Effects of Exercise- and Pacing-Induced Tachycardia on Coronary Collateral Flow in the Awake Dog

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SUMMARY Collateral blood flow was studied in chronically instrumented awake dogs 11-12 weeks after implantation of ameroid constrictors on the right and left circumflex coronary arteries. Using 7-10 μm radionuclide-labeled microspheres, transmural myocardial blood flow was measured during resting conditions and at similar heart rates during ventricular pacing and treadmill exercise. The absence of significant myocardial fibrosis was verified histologically. Control transmural flow was distributed normally in all dogs. In five dogs a marked maldistribution of flow occurred in the collateral region during pacing and exercise. During pacing, flow to the epicardial layers increased while flow to the endocardial layers was unchanged, resulting in a marked endocardial perfusion deficit. During exercise, flow increased substantially to all transmural layers, but the endocardial perfusion deficit remained. Mean transmural blood flow increased similarly in the collateral and noncollateral regions during pacing; however, during exercise, mean flow in the collateral region was significantly lower than in the noncollateral region. These data demonstrate that the collateral vessels became flow limiting and functioned inadequately during tachycardia produced by pacing and exercise; i.e., a marked perfusion deficit occurred in the endocardial layer. Blood flow to all layers in the collateral-dependent region was higher during exercise than during pacing, possibly due to exercise-induced vasodilation of the collateral channels. Circ Res 46: 214-220, 1980

AS MYOCARDIAL viability may depend on collateral channels in advanced occlusive coronary artery disease, a complete understanding of transmural myocardial perfusion in coronary artery disease must include a detailed knowledge of the determinants of flow through collateral vessels. Considerable effort has been devoted recently to developing a large animal model with a collateralized coronary circulation. Chronic gradual coronary artery occlusions, produced by hydroscopic ameroid constrictors in the dog, result in rapid growth of extensive intercoronary collateral channels which may prevent myocardial infarction. Several investigators have shown that these channels perfuse the myocardium adequately to meet metabolic needs during resting conditions (Becker and Pitt, 1971; Elliot et al., 1971; Schaper, 1971; Schaper et al., 1973). However, with the increased myocardial flow requirements of tachycardia or pharmacologically induced coronary vasodilation, these collateral channels may become flow limiting, resulting in a relative perfusion deficit of the endocardial layers (Schaper, 1971; Schaper et al., 1973; Flameng et al., 1975a, 1975b; Cox et al., 1976; Pasyk et al., 1976). Lambert et al. (1977) demonstrated that a well-developed coronary collateral circulation may perfuse the myocardium adequately during moderate treadmill exercise. However, there is a paucity of data defining in vivo the factors influencing collateral flow in response to an increase in myocardial metabolic demands.

Our initial goal was to develop an awake animal model having moderately well-developed coronary vessels with the following requirements: (1) no evidence of myocardial fibrosis or infarction could be demonstrated, (2) the collateral vessels could perfuse the myocardium adequately to prevent myocardial ischemia at rest, and (3) the collateral vessels would become flow limiting during conditions requiring increased flow, resulting in a perfusion deficit. This animal model was used to test whether or not a difference in coronary collateral flow was demonstrable during the tachycardia induced by ventricular pacing or by exercise. These data provide insight concerning the possibility that coronary collateral vessels can dilate in response to the stimulus of exercise.

Methods

Adult, mongrel dogs of both sexes (weighing 20-30 kg) were selected for use according to their
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ability to run on a motor-driven treadmill. Those that would not run were culled. The dogs were anesthetized with sodium thiamylal (30-40 mg/kg, iv), intubated, and maintained with a modified Emerson respirator. A thoracotomy was performed in the fourth left intercostal space, and the heart was exposed. Dogs were studied further if the left circumflex coronary artery was dominant and the left anterior descending coronary artery did not have large anterolateral branches in the proximal 2-cm segment. Twenty-one dogs met these criteria. Small branches of the left anterior descending artery segment were ligated. The proximal 1.5 cm of the left circumflex and the right coronary arteries were exposed. Hydroscopic ameroid constrictors were chosen to fit snugly around both the right (2.0- or 2.5-mm inner diameter) and left circumflex (2.77- or 3.0-mm inner diameter) coronary arteries proximal to any branches. All dogs survived the initial operation, but four died postoperatively at 12, 14, 17, and 21 days. In two of these four animals, significant myocardial fibrosis was found in the distribution of the left circumflex artery.

The surviving dogs were maintained on a normal laboratory diet and kept in open runs. Nine weeks after the initial surgery, they underwent a second left thoracotomy using a similar anesthetic protocol. Polyvinyl chloride catheters (2.5 mm o.d.), filled with heparin, were inserted into the left atrial cavity via the left atrial appendage, the left ventricle via a stab wound in the apex, and the aorta via the left internal mammary artery. An epicardial bipolar pacing electrode was secured to the right ventricle in the region of the right ventricular outflow tract. The ends of the catheters and electrode were tunneled to a subcutaneous pouch at the base of the neck. Four animals died perioperatively. An interval of 10-14 days was allowed for full recovery before the dogs were studied. At this time the animals were free of infection and anemia, hematocrits ranged from 36 to 45 (mean 39 ± 3), and they showed no evidence of ill health.

On the day of study, the catheters and leads were exteriorized from the subcutaneous pouch through a 1-cm skin incision (locally anesthetized with 2% lidocaine). Left atrial, aortic, and left ventricular pressure catheters were attached to Statham P23Db transducers. The zero pressure reference was chosen at the midstest level. Pressures were recorded continuously throughout the study on an eight-channel, direct-writing oscillograph.

Randomly selected radioactive microspheres (7-10 μm) labeled with one of the γ-emitting nuclides (125I, 110mCe, 51Cr, 85Sr, or 46Sc) were used to determine myocardial blood flow (Rudolph and Heymann, 1967; Domenech et al., 1969). The stock solution for each microsphere was obtained as 1.0 mCi in 10 ml of 10% dextran, then it was diluted so that 1 ml, the volume injected, contained approximately three million microspheres; Tween 80 was not added. In our laboratory, this technique produces no measurable changes in hemodynamic indices. The microspheres were mixed by alternate agitation for at least 15 minutes in an ultrasonic bath and with a Vortex agitator, and then injected into the left atrium through high pressure tubing and flushed with 5 ml room-temperature saline. Simultaneously with each injection, reference samples of arterial blood were withdrawn with a Harvard withdrawal pump through the aortic catheter at an average rate of 15.5 ml/min for approximately 90 seconds. Transmural distribution of flow was measured first during resting conditions, i.e., the dogs either lying quietly on their right sides or sitting comfortably beside the treadmill. The dogs were then run for 5 minutes at a sufficient level of exercise (mean 6 mph, 12% grade) to increase the heart rate to approximately 200 beats/min. The animals rested for at least 10 minutes and ran again for 5 minutes at the same speed and grade. When steady state conditions were achieved, a second microsphere injection was given with the dogs continuing to exercise throughout the collection of the reference samples. The animals rested for at least 80 minutes. Ventricular pacing, approximating the rate achieved during exercise, was carried out using a Grass model S88 physiological stimulator delivering 3-msec square wave pulses 25% above threshold through a stimulus isolation unit. Steady state conditions were obtained (5-minute minimum) before the third microsphere injection. Pacing was continued for 90 seconds after the injection.

At completion of the exercise and pacing studies, the dogs were anesthetized with sodium thiamylal and killed with a lethal dose of potassium chloride. After removal of the heart, the right and left main coronary arteries were cannulated with polyvinyl chloride catheters, secured with silk ties, and contrast material was injected under pressure (approximately 120 mm Hg) into each of the coronary arteries. The arteries were examined both during fluoroscopy and with standard roentgenograms to verify complete closure of the ameroid constrictors. In addition, microscopic examination of five of the ameroid constrictors revealed that the arterial lumina were occluded.

To facilitate sectioning, the heart was placed in 10% buffered formalin for 3-5 days. The atrial tissue, right ventricle, pericardium, aorta, and large surface vessels were dissected and removed from the left ventricle. The left ventricle was sectioned into four transverse rings from base to apex, as described previously (Cobb et al., 1974). The two middle rings were sectioned into six anatomical regions: interventricular septum, anterior, anterior papillary muscle, lateral free wall, posterior papillary muscle, and posterior. Each region was cut into four transmural layers from epicardium to endocardium; the resulting tissue samples weighed 1-2 g. The γ radioactivity of both the individual tissues and reference blood were measured with a γ spectrometer (Beckman 16776) with the counting win-
dows set to quantify peak energies emitted by each nuclide. The data were processed on an IBM 1130, System 7, using a computer program that corrected the values for background and cross-channel spill-over contamination. Flow per gram of tissue for each sample \( Q_{m} \) was computed using the following relation: 

\[
Q_{m} = Q_{r} \cdot C_{m}/C_{r}
\]

where \( Q_{r} \) is the rate of withdrawal of the reference sample, \( C_{m} \) is the activity (counts) per gram of tissue sample, and \( C_{r} \) is the activity (counts) of the reference sample.

The anterior, anterior papillary muscle, and septal regions formed the noncollateral area supplied by the left anterior descending coronary artery. The posterior papillary region was used as the collateral-dependent area, because it was supplied by collateral vessels. The posterior and lateral free wall were excluded from evaluation because of the likelihood that the blood supply might arise from both collateral and noncollateral vessels. Multiple histological samples were taken from the posterior papillary region to verify the absence of myocardial fibrosis and infarction. The ratio of endocardial to epicardial (endo/epi) blood flow was computed by dividing flow in layer 4 by flow in layer 1.

Heart rate, aortic systolic and diastolic pressures, and left atrial pressure were measured directly from the oscillographic recordings. The duration of diastole (the fraction of time in diastole expressed as a percentage of the total cardiac cycle) was computed from an average of eight consecutive beats on the aortic pressure tracing. Standard statistical analyses with the Student's \( t \)-test for paired or unpaired data were used as indicated throughout to evaluate the data. A \( P \) value < 0.05 was required for statistical significance.

**Results**

Two dogs were excluded from analysis: one in which an adequate exercise study was not obtained and one in which significant fibrosis was found in the collateral region. Myocardial blood flow data were analyzed from 11 of the remaining dogs, none of which had fibrosis or evidence of myocardial infarction. These dogs were separated into two groups according to the transmural blood flow response during pacing and exercise. In group I (five animals) blood flow in the collateral region was markedly maldistributed during pacing and exercise. In group II (six dogs) only a minimal maldistribution was noted.

**Group I**

Under control conditions, the mean blood flow was \( 1.08 \pm 0.07 \) (mean ± SE) ml/min per g and \( 0.96 \pm 0.03 \) ml/min per g in the collateral and noncollateral regions, respectively. The transmural blood flow was distributed in a similar manner, resulting in a mean endo/epi of \( 1.16 \pm 0.06 \) and \( 1.33 \pm 0.06 \) in the two regions (Fig. 1).

With the onset of pacing-induced tachycardia, mean transmural blood flow increased equally in both the collateral and noncollateral regions, \( 1.63 \pm 0.08 \) and \( 1.61 \pm 0.08 \) ml/min per g. However, a

![Figure 1](http://circres.ahajournals.org/Downloaded from)
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marked difference in blood flow distribution between the regions developed. Blood flow in layers 1 and 2 of the collateral area increased by 0.96 ± 0.11 and 1.06 ± 0.15 ml/min per g (P < 0.001), but remained unchanged in layers 3 and 4 (P > 0.25 and 0.60). Thus, the endo/epi decreased to 0.56 ± 0.09. The incremental increase in flow to the endocardial layer in the noncollateral region was slightly, but significantly, greater (P < 0.001) than in the epicardial layer; 0.71 ± 0.09 and 0.52 ± 0.07 ml/min per g, respectively. The increase in flow to layers 1 and 2 of the collateral region was significantly greater (P < 0.003, 0.005) than in the noncollateral region.

During exercise, the mean flow in the collateral area increased to 2.65 ± 0.15 ml/min per g and was significantly less than in the noncollateral region, 3.18 ± 0.10 ml/min per g (P < 0.02). Blood flow in layer 1 of the collateral area increased substantially; however, the incremental increase in flow to the epicardial layer of 1.22 ± 0.12 ml/min per g was significantly greater (P < 0.05) than the increase to the endocardial layer of 0.75 ± 0.19 ml/min per g. Thus, the transmural perfusion remained maldistributed. In the noncollateral region, exercise resulted in a similar incremental increase in blood flow to all layers, so that the flow remained normally distributed.

Hemodynamic data from the group I dogs are presented in the upper panel of Table 1. The average heart rate was 103 ± 12 beats/min during resting conditions. Ventricular pacing increased the rate to 203 ± 11; this was similar to the rate of 200 ± 7 beats/min. Systolic aortic blood pressure was unchanged from control during pacing, but it increased significantly during exercise to 166 ± 10 mm Hg. Diastolic pressure was 84 ± 6 mm Hg during control conditions, increased significantly to 93 ± 3 mm Hg with pacing, and further increased significantly with exercise to 99 ± 4 mm Hg. At rest the duration of diastole was 70 ± 2.0% of the cardiac cycle and decreased significantly to 56 ± 2.0% (P < 0.01) during pacing and 58 ± 2.0% with exercise.

**Group II**

No differences were noted in the transmural blood flow or flow distribution in either the collateral or noncollateral regions during rest (Fig. 2). During pacing-induced tachycardia, flow increased significantly in all layers in both the noncollateral and collateral regions. However, the incremental increase in flow to layer 4 was significantly less in the collateral region, indicating a minimal perfusion abnormality. During exercise, the mean transmural blood flow increased equally in both the collateral and noncollateral regions and results were not significantly different; 2.50 ± 0.20 ml/min per g and 2.38 ± 0.21 ml/min per g, respectively. Flow increased in all layers of both the noncollateral and collateral regions by a similar amount.

The hemodynamic data from group II are presented in the lower panel of Table 1. There were no significant differences between group I and group II in any of these variables during control observations or in response to the interventions of pacing and exercise-induced tachycardia. The duration of diastole was not significantly different from the value for group I during all three conditions (P > 0.6).

**Discussion**

Blood flow into a segment of myocardium perfused via collateral vessels may be regulated by the resistance in (1) the collateral vessels themselves, (2) the arteriolar or precapillary vessels, or (3) a combination of both. To study the responses of the collateral circulation to various stimuli requiring

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**Table 1 Hemodynamic Data**

<table>
<thead>
<tr>
<th></th>
<th>Heart rate (beats/min)</th>
<th>Left atrial pressure (mm Hg)</th>
<th>Systolic</th>
<th>Diastolic</th>
<th>Mean</th>
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<tr>
<td><strong>Group I</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>103 ± 12</td>
<td>5 ± 1.1</td>
<td>124 ± 6</td>
<td>84 ± 6</td>
<td>97 ± 5</td>
</tr>
<tr>
<td>PACE</td>
<td>203 ± 11</td>
<td>7 ± 1.6</td>
<td>122 ± 4</td>
<td>93 ± 6</td>
<td>102 ± 3</td>
</tr>
<tr>
<td>EX</td>
<td>200 ± 7</td>
<td>9 ± 1.4</td>
<td>166 ± 10</td>
<td>99 ± 4</td>
<td>122 ± 5</td>
</tr>
<tr>
<td>△P</td>
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<td>NS</td>
<td>NS</td>
<td>0.03</td>
<td>NS</td>
</tr>
<tr>
<td>△P/P</td>
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<td>0.02</td>
<td>0.002</td>
<td>0.01</td>
<td>0.001</td>
</tr>
<tr>
<td>*P</td>
<td>NS</td>
<td>NS</td>
<td>0.01</td>
<td>0.003</td>
<td>0.003</td>
</tr>
<tr>
<td><strong>Group II</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>88 ± 7</td>
<td>4 ± 1</td>
<td>119 ± 6</td>
<td>78 ± 5</td>
<td>92 ± 5</td>
</tr>
<tr>
<td>PACE</td>
<td>211 ± 8</td>
<td>7 ± 1.5</td>
<td>127 ± 6</td>
<td>93 ± 5</td>
<td>104 ± 5</td>
</tr>
<tr>
<td>EX</td>
<td>202 ± 5</td>
<td>8 ± 2</td>
<td>168 ± 6</td>
<td>104 ± 4</td>
<td>125 ± 3</td>
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<tr>
<td>△P</td>
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<td>NS</td>
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<tr>
<td>△P/P</td>
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<td>0.01</td>
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</tr>
<tr>
<td>*P</td>
<td>NS</td>
<td>NS</td>
<td>0.01</td>
<td>NS</td>
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</table>

Data are presented as mean ± SE for both group I (n = 5) and group II (n = 6) during control (CONT), ventricular pacing (PACE), and treadmill exercise (EX). There were no significant differences between any of the hemodynamic data in the two groups. However, within-group comparison P values are listed in the following manner: CONT vs PACE (△), CONT vs EX (○), and PACE vs EX (*). NS signifies P > 0.05.
increased flow, it is necessary to develop a model in which the collateral vessels provide the primary loci of resistance i.e., they are flow limiting. The model used in this study was similar to that described by Schaper (1971), who placed ameroid constrictors on both the right and left circumflex coronary arteries. Special care was taken to use dogs in which the anatomy of the coronary vessels was similar. In spite of these techniques, a marked variation in collateral flow response was noted.

Of the 21 dogs prepared initially, eight died prior to study, presumably of cardiac causes. In two of the eight dogs and in one additional dog in which studies were completed, significant myocardial infarction and fibrosis were found at necropsy, indicating that the collateral circulation did not develop with sufficient rapidity to prevent cell death during closure of the ameroid constrictor. A marked transmural redistribution of flow, resulting in endocardial underperfusion, occurred during tachycardia in the group I dogs. Thus, the collateral vessels were flow limiting in only five dogs. In the six dogs of group II, no significant perfusion deficit was noted during either pacing or exercise, indicating adequate function of the collateral vessels.

Among the factors defining the magnitude of collateral flow, the maturity of the collateral vessels is of major importance. Schaper et al. (1976) determined the collateral resistance as a function of time following ameroid placement. These investigators noted that the conductance of the collateral circulation increased markedly during the first few weeks after ameroid placement and, at 8 weeks, reached 33% of the conductance present in the native coronary artery prior to occlusion. No further improvement was noted at 18 weeks. Based on these data and their prior studies, these investigators concluded that the coronary collateral vessels had achieved a high degree of maturity and contained significant vascular smooth muscle 10 weeks after ameroid placement (Schaper et al., 1971; Schaper et al., 1976). Our dogs were studied 11 weeks after initial ameroid placement, when a well-developed collateral circulation should have been present. Lambert et al. (1977) found no significant perfusion deficit in any of the dogs studied during moderate treadmill exercise 6–12 months after implantation of an ameroid constrictor on the left circumflex coronary artery. This lack of perfusion deficit may have been due either to the longer period of time in which the collateral circulation matured or to a difference in the model.

The rate at which the ameroid constrictors occlude the coronary artery is another potential variable in the development of the collateral circulation. Recently, in a separate study carried out in our laboratory, Hill et al. (1978) found that, in seven dogs surviving 4 weeks postoperatively, the mean time of complete ameroid closure was 19 ± 1 days. Thus, it seems unlikely that this factor accounted for the marked variation in collateral function noted in the present study.

The marked maldistribution of flow, with failure of the flows to layers 3 and 4 to increase during pacing-induced tachycardia in the group I dogs, indicates that the collateral circulation was not functioning adequately. During pacing, the mean flow to both the collateral- and noncollateral-dependent areas was not significantly different, making it appear at first examination that the collateral circulation was not truly flow limiting. However, for the collateral vessels to be considered as functioning adequately, not only must the total transmural flow be sufficient, but flow must be optimally distributed as well. The most likely explanation for the maldistribution of flow is that a pressure drop, of sufficient...
magnitude to prevent adequate perfusion of the endocardial layers, developed across the collateral vessels. If the diastolic perfusion pressure in the coronary vessels is reduced below a critical value, then perfusion of the endocardial layer is markedly impaired (Buckberg et al., 1972). Although it was not possible to measure pressure distal to the collateral vessels, it seems reasonable to speculate that an increase in the pressure drop across the collateral bed occurred when the flow increased during pacing in the face of the fixed resistance provided by the collateral channels. The resulting fall in diastolic perfusion pressure was responsible for the maldistribution of flow.

Regarding the maldistribution of flow, a point of interest is that flow to layers 1 and 2 of the collateral region was significantly higher than flow to the noncollateral region. Since flow to the endocardial layers of the collateral region did not increase, it is reasonable to conclude that these layers were ischemic and the functional ability impaired. Thus, the increase in flow to the outer layers occurred as a result of the increase in work required to maintain functional integrity of the myocardium in the collateral region. Since segmental wall motion of the collateral region was not measured in these studies, the precise degree to which contraction of the collateral region was affected was not documented. However, the marked increase in flow to the outer two layers is suggestive evidence that some of the function was maintained. It should be noted that maldistribution of flow occurred despite a mean increase in diastolic aortic perfusion pressure of 9 mm Hg.

The findings in the group I dogs are similar to those of previous studies carried out in our laboratory by Ball and Bache (1976), in dogs with a normal coronary circulation. In these experiments, partial restriction of proximal coronary artery inflow during ischemia-induced vasodilation produced a substantial maldistribution of flow with endocardial underperfusion. Therefore, restriction of coronary inflow during vasodilation produced by other mechanisms results in endocardial ischemia.

During pacing the marked decrease in the length of the diastolic period during which the endocardial layer could be perfused might have been an additional factor preventing an increase in flow to the endocardial layer. However, during pacing in the group II dogs, only a minimal perfusion abnormality was noted and the duration of diastole was the same as in the group I animals, indicating this was not a major factor in producing the endocardial underperfusion.

In the group I dogs, exercise produced a substantial incremental increase in flow to all layers of the collateral region, but the marked maldistribution of endocardial underperfusion was unchanged. It is likely that the same pathophysiology of the collateral vessels was operative during exercise as during pacing-induced tachycardia, resulting in a reduced distal perfusion pressure. The maldistribution of flow in the collateral region was similar to that found during the treadmill exercise in dogs studied in our laboratory by Ball and Bache (1976), in which a partial occlusion of the coronary artery restricted flow. Thus, the collateral vasculature can be equated functionally with a single flow-limiting obstruction in a major coronary vessel.

The increase in flow to all layers of the myocardium during exercise, above that produced by pacing alone, is due to one of two mechanisms: (1) the increase in perfusion pressure or (2) vasodilation of the collateral channels. A mean increase of 44 mm Hg in systolic aortic pressure occurred as a result of exercise and augmented coronary flow during systole. Rembert et al. (1978) demonstrated that when coronary flow was limited to the period of systole in an awake animal, the outer two layers of the heart were perfused primarily with only minimal flow to the endocardial layer. Thus, the increase in systolic pressure could not augment flow to the subendocardial layers of the collateral region by increasing systemic coronary flow. A possible mechanism for the increase in flow, secondary to the increase in systolic pressure, is related to the dynamic capacitance of the collateral bed. If the epicardial collateral vessels were sufficiently compliant, a significant amount of blood could be stored in them during systole and then could perfuse the subendocardial layers during the subsequent diastole. The dynamic capacitance of the epicardial collateral bed is unknown. However, some of the epicardial collateral vessels connect to the distal portion of the occluded circumflex artery. The dynamic capacitance of this vessel was measured by Douglas and Greenfield (1970). Using their data, a mean increase in systolic pressure of 25 mm Hg could be responsible for dynamically increasing the volume of the vessel by 0.01 ml/beat. If, as noted above, this blood perfuses the endocardial layer during the subsequent diastole, then with a heart rate of 200 beats/min, the resulting 2 ml of blood stored during systole may be of a sufficient volume to account partially for the increased subendocardial flow during exercise. Since the pressure pulse was not measured distal to the collateral vessel and the actual change in systolic pressure in the circumflex artery is unknown, the calculation of volume change must be considered speculative. This hypothesis was examined further by plotting the increase in systolic pressure with the change in flow to layer 4 during exercise of the group I dogs. A poor correlation ($r = 0.48$) was obtained. Although the increase in systolic pressure during exercise could not be excluded, it probably was not primarily responsible for the augmented flow to the subendocardial layers. The diastolic pressure increased by a mean of 6 mm Hg during exercise. During pacing, an increase in diastolic pressure of 9 mm Hg did not result in increased flow to the subendocardial layer; thus, it is not likely that this slight
increase in pressure could have been responsible for the increase in subendocardial flow noted during exercise. In addition, a poor correlation ($r = 0.59$) was found between the increase in diastolic pressure and the flow to layer 4.

Vasodilatation of collateral channels secondary to exercise is the most reasonable explanation for the findings. If the resistance to flow in the endocardial layer is calculated using either the mean arterial pressure or the diastolic pressure as the true perfusion pressure, then resistance decreases significantly between pacing and exercise ($P < 0.001$), i.e., with diastolic pressure a decrease from $120 \pm 25$ mm Hg/ml per min per g during pacing to $73 \pm 16$ mm Hg/ml per min per g during exercise. Thus, vasodilatation of the collateral vessels resulted in the increase in flow to the subendocardial layer during exercise. The change in the caliber of the collateral vessels during exercise may have been due to a direct neurohumoral effect on the vascular smooth muscle. Alternatively, dilation of the epicardial collateral vessels may have resulted from altered ventricular geometry. If the external end-systolic dimensions of the heart were smaller during exercise than during pacing, the length of the epicardial collateral vessels may be shortened and the lumen diameters increased. Thus, when the major portion of coronary flow occurred during the initial part of diastole, the collateral vessels could have conducted more blood.

In the group II dogs, only minimal abnormal flow distribution occurred during pacing and exercise, i.e., the endo/epi was slightly less than in the noncollateral region. The hemodynamic data were the same for both groups. This indicates a difference in the collateral vessels and not the hemodynamic determinants of collateral flow as being responsible for the differences in flow distribution between group I and group II.

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References


References


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