Changes in Myocardial Blood Flow and S-T Segment Elevation following Coronary Artery Occlusion in Dogs

By Howard J. Smith, Bramah N. Singh, Robin M. Norris, Murray B. John, and Peter J. Hurley

ABSTRACT

The relationship between regional blood flow and epicardial S-T segment elevation was studied in 26 open-chest anesthetized dogs with left anterior coronary artery ligations. Changes in myocardial blood flow, measured with 15 ± 5 μ (diameter) microspheres labeled with 14Ce, 85Sr, and 169Yb, were correlated with summated S-T segment elevations 15 minutes, 1 hour, and 2 hours after coronary artery occlusion. In normal areas, myocardial blood flow was 113 ± 5 ml/min 100 g⁻¹ and summated S-T segment elevation was 0.3 ± 0.2 mv. Fifteen minutes after coronary artery occlusion in 26 dogs, S-T segment elevation was 5.7 ± 0.7 mv over the center of the infarct and myocardial blood flow was 10 ± 1 ml/min 100 g⁻¹ and S-T segment elevation was 3.1 ± 0.1 mv. One third of the areas with a myocardial blood flow of 10 ml/min 100 g⁻¹ or less had no S-T segment elevation. In the center and border zones of the infarct in 9 dogs, myocardial blood flow increased from 11 ± 2 and 67 ± 8 ml/min 100 g⁻¹ 15 minutes after occlusion to 20 ± 4 and 84 ± 12 ml/min 100 g⁻¹, respectively, 2 hours after coronary artery occlusion. These increases were not associated with a significant reduction in summated S-T segment elevation. The results do not suggest a simple quantitative relationship between epicardial S-T segment elevation and myocardial blood flow following acute coronary artery occlusion.

KEY WORDS myocardial perfusion heart ischemia infarct size radioactive microspheres epicardial electrocardiogram

Myocardial infarction is a dynamic process in which cell necrosis and cell death may be preceded by a phase of reversible ischemia (1). The recent demonstration that the size of experimental infarcts may be altered favorably by therapeutic interventions (2-5), which presumably change the rate at which ischemia progresses, has provided an impetus to define the metabolic and hemodynamic changes which occur following coronary artery occlusion.

The present study was designed to measure the changes in myocardial blood flow over a period of 2 hours following occlusion of the left anterior descending coronary artery in open-chest anesthetized dogs. The degree of S-T segment elevation has recently been used widely (2-5) as an index of ischemic injury, so it was of particular interest to examine the relationship between epicardial S-T segment elevation and blood flow in areas of the myocardium immediately beneath the recording electrodes. Myocardial blood flow was measured using radioactive microspheres (6-8), since this technique allows fine resolution of regional variations in myocardial perfusion (9, 10).

Methods

Twenty-six mongrel dogs (18-30 kg) were given trifluromazine (Siquil, Squibb; 2.7 mg/kg) and anesthetized with sodium pentobarbital (25-30 mg/kg, iv). Anesthesia was maintained with a constant infusion of sodium pentobarbital (5 mg/kg hour⁻¹, iv) administered by a Harvard constant-infusion pump. Positive-pressure breathing was established through a cuffed endotracheal tube with 30% O₂ in room air. Adjustments were made initially to keep the pH, oxygen tension (Po₂), and carbon dioxide tension (Pco₂) of the arterial blood gases within the following ranges: pH 7.35 to 7.45, Po₂ 95 to 115 mm Hg, and Pco₂ 25 to 35 mm Hg. No changes were made in these parameters after coronary artery ligation. Body temperature was monitored with a rectal thermistor probe and kept between 36.5°C and 38.5°C with external heating.

The heart was exposed through a left lateral thoracotomy in the fifth intercostal space. A silk thread was placed around the left anterior descending coronary artery approximately 2 cm from its origin and below its first major diagonal branch, and catheters were placed in the main pulmonary artery, the left atrium, the external jugular vein, and the femoral artery. Left atrial and arterial pressures were measured with Statham P23Db...
transducers and displayed on a Sanborn 350 recorder; the zero reference point was the middle of the left ventricular cavity. Normal saline (100-150 ml) was infused initially to elevate left atrial pressure to 5 mm Hg, and the infusion was continued at 50 ml/hour throughout the experiment. Cardiac output was measured by the dye-dilution technique; 2.5 mg of indocyanine green in 1.0 ml was injected into the main pulmonary artery and followed by a saline flush; blood was withdrawn from the femoral artery by a Harvard pump through a Gilford densitometer.

Epicardial electrocardiograms were recorded after the manner of Maroko et al. (2) with three modifications: a steel-ball electrode (11) was used, measurements were made at predetermined epicardial positions, and the S-T segment was determined 0.06 seconds after the end of the S wave (12). The 14 electrode positions were situated over the anterior surface of the left ventricle and were readily identifiable by their relationship to epicardial blood vessels (Fig. 1). Four positions (normal zone) were above the ligature around the left anterior descending coronary artery, 4 were at the level of the ligature bordering the ischemic area (border zone), and 6 were over the center of the ischemic area (center zone). Within each zone, the center of each position was about 1 cm from that of its neighbor and was marked with light green dye (Light Green SF Yellowish, Allied Chemicals). This histochemical stain, which binds to collagen fibers, did not wash off or alter the epicardial electrocardiogram and lasted 30-45 minutes before fading. The electrode positions corresponded to the tissue samples subsequently taken for measurement of myocardial blood flow, so that the S-T segment changes after coronary artery occlusion could be correlated with the blood flow of each sample.

Myocardial blood flow was measured on three occasions in each dog using carbonized plastic microspheres 15 ± 5μ in diameter, labeled with 144Ce, 85Sr, or 169Yb—gamma-emitting nuclides. Arterial blood (reference blood sample) was withdrawn from the femoral artery at 7 ml/min for 3 minutes by a Harvard pump; withdrawal was started 5-10 seconds before the injection of microspheres was begun. The tip of the catheter was left in the external iliac artery to ensure cross-sectional sampling of arterial blood flow (13). The order of injection of microspheres was varied, but usually 144Ce or 85Sr was followed by 169Yb.

After the final injection of microspheres, the heart was removed and the left ventricle was opened along the obtuse margin. The anterior papillary muscle and the interventricular septum were cut flush with the endocardium of the anterior wall, which was then cut into 14 full-thickness pieces, each approximately 1 cm². These pieces were taken from areas directly corresponding to one of the epicardial electrocardiogram positions (Fig. 1). In the case of the border zone, the pieces of myocardium included the line of demarcation between cyanotic and noncyanotic myocardium which had been present during life. Two further samples were excised from the posterior left ventricle wall (subsequently termed the control zone) remote from the area supplied by the ligated artery. All 16 pieces were divided into endocardial and epicardial halves to give a total of 32 myocardial samples, which were placed in weighed plastic counting tubes. The weights of the samples were in the range of 0.4 to 1.4 g. The radioactivity in the myocardial samples and in 5-ml aliquots of each blood sample was measured in a Packard Autogamma well-type scintillation counter using an integral counting technique (see Appendix). From the radioactivity counts, myocardial blood flow (computed as ml/min 100 g⁻¹) for each area, mean myocardial blood flow for each zone, and the ratios of endocardium perfusion to epicardium perfusion (En-Ep ratios) were calculated. Variations in regional perfusion could then be correlated with the epicardial S-T segment shift over each area of the ischemic myocardium.

Schematic diagram of the experimental preparation used to study the regional distribution of myocardial blood flow and its relationship to epicardial S-T segment elevation following left anterior descending coronary artery occlusion. The location of the numbered sites from which electrocardiographic records and myocardial tissue samples were taken are shown in relation to the level of arterial ligation. The stippled area represents the ischemic zone of the myocardium (see text). L.A. = left atrium, P.A. = pulmonary artery, R.V. = right ventricle, L.V. = left ventricle, Ao. = aorta, L.A.D. = left anterior descending coronary artery, L.C.F. = left circumflex coronary artery, and L.A. Press. = left atrial pressure.
MYOCARDIAL BLOOD FLOW

t-test and the correlation of linear regression. All results are expressed as means ± SE.

Results

HEMODYNAMIC CHANGES

After coronary artery occlusion, there was no significant change in heart rate or mean arterial blood pressure. Mean left atrial pressure rose from 6.4 ± 0.5 mm Hg before the coronary artery ligation to 7.6 ± 0.8 mm Hg 15 minutes after occlusion (P < 0.05), but there was no significant change thereafter. The cardiac index was 123.6 ± 11.9 ml/kg min⁻¹ before coronary artery occlusion; 15 minutes after coronary artery occlusion, it was 103 ± 10.1 ml/kg min⁻¹ (P < 0.05) and it continued to decline progressively to reach 60% of the control value at 2 hours (P < 0.01).

MYOCARDIAL BLOOD FLOW

Fifteen minutes after left anterior descending coronary artery occlusion, the pattern of myocardial blood flow in the 14 areas of the anterior left ventricular myocardium was similar in all dogs (N = 26); the data are summarized in Table 1. There was no significant difference between myocardial blood flow in the nonischemic myocardium anteriorly (areas 1-4, normal zone) and that in the posterior part of the left ventricle in the territory of the left circumflex artery (control zone). The ratio of left anterior descending coronary artery flow to left circumflex artery flow was 0.93 for the normal zone. For the border zone (areas 6-8) this ratio was 0.52 (P < 0.01), and for the center of the infarct (areas 10-14) it was 0.08 (P < 0.001). Electrode positions 5 and 9 (see Fig. 1), contiguous with the interventricular groove, had somewhat higher myocardial blood flows than did the areas immediately adjacent to them (positions 6 and 10, P < 0.01). The En-Ep flow ratios were also greater in these areas, suggesting that their blood supply included a significant contribution from anastomotic vessels from the interventricular septum. For this reason, the flows in positions 5 and 9 were not included in the calculation of the mean myocardial blood flow for the border and center zones as presented in Table 1.

The temporal changes in regional myocardial blood flow in nine dogs are illustrated in Figure 2A. One hour after left anterior descending coronary artery occlusion, the mean myocardial blood flow in the normal zone fell to 108 ± 7 ml/min 100 g⁻¹ from a value of 129 ± 16 ml/min 100 g⁻¹ 15 minutes after left anterior descending coronary artery occlusion.
after coronary artery occlusion ($P < 0.01$), but by 2 hours it had increased to $143 \pm 14$ ml/min/100 g$^{-1}$ ($P < 0.05$). In contrast, myocardial blood flow in the border and center zones increased progressively over 2 hours. In the border zone, myocardial blood flow was $67 \pm 8$ ml/min/100 g$^{-1}$ 15 minutes after coronary artery occlusion; it rose to $84 \pm 12$ ml/min/100 g$^{-1}$ at 2 hours ($P < 0.05$). The myocardial blood flow in the center of the infarct was $11 \pm 2$ ml/min/100 g$^{-1}$ 15 minutes after coronary artery occlusion but increased to $20 \pm 4$ ml/min/100 g$^{-1}$ at 2 hours.

**Endocardial versus Epicardial Flow**

The regional distribution of myocardial blood flow and its relative distribution to the endocardial and epicardial layers of the ventricular wall are shown in Table 1. The En-Ep ratios were highest in the normal and control zones, lowest in the center zone, and at an intermediate value in the border zone. The En-Ep ratios at positions 5 and 9, which were adjacent to the interventricular groove, were much higher than those at their immediate neighbors. Within the border zone, the ratio at position 6 was also somewhat greater than that at position 8 ($P < 0.05$). The reason for this difference is not clear, but, as in the case of positions 5 and 9, it may have been due to the presence of deep anastomoses with vessels in the interventricular septum.

The overall changes in the En-Ep ratios with time (nine dogs) are shown in Figure 2B. There was no significant change in these ratios throughout the period of observation.

**Relationship Between S-T Segment Elevation and Myocardial Blood Flow**

The relevant data 15 minutes after coronary artery occlusion in 26 dogs are summarized in Table 1. The most pronounced S-T segment alterations were found in the center zone, although changes 15 minutes after left anterior descending coronary artery occlusion were also significant in the border zone ($P < 0.01$). Representative records of epicardial electrograms from a typical experiment are reproduced in Figure 3 which shows significant S-T segment elevations without an important lengthening of the duration of the QRS complex over areas of the myocardium rendered ischemic by coronary artery occlusion. In the normal zone, the S-T segment elevation was invariably less than 2 mv (Table 1). However, when S-T segment changes for individual electrode positions were matched with their respective blood flows, a severe reduction in perfusion was not always found to accompany a significant S-T segment elevation. Figure 4 shows the overall relationship between myocardial blood flow and S-T segment elevation 15 minutes after occlusion measured in 10 myocardial samples from the border and center zones of each of 26 dogs (260 samples). It is evident that, although there was wide variation in the levels of S-T segment elevation for each range of myocardial blood flow, a weak but highly significant negative correlation ($r = -0.25, P < 0.001$) was found between myocardial blood flow and S-T segment elevation. S-T segment elevation measured over areas with myocardial blood flows of 0-9 ml/min/100 g$^{-1}$ varied from 0 to 30 mv (mean $5.04 \pm 0.65$ mv) (Fig. 5A). However, 34% of the areas did not show a significant S-T segment elevation (> 2 mv) (Fig. 5B). Within the range of 60 to 79 ml/min/100 g$^{-1}$, S-T segment elevation ranged from 0 to 11 mv (mean $2.0 \pm 0.49$ mv) and 53% of the areas did not have a significant S-T segment elevation (Fig. 5B). The relationship between S-T segment elevation and endocardial myocardial blood flow was similar to that with total flow (Fig. 5A). For clarity of presentation of the temporal changes in S-T segment elevation and its relationship with myocardial blood flow, the myocardial samples from the border and center zones (90 samples from nine dogs) were divided into three groups (Table 2); normal or slightly reduced flow (>60 ml/min/100 g$^{-1}$), moderately reduced flow (30-59 ml/min/100 g$^{-1}$), and low flow (<30 ml/min/100 g$^{-1}$). Although the myocardial blood flow of an individual sample of tissue varied over the 2-hour period, this analysis, which allowed interpretation independent of varying levels of myocardial blood flow, showed that there was no significant change in S-T segment elevation in any of the three ranges of flow over the 2-hour period of observation.

**Discussion**

Our results show that following ligation of the left anterior descending coronary artery in open-chest anesthetized dogs there was a severe (to 10 ml/min/100 g$^{-1}$) and relatively uniform reduction in myocardial blood flow in the center of the developing infarct. A reduction in myocardial blood flow of intermediate severity (to 60 ml/min/100 g$^{-1}$) was found in the border zone. It must be emphasized, however, that our sampling technique did not attempt to distinguish cyanotic and non-cyanotic areas at the periphery of the infarct. Previous evidence, including that from experiments using diffusible indicators, also supports the concept of an ischemic zone with a reduction in myocardial blood flow of intermediate severity (10, 16-19). Variations in tissue transit times for diffusible indicators used in the aforementioned experiments may result in considerable errors in the area.
Changes in epicardial S-T segment elevation and myocardial blood flow (MBF, ml/min 100 g\(^{-1}\)) following proximal ligation of the left anterior descending coronary artery in an open-chest anesthetized dog. A: Electrograms from different areas of the left ventricular surface before ligation. Only T wave inversions were present. B: 15 minutes after ligation, S-T segment elevations developed in the border and center zones of the infarcting myocardium. C: These changes seen 15 minutes after ligation persisted 2 hours after ligation. Note that there was no appreciable prolongation of the duration of the QRS complex in either the center or the border zone after left anterior descending coronary artery occlusion.

measurements of low tissue flows (17, 19, 20), however, and fine resolution of areas of differing perfusion is best delineated by the use of radioactive microspheres, as has been done in our own study and in those of Becker et al. (10, 21).

Using a different tissue sampling technique from our own, Becker and his colleagues (10) found concentric areas of increasing flow surrounding the center zone but within the area of cyanosis. Myocardial blood flow increased from less than 20 ml/min 100 g\(^{-1}\) at all of the margins to 120–140 ml/min 100 g\(^{-1}\) (hyperemic flow) immediately outside the cyanotic area. In their preparation, the entire free wall of the left ventricle was divided into 100–130 pieces weighing 0.3–3.0 g; in the present experiments, part only of the free wall was divided into 28 smaller pieces weighing 0.4–1.4 g. Moreover, only one border zone, that at the anterosuperior border, was sampled in our more standardized preparation, whereas the lateral and apical borders were also sampled by Becker and his associates. Thus, both sets of experiments appear to support the concept of a zone of intermediate reduction in myocardial blood flow surrounding the developing infarct, but the possibility must be considered that in both preparations the border zone at least in part may be artifactual, representing merely an arithmetic mean of areas having normal and severely impaired tissue perfusion. A clearer delineation of the border zone may nevertheless be of practical importance, in that therapeutic interventions which have beneficial effects
on ischemic injury are likely to produce greater changes in areas of myocardium with a moderate rather than a severe reduction in blood flow.

In our experiments, a disproportionate reduction in flow to the endocardial half of the left ventricular wall was found during ischemia; the changes were most striking in the center of the infarct. The ratios of endocardial flow to epicardial flow in the present experiments are similar to those reported by Becker et al. (21) and Kjekshus (22), who used microspheres 15 ± 5 µ in diameter. The vulnerability of endocardial perfusion during ischemia has been emphasized by previous studies with microspheres (10, 20, 22) as well as by those with diffusible radioactive indicators (23). This finding is particularly relevant in the study of the mode of action of antianginal drugs, since both nitroglycerin and propranolol have been shown to improve endocardial flow preferentially in ischemia without altering total myocardial blood flow (21).

A particularly significant feature of our results is the lack of a precise correlation between epicardial S-T segment elevation and regional myocardial perfusion. In recent years, the height of the S-T segment in epicardial leads of the electrocardiogram has been found to be an index of myocardial injury in patients (24) and experimental animals (2). Maroko et al. (2) have shown that the magnitude of epicardial S-T segment elevation 15 minutes after experimental coronary artery occlusion reliably predicts the severity of myocardial damage within the ischemic area (positions 5-14, nine dogs) is compared with the myocardial blood flow at each position 15 minutes, 1 hour, and 2 hours after left anterior descending coronary artery occlusion. All results are expressed as means ± SE.

TABLE 2

<table>
<thead>
<tr>
<th>MBF (ml/min/100g)</th>
<th>S-T segment elevation (mv)</th>
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</thead>
<tbody>
<tr>
<td>100 g⁻¹</td>
<td>15 minutes</td>
</tr>
<tr>
<td>0-29</td>
<td>6.1 ± 0.9</td>
</tr>
<tr>
<td>30-59</td>
<td>2.5 ± 1.1</td>
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<tr>
<td>&gt;60</td>
<td>2.2 ± 0.6</td>
</tr>
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The S-T segment elevation recorded within the ischemic area (positions 5-14, nine dogs) is compared with the myocardial blood flow at each position 15 minutes, 1 hour, and 2 hours after left anterior descending coronary artery occlusion. All results are expressed as means ± SE.
24 hours later, as evidenced by the degree of creatine phosphokinase depletion and histological disorganization. They did not correlate these changes in myocardial enzyme histochemistry or ultrastructure with variations in regional perfusion in the first 2 hours after coronary artery occlusion. Using microspheres, Kjekshus et al. (25) examined the relationship between S-T segment changes 15 minutes after coronary artery occlusion and creatine phosphokinase depletion as well as regional myocardial blood flow 24 hours later. They found that, although local creatine phosphokinase depletion was directly related to the reduction in blood flow both in deep and superficial layers of the left ventricle, S-T segment elevation 15 minutes after coronary artery occlusion was not. They found a linear relationship between acute S-T segment elevation and the reduction in subepicardial blood flow; for the subendocardial blood flow, however, disproportionately less S-T segment elevation for a comparable diminution in perfusion was found. Such a difference was not apparent in our own studies in which measurements of regional myocardial blood flow were made for 2 hours following coronary artery occlusion. Although the highest levels of S-T segment elevation in our experiments occurred within the areas of myocardium with minimum levels of myocardial blood flow, there was no simple quantitative relationship between the degree of S-T segment elevation and the extent of fall in myocardial blood flow. For example, 34% of the areas of the myocardium with a blood flow less than 10 ml/min 100 g⁻¹ had no epicardial S-T segment elevation. Moreover, despite significant increases in myocardial blood flow 2 hours after coronary artery occlusion in both the border and center zones, as reported also by Rees and Redding (26), a reduction in the magnitude of S-T segment elevation did not occur in our study. Paradoxically, Cohen and Kirk (27) recently reported that local S-T segment elevation may be minimized by a maneuver which increases both the area and the depth of ischemia. In particular, they found that S-T segment elevation caused by the ligation of a branch of the left anterior descending coronary artery was often diminished following proximal occlusion of that artery. This phenomenon was related to the presence of an identifiable boundary to the ischemic area, across which injury currents flow from normal to ischemic myocardium during electrical diastole. Since the magnitude of such currents is likely to diminish as the distance from the transition zone increases, it is possible that S-T segment elevation over the center of the infarct may be minimum or even absent as was the case in 30% of the epicardial sites in our experiments. Alternatively, a weak correlation between local S-T segment elevation and reduced myocardial blood flow following coronary artery occlusion may result from the development of focal conduction block with prolongation of the QRS complex when S-T segment elevation disappears. However, this explanation for the expected relationship between S-T segment elevation and myocardial blood flow reported in the present paper is unlikely. In a parallel study using an identical experimental model, prolongation of the QRS complex exceeding 0.06 seconds was found in only 2.5% of the electrode sites from which epicardial electrograms were recorded 15 minutes after left anterior descending coronary artery occlusion in 16 animals (Heng, Singh, and Norris, unpublished observations).

Epicardial S-T segment elevation does not therefore depend solely on a reduction in myocardial blood flow, and it is possible that the critical determinant of S-T segment alterations is the local balance in oxygen demand and supply which affects the functional integrity of the myocardial membrane. It is known that myocardial blood flow has to be reduced to less than 50% of normal before significant S-T segment elevation occurs (28). Furthermore, when flow is very low, as is found in the center of the infarct, no subsequent increases in flow due to collateral perfusion are likely to change the ultimate fate of myocardial cells as reflected by S-T segment elevation or tissue necrosis. The data presented in the present paper suggest that, although S-T segment elevation implies ischemic injury, the lack of S-T segment change over an area of the myocardium does not permit the conclusion that ischemia is absent; moreover, an increase in S-T segment elevation does not necessarily mean that ischemia has become more severe. Similarly, a reduction in S-T segment elevation may result either from an improvement in the extent of ischemic injury or alternatively from progression of an ischemic lesion to necrosis and cell death with loss of electrophysiological functions of the myocardial membrane.

Regional metabolic studies have also emphasized the difficulties in correlating metabolic indexes of ischemia with variations in S-T segment elevation. For example, Karlsson et al. (29) have shown that, although the areas of the ischemic myocardium which have abnormally high concentrations of lactate also show S-T segment elevation, a precise quantitative relationship does not exist between the severity of S-T segment change and

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the levels of lactate in the ischemic myocardium. These results from metabolic studies as well as those from our measurements of regional myocardial blood flow during ischemia are consistent with the histological observations of Reiner et al. (30) who have shown that, in the case of S-T segment changes, the distribution of myocardial necrosis following coronary artery occlusion is seldom uniform. The clinical significance of these results is uncertain. The data do not negate the value of S-T segment changes as a guide to myocardial ischemic injury, but they do suggest that the relationship between the height of S-T segment elevation and the severity of myocardial ischemia is complex and merits critical reappraisal.

Appendix

INTEGRAL COUNTING TECHNIQUE FOR THREE RADIOACTIVE ISOPTES

The different gamma spectrums of the three nuclides used enabled calculation of the individual counts when samples containing all three were measured together. The activity of a pure sample of each nuclide was measured separately in the spectrometer over three different energy ranges (90 to 200, 90 to 380, and 90 to 600 keV). The energy output as a percent of the 90-600-keV value for each nuclide over the different ranges was then found (Table 3).

Activity in each myocardial sample was then measured over each of the energy ranges giving three simultaneous equations:

\[ \text{Counts}_{90-600} = X + Y + Z, \]  
\[ \text{Counts}_{90-380} = X + 0.93Y + 0.46Z, \]  
\[ \text{Counts}_{90-200} = X + 0.43Y + 0.27Z, \]

where \( X \) = counts of \(^{14} \text{Ce} \) in the 90-600-keV range, \( Y \) = counts of \(^{185} \text{Yb} \) in the 90-600-keV range, and \( Z \) = counts of \(^{85} \text{Sr} \) in the 90-600-keV range.

REFERENCE BLOOD SAMPLE

The arterial blood samples were hemolyzed with digitonin and agitated; a 5-ml aliquot was then taken. Measurement of the radioactivity of the hemolyzed 5-ml sample was multiplied by this factor to ensure that all of the measurements were expressed as if they had been made on 1-ml aliquots. The correction factors were: \(^{14} \text{Ce} 1.53, \(^{185} \text{Yb} 1.21, \) and \(^{85} \text{Sr} 1.44. \)

Acknowledgment

The excellent technical assistance of Miss Heather Nisbet and Mr. M. E. Oelner is gratefully acknowledged. We also thank Dr. E. A. Harris and Dr. R. M. L. Whitlock for helpful advice.

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Changes in myocardial blood flow and S-T segment elevation following coronary artery occlusion in dogs.
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Circ Res. 1975;36:697-705
doi: 10.1161/01.RES.36.6.697

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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