application of a critical stenosis, only a 7-mm Hg further decrease was noted when a severe degree of stenosis, associated with an 80% reduction in mean CCA flow, was applied. In contrast, increasing proximal CCA stenosis from critical to this same severe degree of stenosis resulted in a further increase in myocardial carbon dioxide tension of 84 mm Hg and a further increase in myocardial ST segment voltage of 21 mv.

Due to the inherent time delay in the mass spectrometer system, changes in intramyocardial ST segment voltage following placement of a given coronary artery constriction preceded changes in myocardial gas tensions by several minutes. However, both ST segment and gas tension values appeared to reach a plateau in approximately 10 minutes and then remained relatively stable for periods of up to 1 hour (Fig. 4). Linear regression
ST SEGMENTS AND GAS TENSIONS IN ISCHEMIA

TABLE 1

Intramyocardial Gas Tensions and ST Segment Voltages with Graded Coronary Stenosis

<table>
<thead>
<tr>
<th>Dog</th>
<th>PmO₂ (mm Hg)</th>
<th>PmCO₂ (mm Hg)</th>
<th>ST (mv)</th>
<th>Mean ± SE</th>
<th>PmO₂ (mm Hg)</th>
<th>PmCO₂ (mm Hg)</th>
<th>ST (mv)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>23</td>
<td>25</td>
<td>-1.3</td>
<td>0</td>
<td>9</td>
<td>48</td>
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<td>0</td>
<td>7</td>
<td>11.5</td>
<td>66</td>
<td>4.0</td>
</tr>
<tr>
<td>3</td>
<td>20</td>
<td>44</td>
<td>4.5</td>
<td>0</td>
<td>11</td>
<td>73</td>
<td>13.5</td>
</tr>
<tr>
<td>4</td>
<td>27</td>
<td>58</td>
<td>4.0</td>
<td>14</td>
<td>18.5</td>
<td>69</td>
<td>5.0</td>
</tr>
<tr>
<td>5</td>
<td>15.5</td>
<td>50</td>
<td>3.0</td>
<td>13</td>
<td>8</td>
<td>64</td>
<td>3.5</td>
</tr>
</tbody>
</table>

Mean ± SE: 24.9 ± 4.2, 43.4 ± 5.5, 2.0 ± 1.1

Percent reduction in mean CCF: 5.4 ± 3.0, 11.6 ± 1.8, 64.0 ± 4.3, 5.2 ± 2.2

Control*: PmO₂ (mm Hg) 25, 40, 44, 58, 50, 43.4 ± 5.5

Stenosis†: PmO₂ (mm Hg) 25, 58, 53, 50, 42, 45.6 ± 5.8

ST (mv) 0, 4.0, 13.5, 5.0, 3.5, 5.2

Overall Mean ± SE: 24.5 ± 3.4, 45.6 ± 5.8, 4.9 ± 2.9

Mean ± SE: 26.2 ± 2.5, 45.6 ± 5.8, 4.7 ± 3.0

Overall Mean ± SE: 23.0 ± 2.0, 42.8 ± 5.8, 1.9 ± 1.1

* Control = myocardial oxygen tension (PmO₂), myocardial carbon dioxide tension (PmCO₂), and ST segment voltages prior to creation of the first of one or more graded stenoses.
† Stenosis = reduction of mean circumflex coronary flow (CCF), PmO₂, PmCO₂, and ST segment voltages recorded 10-15 minutes after creation of each degree of stenosis.
‡ Overall mean = mean control values for the nine dogs.

Analysis revealed $r = 0.732$ for changes in myocardial carbon dioxide tension vs. changes in intramyocardial ST segment voltage and $r = -0.655$ for changes in oxygen tension vs. changes in ST segment voltage (9).

Discussion

The results of this study support our three initial hypotheses: (1) the changes in myocardial oxygen and carbon dioxide tensions as determined by mass spectrometry are sensitive indicators of myocardial ischemia, (2) the changes in intramyocardial gas tensions correlate with local unipolar ST segment changes recorded from nearby intramyocardial electrodes, and (3) with degrees of proximal coronary stenoses ranging from a critical stenosis with little or no reduction in mean coronary artery flow to severe degrees of stenosis with an 80% reduction in mean flow, the changes in myocardial carbon dioxide tension are of greater magnitude than the changes in oxygen tension and thus provide a more sensitive indicator of the functional significance of a given coronary lesion.

The increases in myocardial carbon dioxide ten-
Myocardial gas tensions ($P_mCO_2$ and $P_mO_2$) and unipolar ST segment voltage changes recorded at comparable depths within the ventricular wall ($\Delta ST$) in a single experiment (dog 4). Four graded proximal stenoses resulting in reductions of mean circumflex flow of 14, 29, 35, and 45%, respectively, followed by total release were studied.

Reduction mean flow % constriction release 14 29 35 45

Myocardial gas tensions recorded during ischemia probably result from changes in myocardial metabolism secondary to the decreased coronary artery flow and the associated decreased delivery of oxygen. Under normal aerobic conditions, myocardial metabolism consists of oxidation of free fatty acids as well as variable amounts of glycolysis. Carbon dioxide under these conditions is generated by oxidative respiration. Under anaerobic conditions, a stimulation of glycolysis can be demonstrated; this stimulation is probably related to decreased tissue levels of adenosine triphosphate, increased levels of inorganic phosphate, adenosine diphosphate, and adenosine monophosphate, and the resultant activation of phosphofructokinase, the rate-limiting enzyme of glycolysis (10). The pyruvic acid produced by glycolysis is converted under these hypoxic conditions to lactic acid. This increased intracellular formation of hydrogen ions then results in increased generation of carbon dioxide according to the relationship:

$$H^+ + HCO_3^- \rightarrow H_2CO_3 \rightarrow H_2O + CO_2$$

Rovetto et al. (11) have demonstrated that the rate of glycolysis increases less under ischemic conditions than it does under anoxic conditions. Previous studies have shown that the rate of glycolysis is increased during ischemia compared with that during normally perfused, normoxic conditions (12, 13). Relative inhibition of one of the enzymes in the glycolytic pathway by the local accumulation of metabolic end products when coronary flow is diminished may explain the relative decrease in glycolysis observed by Rovetto et al. (11) during ischemia. However, buffering of the hydrogen ions associated with the increased production of lactate would result in the increased generation of carbon dioxide under both ischemic and anoxic conditions. During ischemia, a decreased removal of the hydrogen ions or the carbon dioxide generated would probably result in an increase in myocardial carbon dioxide tension. Opie et al. (14) have demonstrated that the difference between coronary arterial and local venous carbon dioxide tension increases and pH decreases during ischemia. Local accumulation of carbon dioxide in the myocardium, therefore, probably reflects the balance between tissue carbon dioxide production and its clearance by local coronary blood flow. However, the relative extent to which increased carbon dioxide production and decreased tissue washout is responsible for the increase in myocardial carbon dioxide tension recorded during ischemia cannot be determined from these experiments. Following total coronary artery occlusion, the spontaneous decline in myocardial carbon dioxide tension after the initial rise suggests the cessation of myocardial carbon dioxide production. Weisfeldt et al. (15) have demonstrated that an increased carbon dioxide tension, independent of pH, results in a significant decrease in ventricular performance during ischemia. It is possible, therefore, that increased tissue carbon dioxide tension may be a mechanism involved in the rise in end-diastolic pressure, known clinically to occur transiently during the pain of angina pectoris.

Other means of assessing the status of local myocardial metabolism include measurement of tissue lactate or of high-energy phosphate levels by biopsy techniques (11, 16). Although tissue biopsy determinations are by necessity periodic, mass spectrometry provides a continuous recording of myocardial gas tensions. Measurements of lactate or carbon dioxide tensions in the coronary sinus blood cannot be reliably employed for the detection of regional ischemia, since this effluent is a mixture of blood draining ischemic as well as nonischemic regions of the myocardium. Opie et al. (14) by selective catheterization of the coronary vein drain-
ing a ligated coronary artery have been able to
detect regional metabolic changes. These authors
noted that following a coronary branch ligation
carbon dioxide tension in the local venous blood
rose from 50 ± 1 to 59 ± 3 mm Hg but that coronary
sinus carbon dioxide tension did not change. fol-
lowing ligation of a larger coronary branch, local
venous carbon dioxide tension rose to 64 ± 4 mm
Hg and coronary sinus carbon dioxide tension rose
from 49 ± 1 to 56 ± 2 mm Hg. These changes in
carbon dioxide tension can be contrasted with the
changes in myocardial carbon dioxide tension re-
corded following comparable branch ligations in
the present study. Myocardial carbon dioxide ten-
sion rose in the present experiments from 44.6 ± 3.6
mm Hg to 166.0 ± 24.4 mm Hg 30 minutes after
ligation. Comparison of the magnitude of the
changes in carbon dioxide tension in local venous
blood with those recorded in the present study
could be interpreted as evidencing a seven- to
eightfold dilution of blood draining the ischemic
myocardium by blood from adjacent nonischemic
regions. If, however, the diffusion of carbon dioxide
from ischemic tissues is impaired under conditions
of low coronary flow, the appearance of carbon
dioxide in the local venous blood would be im-
paired. The relative extent to which each of these
two factors contributes to the smaller magnitude of
changes in carbon dioxide tension in local venous
blood compared with the changes in the myocar-
dium cannot be determined from the present
study. Nevertheless, selective venous catheteriza-
tion and mass spectrometry provide alternative
means of studying regional myocardial ischemia. A
combination of these two techniques could permit
study of local changes in cardiac metabolism such
as changes in the rate of myocardial carbon dioxide
production by ischemic regions of the myocardium.

Changes in myocardial carbon dioxide and oxy-
gen tensions together appear to provide a better
indication of the degree of myocardial ischemia
than do changes in myocardial oxygen tension
alone. Only a 7-mm Hg additional decrease in
myocardial oxygen tension was noted with an
increase in the degree of proximal stenosis from
critical stenosis with little or no reduction in mean
circumflex flow to a severe stenosis associated with
an 80% reduction in mean flow. In contrast, myo-
cardial carbon dioxide tension increased 84 mm Hg
over the same range of coronary stenoses. The more
marked changes in carbon dioxide tension also
related more linearly with changes in intramyocar-
dial ST segment voltage than did the changes in
myocardial oxygen tension.

The presence of significant change in myocardial
gas tensions and intramyocardial ST segment volt-
age following application of a critical stenosis,
which does not significantly alter mean coronary
flow, implies a maldistribution of myocardial blood
flow and resultant ischemia. A decrease in myocar-
dial blood flow to deeper myocardial layers has
been demonstrated by the radioactive microsphere
technique following application of a coronary ste-
nosis which results in a 70–75% reduction in mean
control coronary flow (17). Similar myocardial
blood flow data for stenoses, which result in little or
no reduction in mean coronary flow, are not availa-
ble.

Intramyocardial ST segment changes, an inde-
dependent indicator of regional myocardial ischemia,
increased exponentially when they were plotted
against the percent reduction in mean circumflex
flow. When ST segment changes were recorded in
epicardial electrodes, significant gas tension
changes were noted in all cases. However, ST
segment changes recorded in unipolar epicardial
electrodes were a less-sensitive indicator of myo-
cardial ischemia than were those recorded in in-
tramyocardial electrodes. Epicardial electrodes
sometimes failed to detect the presence of underly-
ing ischemia, as evidenced by significant changes
in local myocardial gas tensions and intramyocar-
dial ST segment voltage. This lack of sensitivity,
which was also found by Sayen et al. (4), may
reflect an inability of the epicardial electrodes to
test ischemic changes in deeper myocardial lay-
ers. In contrast, the correlation between intramyocar-
dial ST segment changes and local myocardial
gas tension changes was excellent.

Changes in myocardial gas tensions from control
values rather than absolute gas tension measure-
ments appear to be a more useful index of myocar-
dial ischemia for comparing or averaging changes
recorded in different animals, since each animal
will have unique mass spectrometer probe place-
ments and control hemodynamic parameters. The
role of factors such as tissue trauma, arterial gas
tensions, myocardial temperature, and systemic
heparinization on the measurement of gas tensions
has been discussed in detail previously (18) and
will be summarized in the following paragraphs.

Insertion of a mass spectrometer probe into the
myocardial wall undoubtedly results in a variable
amount of intramyocardial hemorrhage with re-
sultant admixture of oxygenated arterial blood.
The finding at the end of a study of grossly visible
hematoma formation around the probe tip was
associated with the recording of excessively high
base-line myocardial oxygen tensions (> 40 mm Hg). Data from such probes were excluded from this study. Although Moss (19) found a mean control oxygen tension by the polarographic technique of 10 ± 1.8 mm Hg in deeper myocardial layers and 18 ± 2.3 mm Hg in superficial layers, Winbury et al. (20) found a mean base-line oxygen tension of 16.5 ± 1.9 mm Hg (range 8 to 33 mm Hg) and 25.7 ± 2.1 mm Hg (range 16 to 41 mm Hg) in the deep and superficial layers, respectively. Both studies utilized probes 0.254 mm in diameter. Base-line oxygen tensions obtained by mass spectrometry in the present study are in agreement with tensions recorded in the latter study. The majority of probe insertions resulted in the recording of a stable and reproducible base-line oxygen tension with return to the initial control level following the removal of an ischemic stimulus.

Monitoring of arterial gas tensions by frequent sampling is critical to the interpretation of changes in intramyocardial gas tensions. Intramyocardial carbon dioxide tension has been shown to increase linearly with increasing arterial carbon dioxide tension. In contrast, variation in arterial oxygen tension in the range of 80 to 200 mm Hg has been shown to have only a minor effect on myocardial oxygen tension.

Since mass spectrometer probe calibration varies with temperature, monitoring of myocardial or core temperature and subsequent probe calibration at the appropriate temperature are necessary. Systemic heparinization also has been noted to consistently produce higher myocardial oxygen tensions than those recorded without heparin. This increase is most likely the result of increased admixture of arterial blood.

The results of this study suggest that the mass spectrometer provides a method for continuously monitoring intramyocardial carbon dioxide and oxygen tension and that increases in carbon dioxide tension and in unipolar intramyocardial ST segment voltage provide sensitive quantitative correlates of regional myocardial ischemia. Furthermore, in combination with the studies of Weisfeldt et al. (15), these data suggest that an increase in tissue carbon dioxide tension may be an important pathophysiological mechanism in the reduction of left ventricular function which is known to occur during myocardial ischemia.

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