Transmural Right Ventricular Myocardial Blood Flow during Systole in the Awake Dog

DAVID S. HESS AND ROBERT J. BACHE

SUMMARY This study was designed to quantify and compare transmural myocardial blood flow in the right and left ventricles of awake dogs during systole and diastole, and during total coronary artery occlusion. Studies were performed in six awake dogs chronically prepared with electromagnetic flowmeters and pneumatic occluders on the left circumflex and right coronary arteries. Intermittent coronary perfusion, confined to the interval of cardiac systole or an equivalent period during diastole, was effected by an R wave-triggered pneumatic valve connected to the occluders. To measure regional myocardial blood flow, radionuclide-labeled microspheres, 7-10 μm in diameter, were injected into the left atrium. We found that when arterial inflow was confined to systole, blood flow was normal in the left ventricular subepicardium, whereas flow to deeper myocardial layers was decreased as a linear function of tissue depth. When coronary arterial inflow was limited to an equivalent period in diastole, left ventricular transmural flow was uniform. In contrast to this, right ventricular transmural myocardial blood flow was unchanged from control values when coronary inflow was confined to systole or to an equivalent interval in diastole. During total coronary artery occlusion, absolute blood flow decreased similarly in myocardial areas perfused by the left circumflex and right coronary arteries. However, the transmural distribution of collateral blood flow was markedly different in the two ventricles. Left ventricular collateral flow was preferentially directed to the subepicardial layers, but right ventricular collateral flow was distributed uniformly across the ventricular wall. The transmural gradients observed in the left ventricle during total coronary artery occlusion and when arterial inflow was confined to systole can be explained by the functioning of a vascular waterfall during left ventricular contraction.


SINCE Langendorff (1900) first observed that cardiac contraction impedes coronary blood flow, the effect of ventricular systole on myocardial perfusion has been studied by several investigators (Anrep et al., 1927; Green et al., 1935; Sabiston and Gregg, 1957). Although blood flow to the subendocardial myocardium equals or exceeds subepicardial flow in the left ventricle of the dog during conditions of normal coronary inflow (Cobb et al., 1974), when coronary inflow is confined to systole, a significant transmural perfusion gradient develops with selective underperfusion of the subendocardium (Downey and Kirk, 1974; Downey et al., 1974b; Hess and Bache, 1976). The most widely accepted mechanism for production of this systolic perfusion gradient invokes the functioning of vascular waterfalls controlled by the intramural compressive forces generated by left ventricular contraction (Permutt and Riley, 1963; Downey and Kirk, 1974; Hess and Bache, 1966). A second possible mechanism would involve distortion of the intramural coronary vasculature by myocardial shear forces which may occur as the myocardium shortens during left ventricular ejection (Downey et al., 1974b). Since the normal right ventricle undergoes considerable systolic shortening at low intracavitary pressures (and presumably low intramural pressures), comparison of the distribution of myocardial blood flow in the right and left ventricles during systole would provide insight into the relative importance of myocardial shortening (myocardial strains) and pressure development in transmural myocardial blood flow within the two ventricles. Consequently, the present study was designed to measure transmural myocardial blood flow in the right and left ventricles of dogs when coronary arterial inflow was limited to the interval of systole. All studies were performed in chronically instrumented awake dogs to avoid possible interfering effects associated with general anesthesia and acute surgical trauma.

Methods

Six adult mongrel dogs weighing 20-32 kg were anesthetized with sodium thiopental (20-25 mg/kg, iv), ventilated with a respirator, and underwent a left thoracotomy in the 4th intercostal space. A heparin-filled polyvinyl chloride catheter (outside diameter 3.0 mm) was introduced into the arch of the aorta via the left internal thoracic artery. The pericardium was opened and a similar catheter was introduced into the left atrial cavity through the atrial appendage and secured with a purse-string suture. A third catheter was introduced into the
right ventricular cavity and secured with a purse-string suture. The proximal 1.5 cm of the circumflex branch of the left coronary artery was dissected free and a pneumatic occluder constructed in our laboratory of polyvinyl chloride tubing (outside diameter 2.7 mm) was placed around the artery proximal to any branches (Debley, 1971). An electromagnetic flowmeter probe (Statham Instruments) was positioned around the left circumflex coronary artery proximal to the pneumatic occluder. The proximal 1.5 cm of the right coronary artery was dissected free and a similar occluder was positioned around the artery proximal to any branches. In two dogs, an electromagnetic flowmeter probe was positioned around the right coronary artery proximal to the pneumatic occluder. The catheters, occluder tubing, and electromagnetic flowmeter leads were tunneled dorsally into a subcutaneous pouch at the base of the neck but were not exteriorized to protect them from damage.

Studies were conducted 7–13 days after the initial surgery. All dogs were active and fully recovered from surgery without fever, anemia, or other evidence of ill health. On the morning before study, the catheters, occluder tubing, and flowmeter leads were exteriorized through a 1-cm skin incision, using 2% lidocaine infiltration anesthesia. Dogs were trained to lie quietly on their right sides during study. The laboratory was dimly illuminated and free from noise or other activity which might disturb the animal. Coronary blood flow was measured with Statham M-400 electromagnetic flowmeters and pressures were measured with Statham P23Db pressure transducers. Lead II of a standard electrocardiogram was obtained. Data were recorded on an 8-channel analog magnetic tape recorder. After all recording instruments had been connected, a 60-minute interval was allowed for the dog to adjust to the laboratory conditions. During this time, hemodynamic variables were recorded continuously to ensure that a control steady state had been achieved.

A previously described electrical circuit was used to control the coronary artery occluders and to confine right and circumflex coronary arterial inflow to either systole or diastole (Hess and Bache, 1976). Briefly, an R wave trigger coupled to a variable delay circuit was used to actuate a solenoid valve. When triggered by this circuit, the valve inflated the occluder from a compressed air source at a pressure of 1500 mm Hg. By adjusting the delay circuit, coronary arterial inflow began 100 msec after the dicrotic notch for an interval equal to the period of ventricular ejection. The term "diastolic perfusion" will refer to that situation in which the occlusion circuitry was adjusted so that coronary arterial inflow began 100 msec after the dicrotic notch for an interval equal to the period of ventricular ejection. Sustained coronary artery occlusions were accomplished by manually triggering the solenoid valve. Proper functioning of the trigger device and pneumatic occluders was monitored by observing the coronary blood flow signal from the electromagnetic flowmeter probes. In those dogs without electromagnetic flowmeter probes on the right coronary artery, proper function of the occluder was verified by demonstrating at autopsy that inflow of normal saline from a pressure head of 200 mm Hg into the coronary system was prevented by the inflated pneumatic cuff.

Regional myocardial blood flow was measured by injecting into the left atrium microspheres 7–10 μm in diameter and labeled with a gamma-emitting radionuclide, either 141Ce, 51Cr, 85Sr, or 46Sc. The microspheres were diluted in 10% low molecular weight dextran so that 1.0 ml, the volume injected, contained approximately 3 x 10^6 microspheres. Before injection, the microspheres were mixed by alternate agitation for at least 15 minutes in an ultrasonic bath and a Vortex agitator. Complete dispersion of microspheres was verified by examining a drop of microsphere suspension with a light microscope. During each intervention, the microspheres were injected over a 5-second interval and flushed in with 5 ml of normal saline. Transmural myocardial blood flow was measured during four different experimental conditions. (1) To study the effect of cardiac contraction on transmural right and left ventricular blood flow, 1.0 ml of microsphere suspension was injected into the left atrium during systolic perfusion. The microsphere injection was begun 30 seconds after systolic perfusion was begun, and this perfusion was continued for an additional 30 seconds. (2) To ensure that any transmural gradient observed during systolic perfusion was related to ventricular contraction and was not an artifact related to intermittent coronary perfusion, a second injection of microspheres was made using an identical protocol during diastolic coronary perfusion. (3) To provide a control measurement of transmural myocardial perfusion, microspheres were injected during resting control conditions in the presence of unimpeded coronary blood flow. (4) To evaluate the magnitude and distribution of left and right ventricular intercoronary collateral blood flow, a final injection of microspheres was performed 5 seconds after the onset of a total coronary artery occlusion. The occlusion was maintained for 45 seconds after the injection was completed to ensure complete dispersion of microspheres before the occluder was deflated. Beginning simultaneously with each microsphere injection and continuing for 90 seconds, a reference sample of arterial blood was
weighed, and placed in vials for counting. These tissue blocks were removed from the uniformly stained region of the posterior left ventricular weight, were used for study. Duplicate full thickness tissue blocks were obtained from the densely stained area of the right ventricular free wall, including the region of the posterior papillary muscle. Similarly, duplicate full thickness tissue blocks were obtained from the densely stained region of the posterior left ventricular free wall, including the region of the posterior papillary muscle. These layers subsequently were referred to as layers 1 through 4, layer 1 being most epicardial and layer 4 the most endocardial. Myocardial and blood reference specimens were counted in a Beckman gamma spectrometer (model 16776) at window settings corresponding to the peak energies emitted by each radionuclide. The activity recorded in each energy window was corrected for contaminant activity contributed by the associated nuclides and for background activity. Knowing the rate of withdrawal of the reference sample (Qc) and the radioactivity of the reference sample (Cc), used myocardial sample radioactivity (Cm) to compute myocardial blood flow (Qm) as Qm = Qc - Cm/Cc. Blood flow to each myocardial specimen was then divided by the sample weight and expressed as ml/min per g of myocardium.

Results

Heart rate, mean aortic pressure, and left and right ventricular pressures measured in six dogs are shown in Table 1. During control conditions, heart rates ranged from 54 to 96 beats/min, and mean arterial pressures were 71 to 104 mm Hg. Left ventricular systolic pressures ranged from 80 to 130 mm Hg, while diastolic pressures ranged from 3 to 7 mm Hg. Right ventricular systolic pressures ranged from 15 to 26 mm Hg with diastolic pressures of 0 to 4 mm Hg. At the time of injection of microspheres, no change in any of these variables occurred during systolic or diastolic perfusion, or during total occlusion of the circumflex and right coronary arteries.

Mean myocardial blood flow to the left ventricle during control conditions was 0.72 ± 0.07 ml/min per g (Table 2, Fig. 1). Subendocardial blood flow was slightly greater than subepicardial flow, but this difference was not significant (P > 0.1). With the onset of intermittent coronary artery perfusion (either systolic or diastolic), mean coronary flow immediately fell as a result of the decreased interval available for blood flow. However, as vasodilation occurred in response to the shortened perfusion interval, coronary blood flow increased to achieve a new steady state within the first 8 to 10 heart beats. Mean left ventricular blood flow during systolic perfusion was 0.44 ± 0.05 ml/min per g, significantly less than control (P < 0.01). As shown in Figure 1, during systolic perfusion, flow to layer 1 was not significantly different from control, whereas flow to deeper myocardial layers decreased regularly from layer 2 through layer 4, resulting in an inverse correlation between blood low and the depth of the muscle layer. During diastolic perfusion, neither mean myocardial blood flow nor the transmural distribution of left ventricular perfusion was significantly different from control (Table 2, Fig. 1). Thus, the decrease in absolute blood flow to myocardial layers 2 through 4 observed during systolic perfusion was related to ventricular systole and was not merely an artifact due to intermittent coronary artery perfusion.

Right ventricular myocardial blood flows during control conditions and during systolic and diastolic perfusion are shown in Figure 2 and Table 2. Mean right ventricular myocardial blood flow during control conditions was 0.37 ± 0.05 ml/min per g, and subendocardial flow significantly exceeded subepicardial flow (endo/epi = 1.35; P < 0.01). Neither mean right ventricular myocardial blood flow nor the transmural distribution of right ventricular perfusion was significantly altered during systolic per-

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<th>Table 1 Hemodynamic Data</th>
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<tr>
<td>Heart rate (beats/min)</td>
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<td>Control</td>
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<tr>
<td>Systolic perfusion</td>
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<td>Diastolic perfusion</td>
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<td>Coronary occlusion</td>
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RIGHT VENTRICULAR BLOOD FLOW/Hess and Bache

Table 2  Mean Myocardial Blood Flow and the Ratio of Subendocardial/Subepicardial Flow (Endo/Epi)

<table>
<thead>
<tr>
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<th>Left ventricle</th>
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<th>Right ventricle</th>
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<tr>
<td></td>
<td>Myocardial</td>
<td>Endo/Epi</td>
<td>Myocardial</td>
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<td>(ml/min per g)</td>
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<tr>
<td>Control</td>
<td>0.72 ± 0.07</td>
<td>1.13</td>
<td>0.37 ± 0.05</td>
<td>1.35</td>
</tr>
<tr>
<td>Systolic perfusion</td>
<td>0.44 ± 0.05*</td>
<td>0.34*</td>
<td>0.31 ± 0.07</td>
<td>1.30</td>
</tr>
<tr>
<td>Diastolic perfusion</td>
<td>0.72 ± 0.12</td>
<td>1.15</td>
<td>0.32 ± 0.04</td>
<td>1.42</td>
</tr>
<tr>
<td>Total occlusion</td>
<td>0.15 ± 0.08*</td>
<td>0.28*</td>
<td>0.09 ± 0.02*</td>
<td>1.13</td>
</tr>
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* P < 0.05, compared with control measurements.

fusion. Similarly, diastolic perfusion did not result in a significant change of either mean right ventricular blood flow or the transmural distribution of this flow.

During total coronary artery occlusion, mean flow to the posterior left ventricular wall (representing intercoronary collateral inflow) fell to 21 ± 7% of the control value while mean right ventricular blood flow fell to 24 ± 2% of control (Table 2). However, as shown in Figure 3, the distribution of the intercoronary collateral inflow was different in the left and right ventricles. In the left ventricle, blood flow progressively decreased from epicardium to endocardium (endo/epi = 0.28), whereas in the right ventricle the transmural distribution of collateral flow during coronary occlusion was uniform (endo/epi = 1.13).

Discussion

In previous studies of the mechanism by which cardiac contraction influences myocardial perfusion, Downey and associates (1974b) examined the transmural distribution of myocardial blood flow by administration of 42K or 86Rb into the cannulated left main coronary artery of open-chest dogs. These investigators demonstrated that limiting coronary perfusion to the interval of systole in isovolumic contracting hearts resulted in a significant transmural gradient of flow favoring the subepicardium (endo/epi = 0.36). To study the importance of left ventricular pressure development in production of this transmural perfusion gradient, these workers then compared systolic perfusion in hearts contracting against a normal afterload with that of hearts contracting against a reduced afterload produced by abruptly severing the thoracic aorta. In the normally afterloaded hearts, a systolic perfusion gradient existed that favored perfusion of the subepicardium (endo/epi = 0.63); in contrast to this, transmural perfusion was essentially uniform when the left ventricle was allowed to contract against a reduced afterload (endo/epi = 1.04). From these data it was concluded that development of pressure, and not myocardial shortening, was the predominant factor responsible for the transmural left ventricular perfusion gradient observed during systolic perfusion. Although absolute blood flow rates could not be observed with the indicator techniques used in that study, it was of interest that the relative systolic underperfusion observed in the isovolumically contracting hearts (endo/epi = 0.37) was considerably more marked than in the afterloaded hearts which were allowed to shorten (endo/epi = 0.62), despite similar peak left ventricular pressures in the two preparations. One might rather have expected similar transmural perfusion gradients if pressure development was the dominant factor in determining the pattern of transmural blood flow. It is possible that these observed differences may

![Figure 1](https://example.com/figure1.png)  
**Figure 1**  Mean left ventricular myocardial blood flow (ml/g per min) ± SEM to all transmural layers measured during control conditions (solid line), systolic perfusion (A, broken line), and diastolic perfusion (B, broken line).

![Figure 2](https://example.com/figure2.png)  
**Figure 2**  Mean right ventricular myocardial blood flow (ml/g per min) ± SEM to all transmural layers measured during control conditions (solid line), systolic perfusion (A, broken line), and diastolic perfusion (B, broken line).
relate in part to differences in experimental preparation between the isovolumic and the afterloaded but shortening hearts (the former used an isolated heart preparation and the latter was carried out in open-chest dogs). However, the availability of only two indicators in that study prevented examination of the influence of the experimental preparation on the control transmural myocardial perfusion. In the present study, these technical difficulties were circumvented by employing the radioactive microsphere technique, which allowed serial measurements of blood flow during control conditions and several experimental interventions, as well as calculation of absolute blood flow rates. Furthermore, studies were performed with awake dogs to avoid any possible influence of anesthesia or acute surgical trauma on cardiac contraction or myocardial blood flow. Finally, an attempt was made to assess the possible effect of intercoronary collateral inflow on the data obtained during intermittent coronary artery perfusion.

In the present study, systolic perfusion of the left ventricle resulted in a gradient of blood flow decreasing from epicardium to endocardium, with absolute hypoperfusion of the subendocardium. That this perfusion gradient was a function of ventricular systole was demonstrated by the uniform transmural myocardial blood flow observed during diastolic perfusion. Any possible influence of intercoronary collateral flow during systolic perfusion was evaluated by measuring transmural myocardial blood flow during total coronary artery occlusion. In all dogs, coronary occlusion produced a marked reduction in total blood flow, with the subendocardium receiving significantly less collateral flow than the subepicardium. If systolic perfusion resulted in uniform ischemia and maximal collateral flow occurred, the observed systolic perfusion gradient would be unaffected by correcting for this collateral flow. For example, subepicardial flow would correct to 0.46 ml/min per g (0.69 ml/min per g systolic perfusion — 0.23 ml/min per g coronary collateral flow) and subendocardial flow to 0.16 ml/min per g (0.23 ml/min per g systolic perfusion — 0.07 ml/min per g collateral flow) with a resultant endocardial-to-epicardial ratio of 0.34, the same as before correction. However, if systolic perfusion resulted in selective ischemia of the subendocardial layers, then collateral inflow may have been directed preferentially to the ischemic subendocardium. If this were the case, correction for collateral flow would result in accentuation of the observed gradient. Thus, the observed transmural perfusion gradient during systolic perfusion was not an artifact of intermittent perfusion or collateral inflow, but was related to events associated with left ventricular contraction.

The myocardial blood flow characteristics of the right ventricle are of interest in interpreting the flow data from the left ventricle. Average myocardial blood flow to the right ventricle during control conditions was 0.37 ml/min per g, in agreement with data reported by Lowensohn and associates (1976). In the present study, the right ventricular subendocardium received significantly more blood flow than did the subepicardium. We have previously reported that in the resting awake dog the right ventricular endocardial-to-epicardial blood flow ratio was not significantly different from 1.0 (Ball et al., 1975). The discrepancy between these results may be related to sampling technique. In the previous study, the right ventricular wall was divided into only two equal outer and inner portions whereas, in the present study, the wall was divided into four layers. Thus, the present study may have detected a gradient which did not achieve statistical significance in the previous report because of sample averaging. In the present study, right ventricular myocardial blood flow did not change significantly during systolic or diastolic perfusion. The finding that right ventricular contraction did not alter myocardial blood flow supports the hypothesis that myocardial shear forces are a minor determinant of systolic coronary vascular resistance. Any influence of right ventricular collateral inflow on these observations was minimal because: (1) with normal flows, ischemia did not appear to be present, as witnessed by lack of reactive hyperemia after systolic or diastolic perfusion in the two dogs with right coronary flowmeters, and (2) coronary collateral flow was distributed uniformly across the right ventricular wall.

These observations help to explain the phasic pattern of right coronary artery blood flow. In con-

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**FIGURE 3 Mean left ventricular (A) and right ventricular (B) myocardial blood flow (ml/g per min) ± SEM to all transmural layers from epicardium to endocardium (1 to 4) measured during control conditions (solid line) and during total coronary artery occlusion (broken line).**
trast to flow in the left coronary arteries, a substantial portion of right coronary flow occurs during systole, so that average systolic flow rates are equal in magnitude to diastolic flow rates (Gregg, 1937). Lowensohn and associates (1976) found that this unique pattern of right coronary artery flow disappeared when severe right ventricular systolic hypertension was present. These observations may be interpreted by considering the factors that determine systolic coronary vascular resistance. Impedance to systolic blood flow is determined by (1) vasomotor tone, (2) myocardial shear forces, and (3) intramyocardial pressure (Downey and Kirk, 1974; Hess and Bache, 1976). Since myocardial shear forces appear to be of relatively minor importance, systolic blood flow is determined principally by the dynamic interaction of vasomotor tone and intramyocardial pressure (Downey et al., 1974b). This interplay results in flow dynamics which may be described by the vascular waterfall (Permutt et al., 1962; Downey and Kirk, 1974; Hess and Bache, 1976). Briefly, flow in such a system is determined by vasomotor tone as long as capillary outflow pressure exceeds the surrounding tissue pressure, but flow becomes a function of tissue pressure whenever it exceeds capillary outflow pressure. These dynamics not only explain the observed left ventricular systolic perfusion gradient, but also may pertain to right ventricular flow. Although right ventricular intramyocardial pressure has not been investigated systematically, it is likely that the pressure distribution resembles that of the left ventricle (Brandi and McGregor, 1969). Thus, right ventricular intramyocardial pressure would increase from epicardium to endocardium, with subendocardial pressure approaching right ventricular intracavitary pressure. Since the normal intracavitary pressure during systole (approximately 20 mm Hg) is similar to capillary outflow pressure (Permutt and Riley, 1965; Landis, 1930), vasomotor tone would control coronary resistance across the entire right ventricular wall during systole. This postulate is supported by the finding that the pattern of transmural perfusion during systolic perfusion in the right ventricle was normal, with the subendocardium receiving slightly more flow than the subepicardium. This transmural gradient can be explained only by the effects of active coronary vasomotion, since intramyocardial pressure would function to impede subendocardial flow. On the basis of these considerations, it is not unexpected that systolic flow rates are equal to diastolic flow rates in the normal right coronary artery (Gregg, 1937), since vasomotion, and not intramyocardial pressure, determines systolic resistance. However, if right ventricular systolic pressures were increased, we predict that intramyocardial pressure would become a major determinant of systolic flow as it is in the left ventricle. This effect was fact observed by Lowensohn and associates (1976) in dogs with varying degrees of right ventricular hypertension. Thus, the ratio of systolic to diastolic flow in the right coronary artery decreased from 0.37 in the presence of normal right ventricular pressure, to 0.33 with moderate right ventricular hypertension, to 0.12 in dogs in which right ventricular pressures equaled or exceeded aortic pressure. This increasing impedance to right coronary artery systolic flow concomitant with increasing right ventricular systolic pressure is consistent with the vascular waterfall hypothesis.

The transmural distribution of the collateral blood flow measured during total coronary artery occlusion deserves discussion. Several investigators have demonstrated that coronary occlusion involving the left ventricle results in a marked decrease in absolute blood flow, with hypoperfusion most severe in the subendocardium (Kjekshus et al., 1973; Downey et al., 1974a). Downey et al. (1974a) demonstrated that this collateral flow gradient could be reversed by using a cannula to collect retrograde collateral flow entering the occluded epicardial coronary arteries, thus diverting blood away from the myocardium. This diversion of epicardial flow resulted in an 80% reduction in total collateral inflow. These investigators interpreted their data to indicate that most of the collateral inflow available immediately after acute coronary artery occlusion is delivered via epicardial collateral channels. This study may be criticized, however, because it is possible that a portion of the blood delivered via subendocardial collaterals might also have been diverted into the epicardial system via intramural communications between the subendocardial and epicardial vessels. Nevertheless, these data are in agreement with the anatomic studies of Schaper (1971) showing that, in the dog, the intercoronary collateral vasculature resides principally at the epicardial surface of the heart. The previously discussed characteristics of systolic and diastolic perfusion in the left ventricle indicate that collateral flow delivered into the epicardial vessels would be distributed uniformly across the left ventricular wall during diastole, but would be distributed according to the vascular waterfall during systole. Since the subepicardial layers could receive collateral flow throughout the cardiac cycle, whereas collateral perfusion of the subendocardium can occur only during diastole, a net perfusion gradient favoring the subepicardium would result. Failure to observe a transmural gradient in coronary collateral flow in the right ventricle suggests that, in the presence of normal right ventricular pressures, collateral flow is a function of local arteriolar resistance and not intramural mechanical factors. Thus, ischemia of the right ventricular myocardium produced by total right coronary artery occlusion resulted in maximal coronary arteriolar vasodilation, but the transmural distribution of the collateral blood flow remained uniform.
In conclusion, systolic perfusion resulted in a transmural gradient of left ventricular blood flow with normal subepicardial flow but subendocardial underperfusion. Because a similar gradient did not occur during systolic perfusion of the right ventricle, it is concluded that pressure development, and not myocardial shortening, is responsible for the systolic impedance to ventricular blood flow according to the flow dynamics described by the vascular waterfall theory. In addition, the observed transmural gradient of collateral blood flow that occurred during acute left coronary artery occlusion, but not right coronary artery occlusion, can be attributed to the functioning of vascular waterfalls during left ventricular systole. It should be noted that, although these findings characterize the role of intramyocardial tissue pressure in regulating transmural myocardial blood flow during systole, the present data do not define the extraordinarily complex pattern of local stresses which may occur within the left ventricular wall during systole (Mirsky et al., 1974).

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Transmural right ventricular myocardial blood flow during systole in the awake dog.
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Circ Res. 1979;45:88-94
doi: 10.1161/01.RES.45.1.88

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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