Relationship Between Epicardial ST-Segment Elevation, Regional Myocardial Blood Flow, and Extent of Myocardial Infarction in Awake Dogs

ROBERT G. IRVIN, M.D., AND FREDERICK R. COBB, M.D.

SUMMARY This study was designed to examine the relationship between epicardial ST change (EpST) and regional myocardial blood flow (RMBF) following coronary occlusion and extent of myocardial infarction (MI) in awake dogs. Fifteen min and two hr after coronary occlusion simultaneous measurements of EpST and RMBF were made. Six days later histologic MI and RMBF were determined in transmural myocardial samples from each electrode site. Greatest ST elevation occurred at sites of greatest ischemia and MI. However, 15 min after occlusion 29% of sites with greater than 50% MI and 39% of sites with greater than 50% reduction in RMBF did not demonstrate ST elevation greater than 2 mV. There were poor correlations between EpST and MI (r = 0.59) and RMBF (r = 0.57). Comparable relationships were observed two hr after occlusion. In the present study, there were not close quantitative or qualitative relationships between EpST and MI or RMBF. A good correlation was observed between RMBF at two hr and MI (r = 0.89).

EPICARDIAL ST-SEGMENT CHANGES are widely used as an index of myocardial injury resulting from ischemia in experimental animals. Numerous investigators have reported that the extent and magnitude of epicardial ST elevation early after initiation of ischemia reliably indicate the degree of ischemic injury and predict the extent of subsequent infarction in the tissue underlying the electrode. These studies were performed in anesthetized animals and myocardial infarction was determined in transmural biopsies by measuring myocardial creatine phosphokinase (CPK) depletion and evidence of early histologic injury 24 hours after occlusion. Similar quantitative relationships have been reported between ST changes from intramyocardial electrodes and CPK depletion at 24 hours in chronically prepared dogs. Other observers have indicated that there is not a close relationship between epicardial ST elevation and certain indices of myocardial injury.

The present study was designed to evaluate further the relationship between epicardial ST segment changes measured 15 min and 2 hours after permanent coronary artery occlusion and extent of subsequent myocardial infarction. Myocardial infarction was quantitated from serial histo-logic sections determined 6 days after occlusion at which time intact and infarcted myocardium were sharply delineated. Epicardial ST changes were also related to simultaneous measurement of regional myocardial blood flow using radioisotope labeled microspheres. Studies were carried out in chronically prepared awake dogs to avoid the variables introduced by anesthesia and acute surgery.

Methods

Seventeen adult mongrel dogs of both sexes weighing 20-30 kg were studied. They were anesthetized with sodium thiamylal (30-40 mg/kg, i.v.) and ventilated with a respirator (Harvard model 607). The heart was exposed through a left thoracotomy at the fourth intercostal space. Polyvinyl chloride catheters with outer diameters of 3 mm were introduced into the aortic arch via the left internal mammary artery and into the left atrium via the atrial appendage. Both catheters were filled with heparin and secured by sutures. The left anterior descending coronary artery (LAD) was isolated approximately 2 cm from its origin and either a snare (13 dogs) or adjustable pneumatic (4 dogs) occluder was positioned around it just distal to the first diagonal branch. The snare occluder consisted of a 2.5 mm outer diameter polyethylene tubing flared at one end to allow suturing to the epicardial tissue adjacent to the coronary artery and 4 silk which was secured to the end of the tubing, looped loosely around the coronary artery, and then threaded through the tubing.

Ten color-coded epicardial electrodes, insulated except for the distal 2 mm contact site, were sutured to the anterolateral surface of the left ventricle. The electrodes were positioned approximately 1 cm apart so that some sites would be
within and some clearly outside of the area supplied by the artery to be occluded (fig. 1). The catheters, occluder, and electrode wires were tunneled to a subcutaneous pouch. The chest was closed and the dogs were allowed to recover from surgery.

Studies were performed 6-8 days postoperatively at which time the dogs had fully recovered and had no evidence of infection. Mean hematocrit was 44 (range 39-50). Morphine sulfate (10 mg, i.m.) was given 1 hour before the study for mild sedation. Local anesthesia with 2% lidocaine was used when the catheters, occluder, and electrode wires from the subcutaneous pouch were brought to the exterior surface. Studies were performed in a quiet, dimly illuminated laboratory with the dogs awake, loosely restrained, and resting quietly on their right sides. Standard lead I of the electrocardiogram was recorded. Phasic and mean aortic and left atrial blood pressures were measured with pressure transducers (Statham model 3917-A). Epicardial electrograms were recorded as unipolar leads at a sensitivity of 5 mV/cm. At a paper speed of 50 mm/sec, ST segments were measured 100 msec from the beginning of the QRS. The T-P segment was considered the isoelectric line, and the mean of five complexes was used as the value for each electrode site. Sites demonstrating conduction disturbances were excluded from analysis. Data were recorded on an eight-channel direct-writing oscillograph (Hewlett-Packard model 8800) and an eight-channel magnetic tape recorder (Hewlett-Packard model 3917-A).

The four animals with adjustable pneumatic oculiders were subjected to serial transient occlusions to vary the reproducibility of the electrograms from chronically implanted electrodes. Serial transient complete occlusion resulted in reproducible ST elevation. These animals were not subjected to permanent occlusion and were not included in the following protocol. The remaining 13 animals with a snare type occluder were subjected to permanent complete coronary occlusion. Prior to coronary occlusion, an intravenous bolus injection of lidocaine, 2 mg/kg, was given prophylactically to each dog to minimize early arrhythmias. Following control hemodynamic and electrocardiographic measurements, the left anterior descending coronary artery was gradually occluded over a 10 min interval. Intravenous morphine sulfate, total dose 10 mg, was given in 2-3 mg increments to minimize discomfort resulting from coronary occlusion. Hemodynamic measurements and epicardial electrograms were recorded 15 min and 2 hours after permanent LAD occlusion.

Regional myocardial blood flow was determined as previously described\(^{17}\) at 15 min and 2 hours after LAD occlusion by injecting carbonized microspheres 7-10 μm in diameter labeled with \(^{46}\)Sc and \(^{85}\)Sr into the left atrium. A reference blood sample was collected at a constant rate from the aortic catheter beginning simultaneously with each injection and continuing for 90 sec using a withdrawal pump (Harvard model 1210). There was no change in heart rate or arterial or left atrial pressures with the microsphere injections.

Six days after infarction the dogs were sacrificed and the hearts placed in a 10% buffered formalin solution for 4-6 days. The lumen of the LAD coronary artery was examined to verify total occlusion by the snare. The great vessels, atria, right ventricle, and large epicardial blood vessels and fat were removed from the left ventricle. The average weight of the left ventricle was 118.7 g (range 90-152). The ventricle was cut into four transverse rings from base to apex as previously described.\(^{17}\) Transmural myocardial specimens (average weight 500 mg) were taken at the site of each epicardial electrode and prepared for histologic sections. Four longitudinal sections were taken at different depths through each specimen and stained with hematoxylin and eosin. Infarcted myocardium was clearly delineated from intact myocardium 6 days after coronary occlusion. Infarcted myocardium was characterized by infiltration of inflammatory cells, cellular dissolution, hemorrhage, and loss of normal cellular architecture. Inflammatory cells and cellular dissolution were greatest at the periphery of the infarct zone and extended in varying degrees into the center of the necrotic area. In the center of the necrotic zone myocytes had undergone karyolysis. Hemorrhage was usually present when the extent of infarction exceeded 75% of the myocardial sample and was usually absent at the periphery of the infarcted zone and in areas of patchy infarction. Sketches were made of the intact and infarcted myocardium in each histologic section using a projection microscope. The percentage infarction at each biopsy site was determined by planimetry using a sonic X-Y digitizer (Graf-Pen) interfaced to an IBM computer programmed to calculate areas.

Transmural myocardial sections 5-6 g in size were taken from posterior nonischemic regions and each region from which core specimens were taken. Each transmural section was subdivided into four equal layers from epicardium to endocardium. Each myocardial sample was placed in a counting vial and counted with the reference blood samples.

**Figure 1.** Schematic representation of the distribution of the epicardial electrodes on the anterolateral surface of the left ventricle and position of the snare on the left anterior descending coronary artery. The right panels are epicardial electrograms before and 15 min after complete coronary artery occlusion.
in a gamma spectrometer at optimum window settings to correspond to the peak energies of each nuclide. Flow to each region of myocardium was calculated by using the formula \( Q_m = Q_r \cdot C_m/C_e \), where \( Q_m \) = myocardial flow (ml/min); \( Q_r \) = reference blood flow (ml/min); \( C_m \) = counts/minute in myocardium; and \( C_e \) = counts/minute in reference blood samples. Myocardial blood flow (ml/min) was divided by the sample weight and expressed as ml/min per g.

Hemodynamic data were measured directly from the oscillographic records. Regression analyses were carried out using the least squares method and correlation coefficients.

**Results**

**Hemodynamic Changes**

Table 1 presents hemodynamic measurements before and 15 min and 2 hours after coronary occlusion. During the control period the mean heart rate was 82 ± 4, aortic pressure 96 ± 3 mm Hg, and left atrial pressure 5 ± 2 mm Hg. Fifteen minutes after coronary occlusion, there was a highly significant increase in all parameters with a heart rate of 112 ± 5, aortic pressure 109 ± 3 mm Hg, and left atrial pressure of 10 ± 1 mm Hg. Two hours after occlusion, the heart rate had decreased to 91 ± 4 with no significant change in aortic or left atrial pressure.

**ST-Segment Changes**

Figure 1 (right panel) demonstrates representative epicardial electrograms from an individual dog recorded before and 15 min after coronary occlusion. Prior to coronary occlusion, control values for epicardial ST segments average 1.1 mV with a range from -1.0 to +3.0 mV. Fifteen minutes after coronary occlusion, 48 of 125 electrode sites demonstrated ST elevation greater than 2 mV above control values and the sum of ST elevations was 280 mV. Two hours after coronary occlusion, 33 electrode sites demonstrated greater than 2 mV ST elevations above control and the sum of ST elevations had decreased 32%, range 10% increase to 93% decrease. Seven electrode sites at 15 min and nine sites at 2 hours demonstrated greater than 1 mV ST depression from control. Conduction disturbances were present in ten sites from five different dogs out of a total of 250 epicardial electrograms. The latter data were not included in the analyses.

**Relationship between ST Changes and Extent of Histologic Infarction**

Figure 2 demonstrates the relationship between epicardial ST changes 15 min after coronary occlusion and the extent of histologic myocardial infarction in the transmural specimen from each electrode site. The greatest degree of ST elevation occurred at sites with the greatest extent of myocardial infarction. However, for a given degree of infarction, there was a wide range of ST changes. Regression analysis demonstrated a correlation coefficient of 0.59. In addition, ST changes did not predict areas of subsequent infarction in many instances. Fourteen of 48 sites (29%) with greater than 50% infarction and ten of 37 sites (27%) with greater than 80% infarction demonstrated 2 mV or less ST elevation 15 min following coronary occlusion. Using 2 mV increase in ST segments as an index of "significant" ischemic injury,^1,6,9^ we found that six of 50 sites (12%) demonstrated 2 mV or greater ST elevation but 1% or less myocardial infarction. Figure 3 demonstrates the relationship between ST segment changes at 2 hours post occlusion and extent of myocardial infarction. ST segments decreased at most sites between 15 min and 2 hours. The relationship between ST-segment elevation and myocardial infarction was similar at 15 min and 2 hours. Many sites with extensive infarction did not demonstrate ST elevation greater than 2 mV and a wide range of ST change was present for a given degree of infarction. Regression analysis demonstrated a correlation coefficient of 0.61.

To determine whether epicardial ST changes were more sensitive to ischemic injury occurring in the epicardial layer, ST changes at 15 min were plotted against the extent of histologic infarction in the epicardial half of each myo-
cardiac specimen (fig. 4). It is apparent that the correlation was not improved by relating the ST changes to infarction in the epicardial half of the myocardium.

**ST-Segment Depression**

ST depression greater than 1 mV occurred at seven sites 15 min after occlusion. Five of these sites had blood flow comparable to nonischemic regions and developed no infarction. Two sites had greater than 60% infarction and myocardial blood flow less than 33% of control. Nine sites had ST depression greater than 1 mV at 2 hours. Only two of these sites had greater than 25% infarction or myocardial blood flows less than 33% of control. There was no consistent pattern between the transmural distribution of infarction and ST depression. Five sites demonstrated greater than 2 mV ST depression and are not plotted on the graphs.

**Relationship between ST Changes and Regional Myocardial Blood Flow**

Figure 5 demonstrates the relationship between epicardial ST changes and myocardial blood flow to the region in which the epicardial electrode was located. Blood flow to the ischemic region is expressed as the percent of flow to the nonischemic posterior regions. Both measurements were obtained simultaneously 15 min after coronary occlusion. In general, ST elevation greater than 2 mV did not occur unless myocardial blood flow was reduced to less than 50% of that to the nonischemic regions. The greatest degree of ST elevations occurred in the lowest flow regions. However, it is apparent that there is a wide range of ST elevation for a given flow value. Twenty-six of 66 electrode sites (39%) with blood flow less than 50% of that to nonischemic regions had less than 2 mV ST elevation. Regression analysis demonstrated a correlation coefficient of 0.57.

Two hours postcoronary occlusion, except for a single electrode site, ST elevation greater than 2 mV did not occur unless simultaneously measured blood flow was less than 50% of that of nonischemic regions (fig. 6). However, as was noted at 15 min, there was a wide scatter of data points in the region in which blood flow was reduced to less than 50%

---

**Figure 3.** The percent myocardial infarction in the transmural specimens at each electrode is plotted as a function of the corresponding epicardial ST-segment change recorded 2 hours after coronary artery occlusion. All electrode sites with measurable ST segments from all 13 dogs are plotted.

**Figure 4.** The percent myocardial infarction in the epicardial half of the biopsies at each electrode site is plotted as a function of the corresponding epicardial ST-segment change recorded 15 min after coronary artery occlusion.

**Figure 5.** Epicardial ST-segment changes from control are plotted as a function of simultaneous myocardial blood flow to the region of the electrode. Both measurements were made 15 min after coronary artery occlusion.
of nonischemic area flow. Twenty-two of 54 electrode sites (41%) with blood flow less than 50% of blood flow to nonischemic regions had less than 2 mV ST elevation. Regression analysis demonstrated a correlation coefficient of 0.57.

Epicardial ST changes at 15 min were compared to simultaneous measurements of blood flow in the outer half of the myocardial sample underlying the electrode. Figure 7 illustrates that the correlation between ST elevation and blood flow was not improved by using blood flow to the epicardial half of the myocardial region.

Although the summed ST segments decreased in 12 dogs and increased in one dog and blood flow to the ischemic region increased in 12 dogs and decreased in one dog, there was not a close correlation between changes in the two measurements \( r = 0.52 \). There was also a poor correlation between the change in ST segments to individual electrode sites and changes in blood flow to the region of the electrode \( r = 0.19 \).

**Relationship between Myocardial Infarction and Myocardial Blood Flow**

The relationship between extent of myocardial infarction at each electrode site and myocardial blood flow to the circumferential regions from which the specimens were taken is illustrated in figure 8. Although there was scatter in data points, there was an inverse relationship between blood flow at 15 min and subsequent histologic infarction. Regression analysis demonstrated a correlation coefficient of 0.83. Figure 9 illustrates that the relationship between blood flow and infarction was improved 2 hours after coronary occlusion. Regression analysis demonstrated a correlation coefficient of 0.89. The improved relationship resulted from increments in blood flow between 15 min and 2 hours to certain of the sites which had initial low flow values but which did not develop extensive subsequent infarction.

**Discussion**

The purpose of the present study was to examine further the relationship between epicardial ST segment changes early after coronary artery occlusion and the extent of subsequent myocardial infarction. Epicardial ST changes were also related to simultaneous measurements of regional myocardial blood flow. The animals in the present study differed in certain aspects from those used by others. To avoid variables introduced by acute surgery and anesthesia, the present studies were carried out in chronically prepared awake animals fully recovered from surgery. Epicardial ST segments were recorded from multiple epicardial electrodes 15 min and 2 hours after ligation of the proximal left anterior descending coronary artery. ST-segment changes were related to simultaneous measurements of regional myocardial blood flow and extent of histologic infarct determined in transmural sections of the subjacent myocardium six days after coronary occlusion.

Previous studies have been carried out in open-chest anesthetized animals in which epicardial ST-segment changes after ligation of a branch of the left anterior descending cor-
Coronary artery were related to the extent of myocardial CPK depletion and histologic evidence of tissue necrosis 24 hours later. These investigators reported a close quantitative relationship between the extent and magnitude of ST-segment changes and these indices of myocardial injury. Hirshfeld et al. have also reported a direct relationship between ST elevation recorded from chronically implanted subepicardial electrodes and CPK depletion at 24 hours. Ginks et al., however, did not observe a close correlation between epicardial ST elevation 15 min following occlusion of the left anterior descending coronary artery proximal to the apical branch and CPK depletion at 7 days (r = 0.66).

In the present study there was not a close quantitative relationship between epicardial ST segment changes and the extent of myocardial infarction determined from histologic sections at 6 days (r = 0.59). Fifteen minutes following permanent occlusion, ST segments increased less than 2 mV in 29% and 27% of sites with greater than 50 and 80% histologic infarction, respectively. ST segments increased greater than 2 mV in 17% of the sites with 1% or less myocardial infarction. Similar changes were present 2 hours postocclusion.

Other reports have also pointed out certain limitations to using ST-segment changes as indices of myocardial injury. Kjekshus et al. reported that ST-segment changes were closely related to epicardial injury as measured by CPK depletion, but that ST-segment elevation frequently did not occur when the myocardial injury was limited to the endocardium. They concluded that the subendocardium was relatively "silent" with respect to elevation of epicardial ST segments. In the present study, the relationship between ST segment changes and infarction was not improved by limiting the analyses to infarction in the epicardial half of the biopsy.

Some investigators have observed considerable variability in the relationship between epicardial ST-segment changes and other indices of tissue injury. Karlsson et al. observed that ST increase was associated with metabolic indices of ischemia but that there was no relationship between the magnitude of epicardial ST changes and intramyocardial metabolic changes. Angell et al. noted that although myocardial oxygen tension was reduced at sites of ST elevation, regression analyses between the two measurements resulted in an average correlation coefficient of 0.66, indicating considerable variability in the relationship.

Cohen and Kirk, using open-chest dogs, observed that ST elevations following occlusion of a distal coronary artery were reduced when the ischemia was increased by more proximal occlusion of the artery. Studies by Holland and Brooks may explain this paradoxical reduction in ST segments as the ischemic region increased. These investigators examined the relationship between epicardial ST changes and the size and shape of the ischemic area. Mathematical predictions of ST changes based on the solid angle theorem conformed to measured ST-segment changes following coronary occlusion in closed-chest anesthetized pigs. These investigators concluded that ST changes were a complex function of size, shape, and transmural location of the ischemic area, and that epicardial ST-segment elevation is inversely related to the area of ischemia.

In the present study, the occluders were placed on the proximal left anterior descending coronary artery rather than a distal or side branch. The ischemic area would be expected to be larger in the present study than in those carried out by Maroko. It is possible that such differences may have contributed to the different results in the present and previous studies. Hirshfeld and associates, however, have utilized proximal left anterior descending coronary artery
occlusion and observed a direct relationship between ST-segment changes and CPK depletion at 24 hours.

In the present study, myocardial necrosis was quantitated six days after infarction. The six-day period was used so that the infarcted myocardium would be sharply delineated from the intact myocardium, and thus easily quantitated by routine histologic stains. The extent of infarction was quantitated from serial step histologic section through the transmural specimen. Early after acute infarction, histologic evidence of myocardial necrosis is still evolving and it is difficult to distinguish reversible and irreversible cellular injury on the basis of anatomical criteria. 

CPK depletion at 24 hours has been used in preference to histologic necrosis at 24 hours to estimate extent of myocardial infarction. The use of CPK depletion to quantitate ischemic injury is based on studies carried out in anesthetized rabbits subjected to complete coronary occlusion in which grossly identified myocardial infarction 24 hours after occlusion was related to CPK depletion in homogenates of the entire heart. Regression analysis between enzyme depletion and gross histology demonstrated a correlation coefficient of 0.70. CPK depletion in the center of an ischemic area did not decrease to zero at 24 hours but remained approximately 20% of that in the nonischemic myocardium. Sobel et al. have pointed out that following myocardial infarction a fraction of the myocardial CPK activity is degraded locally, a fraction is released into the blood, and an additional fraction remains in the heart as enzymatically active CPK. Using myocardial CPK depletion 24 hours after coronary occlusion as an index of infarction at that point is based on the data which indicate that each of CPK fractions is constant.

In the present study, there was not a close relationship between ST-segment changes and simultaneously measured regional myocardial blood flow. Although ST segments did not increase unless myocardial blood flow was reduced less than 50%, 39% of the site with less than 50% of controlled flow demonstrated less than 2 mV ST elevation. Thus in the present study significant ST elevation did not occur in many sites which demonstrated both reduced flow and subsequent infarction. The presence of reduced flow to the region which simultaneously did not demonstrate ST elevation indicates that extension of the infarction later in the course of the study would not explain the inconsistent relationship between ST elevation and subsequent myocardial infarction. Since ST-segment elevation was limited to the region in which blood flow was reduced 50% or greater, the presence of ST elevation reliably indicated ischemia, although this index of ischemia did not always predict myocardial infarction.

These results are comparable to the data reported by Smith et al. using anesthetized dogs. These investigators observed that epicardial ST segments were not significantly increased in 34% of the electrode sites positioned in the regions of greatest ischemia. A wide range of ST changes were recorded at a given blood flow measurement. In the study by Smith et al. ST segments did not change between 15 min and 2 hours, although blood flow to the ischemic region increased significantly. In the present study, blood flow increased and ST segment decreased in 12 of 13 dogs between 15 min and 2 hours; however, there was not a close relationship between the increase in blood flow and decrease in ST segments. Timogiannakis et al. also observed a wide range of ST-segment changes for a given degree of myocardial ischemia during partial coronary artery occlusion. Kjekshus et al. reported a close relationship between ST-segment changes 15 min postocclusion and blood flow but the blood flow measurement was made 24 hours later rather than simultaneously.

Previous studies from this laboratory have examined the relationship between regional myocardial blood flow measured at various intervals after coronary occlusion and extent of subsequent myocardial infarction. The entire left ventricle was sectioned into transverse rings, circumferential regions, and then into transmural layers. Blood flow and the extent of histologic infarction were then determined in each myocardial sample. There was an inverse relationship between blood flow and infarction which was a function of transmural position of the myocardial sample and the intervals after coronary occlusion that blood flow was determined. In the present study, the extent of infarction in the transmural specimen at each electrode site was related to myocardial blood flow in the circumferential region from which the specimen was taken. To conform to the technique used in previous studies, the myocardial specimens at the sites of the epicardial electrodes were limited to approximately 0.5 g specimens. Blood flow was not determined in these samples since validation of flow to such size segments at low flow states has not been established. Blood flow was measured in the larger myocardial segment from which the specimen was taken. Regression analyses between blood flow and infarction resulted in correlation coefficient of r = 0.83 at 15 min and 0.89 at 2 hours. Although there was significant scatter in data points, the correlation between blood flow to the region of the transmural specimen and extent of infarction in the specimen was considerably better than that between ST segments and infarction. It is likely that a certain degree of variability resulted from failure to account for transmural differences in the relationship between blood flow and infarction and failure to measure blood flow and infarction in the same sample. The major objective of this study was to examine the relationship between epicardial ST changes and subjacent infarction using previously reported techniques, but using the extent of histologic necrosis rather than CPK depletion.

The precise region of tissue that influences the electromograms recorded from epicardial electrodes has not been established. To conform to previous studies, the extent of myocardial infarction was determined in 0.5 g transmural core tissue specimens taken from the region beneath each electrode. It is unlikely that sampling a smaller region would have improved the relationship; ST segments did not increase at many electrode sites overlying tissue which were almost completely infarcted. Since blood flow was measured in considerably larger samples from the electrode sites and correlated reasonably well with the extent of infarction in the smaller tissue sample, the extent of infarction in the large and small specimens was probably comparable and increasing the size of the samples would not have improved the relationship between infarction and ST changes.

There is considerable interest in the application of interventions designed to limit the extent of myocardial injury resulting from acute myocardial ischemia. Many of the currently advanced interventions have been evaluated in ex-
Experimental animals models in which epicardial ST-segment changes have been used as indices of subsequent myocardial infarctions. However, a number of studies have indicated limitations of this technique in providing quantitative data. In the present study, epicardial ST-segment elevation following permanent coronary occlusion indicated underlying ischemia but did not necessarily predict subsequent infarction. There was not a close qualitative or quantitative relationship between ST-segment changes and histologic myocardial infarction or simultaneously measured regional myocardial blood flow. Changes in ST segments between 15 min and 2 hours were not closely related to changes in blood flow to the ischemic region.

Acknowledgments

We acknowledge the following persons who rendered valuable assistance in carrying out this study: Dr. Joseph C. Greenfield, Jr. for his continuing support; Dr. Philip A. McHale for assistance with the statistical analysis; Dr. Judith C. Rembert for assistance with radioisotope measurements; Mr. Kirby Cooper, Mr. Eric Fields, and Mr. Joe Gates for expert technical assistance; Mr. Michael Taylor and his staff of the Durham Veterans Administration Hospital Animal Care Facility; Mr. Donald G. Powell and Ms. Linda Kohl of the Durham Veterans Administration Hospital Medical Media Production Service; and Mrs. Rosa B. Ethridge and Ms. Sandra G. Rexrode for secretarial assistance.

References

Relationship between epicardial ST-segment elevation, regional myocardial blood flow, and extent of myocardial infarction in awake dogs.

R G Irvin and F R Cobb

Circulation. 1977;55:825-832
doi: 10.1161/01.CIR.55.6.825

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1977 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/55/6/825

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/