Study of Luminal Coronary Collateral Circulation in the Beating Canine Heart

By Thomas W. Moir, M.D.

ABSTRACT
Coronary collateral circulation from the cardiac lumen was studied in 36 anesthetized, open-chest dogs with hearts beating in situ. The coronary arteries were cannulated and perfused from an isolated, nonradioactive blood source while $^{131}$I-labeled blood circulated through the cardiac lumen for 2 minutes. Ventricular fibrillation was then produced, and luminal blood samples and myocardial tissue samples were obtained for radioactive assay and calculation of the amount of luminal blood appearing as a part of the myocardial blood volume. These volumes were obtained from both ventricles and the atria under (1) normal antegrade coronary flow conditions, (2) myocardial ischemia, (3) left and right ventricular hypertension, and (4) coronary occlusion. Small quantities of luminal blood appeared in the left ventricular myocardium under normal coronary flow conditions while considerably larger amounts were found in the right ventricle and atria. There was no significant increase of the luminal contribution to the myocardial blood volume in response to the various experimental conditions except that of coronary occlusion. Although in the latter, the amount of left ventricular luminal blood was greater than with normal coronary flow conditions, right ventricular and atrial myocardial blood volumes remained greater than the left.

ADDITIONAL KEY WORDS arterioluminal and arteriosinusoidal vessels thebesian flow retrograde coronary sinus flow septal artery anterior cardiac veins left anterior atrial artery

Bloor and Liebow classified the varieties of mammalian coronary collateral circulation as: intercoronary, retrocardiac, transepicardial, and endomural (1). The functional role in the coronary circulation has been substantially documented for these types of vessels except the endomural, or luminal, vessels for which data are scarce, particularly under conditions of coronary flow that are representative of clinically important hemodynamic abnormalities.

Luminal coronary vessels are vascular communications between the chambers of the heart and the coronary circulation. These vessels may connect with the coronary arterial circulation and have been designated as arterioluminal and arteriosinusoidal vessels by Wearn et al. (2). Alternatively, they may connect the cardiac chambers with the coronary venous system, as originally described by Thebesius (3). Some of these vessels have been shown to serve as drainage channels from the coronary circulation into the cardiac chambers of the normally beating dog heart (4, 5), and this information has reawakened interest in the possibility that this system might serve as a source of coronary collateral circulation. To date, study of this possibility has been scanty, not representative of abnormal coronary flow conditions, and inconclusive (6-9).

The present experiments were designed to make a systematic evaluation of the possibility of coronary collateral circulation through the luminal system in response to acute hemodynamic interventions that were designed to simulate important clinical abnormalities.

Methods
Thirty-six mongrel dogs weighing 18 to 22 kg were anesthetized with morphine, 1 mg/kg sc,
and pentobarbital, 20 mg/kg iv, and positive-pressure breathing was established through an occlusive intratracheal tube. After the left chest was opened, the internal mammary (internal thoracic) arteries were ligated as close as possible to their origin at the subclavian artery, the pericardium was incised, heparin (10 mg/kg iv) was given, and the left jugular vein and the right common carotid artery were cannulated. In some animals, the coronary sinus was cannulated with an occlusive balloon-tip Morawitz cannula inserted through the right atrial appendage and the sinus outflow diverted through an external circuit into the cannulated left jugular vein; this flow could be drained to atmospheric pressure by appropriate clamping of the circuit. The common left coronary artery was cannulated through its aortic ostium via the left subclavian artery and perfused from an air-pressurized reservoir, which was kept filled with blood delivered from the right carotid artery by a pump. Left coronary artery perfusion pressure could be held constant at any desired level, independent of aortic pressure, by appropriate setting of the air pressure in the chamber. This reservoir was also connected to a nonradioactive blood source so that left coronary arterial perfusion could be isolated from the animal's systemic circulation by appropriate clamping. Ligatures were placed around the right coronary artery as close as possible to its aortic ostium and around the right coronary accessory artery, where identified. These arteries were usually ligated just before the acute interventions of the study; however, in animals in which right coronary artery constriction, the right coronary artery was cannulated through its aortic ostium via the left subclavian artery and perfused from the left coronary perfusion reservoir.

The thoracic aorta was ligated and the proximal portion cannulated and allowed to bleed into another air-pressurized blood reservoir so that aortic pressure could also be held at any desired level independent of coronary perfusion pressure.

The left coronary flow rate was measured by an electromagnetic flowmeter from a cannulating probe inserted in the left coronary arterial circuit. Mean aortic pressure, mean left coronary perfusion pressure, mean right atrial pressure, phasic left ventricular pressure and, in some cases, phasic right ventricular pressure were continuously monitored by pressure transducers and recorded together with the coronary flow curve. An electrocardiogram was also obtained with each study.

With these arrangements the coronary circulation could be isolated from the animal's systemic circulation, and labeled systemic blood appearing in the myocardium during the study was assumed to have appeared via channels other than the coronary arteries. When the conditions of the study were established and the coronary circulation was isolated and perfused from a donor source, systemic blood was labeled by left jugular vein injection of 50 μc of 131I albumin that circulated through the cavities of the heart for 2 minutes. The heart was then electrically fibrillated, cavitary blood samples quickly obtained, and the heart immediately excised.

Assay of the myocardium for the presence of labeled luminal blood was determined by two methods of preparing the excised hearts. In each study group there were six animals: in three the entire heart, including the lumen, was thoroughly washed with tap water for 2 to 3 minutes after ligation of the coronary sinus orifice and the aortic ostium of the left coronary artery; in the other three the heart was immediately frozen in a flask of liquid nitrogen. In each heart, six transmural cores were obtained from the free wall of the left ventricle, three from the interventricular septum and five from the right ventricle. The samples from the left ventricular free wall included one each from the anterior and posterior papillary muscles. With the washed hearts, the tissue samples were obtained with a trephine, while the frozen heart samples were obtained with a hammer and chisel after scraping all visible blood and 0.5 to 1.0 mm of tissue from the endocardial surface. Three samples were obtained from the right and left atrium, but only in the washed hearts; it was impossible to completely remove the luminal blood from the endocardial surface of the frozen atria because of the numerous trabeculae. The ventricular and septal samples were divided into inner and outer halves, and weighed in previously tared plastic tubes on an analytic balance; sample weight varied between 0.5 and 1.0 g. The atrial samples were not divided into inner and outer portions but were otherwise similarly handled; their weight also varied between 0.5 and 1.0 g. These specimens were then assayed for radioactivity in a scintillation counter together with a 1-ml sample of the systemic blood obtained from the ventricular lumen in each case. The well counter used insured constant geometry with sample volumes sizes up to 5 ml; all tissue samples counted were within this volume limit. Each tissue specimen was counted for a minimum period of 4 minutes, and the absolute counting rates varied between 200 to 1000 counts/min with the standard deviation ranging between 1.5% and 3.5%. The amount of luminal blood that accumulated in the myocardium was estimated by the ratio of radioactivity in the myocardium during the study was assumed to have appeared via channels other than the coronary arteries.
myocardial sample, in counts/min/g, to that in the luminal blood aliquot, in counts/min/ml, with the resultant expression of ml/g. This figure multiplied by 100 gave a value of ml/100 g of myocardium and as such represents that volume of myocardial blood which came through luminal communications.

The distribution of the luminal blood in the endocardial and epicardial layers of the right and left ventricular myocardium was estimated by subtraction of the volume of labeled blood of the outer from the inner halves of the respective ventricles and notation made of positive or negative differences; the mean values found in the washed and frozen techniques were pooled for this evaluation after individual analysis showed no difference between the two techniques as to location. The significance of the differences found between these sites was evaluated by the sign test (10) and are shown in Tables 4 and 5.

The two methods of handling the heart after excision were used to obtain an average figure for the accumulation of labeled blood in the myocardium. On the one hand, washing the heart more completely eliminates endocardial surface radioactivity, but may allow blood in luminal channels to run out. On the other hand, freezing the heart prevents blood from leaking out of luminal vessels but endocardial surface contamination may be relatively great. Thus, the tissue radioactivity represented in the washed samples is regarded as a minimum value, while that of the frozen samples is maximal.

Groups of six dogs each were studied under six different conditions of coronary perfusion pressure and flow, and intraventricular pressure:

1. **Normal:** maintenance of normal left coronary pressure and flow, maintenance of normal left ventricular pressure.

2. **Ischemia:** marked lowering of left coronary pressure and flow, maintenance of normal left ventricular systolic pressure, elevation of left ventricular diastolic pressure.

3. **Left ventricular hypertension:** moderate lowering of left coronary perfusion pressure and flow, elevation of left ventricular systolic pressure, maintenance of normal left ventricular diastolic pressure.

4. **Right ventricular hypertension:** moderate lowering of right and left coronary perfusion pressure and flow, lowering of left ventricular systolic pressure, maintenance of normal left ventricular diastolic pressure, elevation of right ventricular systolic and diastolic pressure by pulmonary artery constriction.

5. **Coronary occlusion (coronary sinus intact):** complete coronary occlusion resulting in low left ventricular systolic pressure and high left ventricular diastolic pressure, coronary sinus draining normally into the right atrium.

6. **Coronary occlusion (coronary sinus draining out):** complete coronary occlusion resulting in low left ventricular systolic pressure and high left ventricular diastolic pressure, coronary sinus draining out to atmospheric pressure.

The average values for pressure and flow in each study group are presented in Table 1.

### Results

#### Luminal Myocardial Blood Volume

The mean volumes of labeled luminal blood accumulating in the myocardium of the right and left ventricles and the atria after the hemodynamic interventions of the study are presented in Tables 2 and 3.

**Normal.** The mean values of labeled blood found in the right ventricular myocardium of the washed and frozen preparations were not significantly different from each other. Similarly, in the left ventricle there were no differences between the septum and free wall as to technique or location, although the mean radioactive content of the myocardium in these areas was significantly less than the right ventricle (P < 0.05; Wilcoxon rank sum test [10]). Both atria had significantly larger amounts of radioactivity than the left ventricle (P < 0.05) but were not significantly different from each other or the right ventricle. Vascular pressures, coronary flow, and the electrocardiogram remained normal in this group.

**Myocardial Ischemia.** The amount of labeled blood in the right ventricular myocardium was not significantly greater than normal in the washed hearts, but the increase in the frozen hearts was significant (P < 0.05). In the septum and free wall of the left ventricle there was no significant difference in the accumulation of labeled blood over that of the normal group, and there was no difference between the septum and the free wall. The left ventricles of the frozen hearts did contain more than the washed hearts (P < 0.05). Both atria had significantly larger amounts of labeled blood than the left ventricle (P < 0.05). Additionally, the right atrium contained significantly more labeled blood than the right ventricle or left atrium (P < 0.05).
**TABLE 1**

Average Vascular Pressures and Left Coronary Flow during the Various Conditions of the Study

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mean pressures (mm Hg)</th>
<th>Mean left coronary flow (ml/100 g/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean aortic</td>
<td>Mean coronary perfusion</td>
</tr>
<tr>
<td>Normal</td>
<td>107</td>
<td>107</td>
</tr>
<tr>
<td>Ischemia</td>
<td>108</td>
<td>46</td>
</tr>
<tr>
<td>LV hypertension</td>
<td>164</td>
<td>86</td>
</tr>
<tr>
<td>RV hypertension</td>
<td>66</td>
<td>66</td>
</tr>
<tr>
<td>Coronary occlusion (coronary sinus intact)</td>
<td>107</td>
<td>12</td>
</tr>
<tr>
<td>Coronary occlusion (coronary sinus draining out)</td>
<td>103</td>
<td>14</td>
</tr>
</tbody>
</table>

LV = left ventricle; RV = right ventricle.

*Left plus right coronary artery flow.

**TABLE 2**

Mean Volumes of Isotope-Labeled Blood in the Ventricular Myocardium

<table>
<thead>
<tr>
<th>Condition</th>
<th>Technique</th>
<th>Right ventricle (ml/100 g)</th>
<th>Left ventricle (ml/100 g)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean ±1 SD</td>
<td>Mean ±1 SD</td>
</tr>
<tr>
<td></td>
<td>n</td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>Normal</td>
<td>Washed</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Freeze</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>Ischemia</td>
<td>Washed</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Freeze</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>LV hypertension</td>
<td>Washed</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Freeze</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>RV hypertension</td>
<td>Washed</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Freeze</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>Coronary occlusion (coronary sinus intact)</td>
<td>Washed</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Freeze</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>Coronary occlusion (coronary sinus draining out)</td>
<td>Washed</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Freeze</td>
<td>3</td>
<td>15</td>
</tr>
</tbody>
</table>

N = number of animals; n = number of observations. Each n observation is the average of the values in the inner and those in the outer halves of the transmural cores of myocardium. Means and standard deviations refer to these n observations.
induced myocardial ischemia caused left ventricular deterioration as manifested by ST-T abnormalities in the cardiogram and elevation of the left ventricular diastolic pressure.

**Left Ventricular Hypertension.**—There was no significant difference between the washed and frozen hearts. Although the mean value for right ventricular labeled blood was greater than in the normal group the range of individual values was wide and the difference was not significant. However, the right ventricular accumulation was significantly greater than the left ventricle ($P < 0.05$). In the latter there was no increase over the normal group, nor was there any difference between the septum and the free wall. Both atria contained significantly more labeled blood than the left ventricle and the atria of the normal group, but were not different from the right ventricle. There was no evidence of myocardial deterioration in these hearts as reflected by the cardiogram or pressure pulses.

**Right Ventricular Hypertension.**—Neither the right nor left ventricle showed any increase in accumulation of labeled blood over that found in the normal group. Of particular importance is the lack of difference between the septum and the free wall of the left ventricle. The amount of right ventricular labeled blood was somewhat less than that in the normotensive right ventricle but not significantly so. The right atrium contained significantly more than either ventricle or the left atrium ($P < 0.05$). Although in this group there were variable abnormalities in the ST-T waves of the cardiogram and elevation of both systolic and diastolic pressure of the right ventricle, there was no hemodynamic evidence of left ventricular deterioration.

**Total Coronary Occlusion.**—Significant increases in the amount of labeled blood were seen in the atria, right ventricle, septum, and free wall of the left ventricle. This was true for both the washed and frozen hearts, although the latter had consistently greater amounts than the former. Similarly, in the coronary occlusion group in which the coronary sinus was drained out, significantly greater amounts of labeled blood appeared in all areas of the myocardium than in the normal animals, and these amounts were not significantly different from those in dogs with coronary occlusion but an intact coronary sinus. Within these coronary occlusion groups, the amounts of labeled blood were significantly greater in the right ventricle and atria than in the septum and free wall of the left ventricle. Again, the amounts in the septum and in the free wall did not differ significantly from each other. In every case rapid deterioration of cardiac function leading to ventricular fibrillation or arrest occurred within 2 to 3 minutes after coronary occlusion.

**Distribution of Luminal Myocardial Blood Volume**

In the right ventricle (Table 4) there was no significant difference in accumulation of labeled blood between endocardium and epicardium in the various study groups except
TABLE 4
Difference Between Endocardial (I) and Epicardial (O) Labeled Blood in the Right Ventricular Myocardium

<table>
<thead>
<tr>
<th>Condition</th>
<th>N</th>
<th>N+</th>
<th>N0</th>
<th>N-</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>24</td>
<td>17</td>
<td>0</td>
<td>7</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Ischemia</td>
<td>24</td>
<td>15</td>
<td>0</td>
<td>9</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>LV hypertension</td>
<td>24</td>
<td>15</td>
<td>1</td>
<td>8</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>RV hypertension</td>
<td>24</td>
<td>20</td>
<td>2</td>
<td>2</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Coronary occlusion (coronary sinus intact)</td>
<td>23</td>
<td>9</td>
<td>0</td>
<td>14</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Coronary occlusion (coronary sinus draining out)</td>
<td>24</td>
<td>10</td>
<td>1</td>
<td>13</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

N = number of observations; N+ = number of positive differences (I —O), N0 = number of no differences, N- = number of negative differences (I —O).

TABLE 5
Difference Between Endocardial (I) and Epicardial (O) Labeled Blood in the Left Ventricular Myocardium

<table>
<thead>
<tr>
<th>Condition</th>
<th>N</th>
<th>N+</th>
<th>N0</th>
<th>N-</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>53</td>
<td>27</td>
<td>3</td>
<td>23</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Ischemia</td>
<td>54</td>
<td>28</td>
<td>2</td>
<td>24</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>LV hypertension</td>
<td>50</td>
<td>25</td>
<td>0</td>
<td>25</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>RV hypertension</td>
<td>53</td>
<td>17</td>
<td>1</td>
<td>35</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Coronary occlusion (coronary sinus intact)</td>
<td>54</td>
<td>14</td>
<td>1</td>
<td>39</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Coronary occlusion (coronary sinus draining out)</td>
<td>54</td>
<td>11</td>
<td>0</td>
<td>43</td>
<td>&lt; 0.01</td>
</tr>
</tbody>
</table>

Symbols the same as in Table 4.

Discussion

The major purpose of this study was to show qualitatively whether blood could move from the cardiac lumen into the myocardium. However, a method which offered some degree of quantification seemed desirable, since different hemodynamic conditions were to be imposed. The method chosen was the determination of the ratio of myocardial to luminal radioactivity of a poorly diffusible whole blood isotope. The resultant expression of ml/100 g of myocardium represents that part of the total myocardial blood volume which accumulated through vascular channels other than the antegrade coronary circulation. This expression, however, neither indicates the amount of flow nor the types of luminal vessels in which the labeled blood accumulated.

Total left ventricular myocardial blood volumes in dogs with intact coronary circulations average 6 ml/100 g (11-13) and are somewhat lower, 5 ml/100 g, for the right ventricle (13). There are no reported values for the total atrial myocardial blood but we have found values similar to the right ventricle with no difference...
between right and left atrium (unpublished observations). In our normal animals the combined (washed and frozen) mean volumes of luminal blood of 0.40 ml/100 g for the left ventricle represents 7% of the total myocardial blood volume. The average value of 1.5 ml/100 g for the left atrium represents approximately 30% of its total myocardial blood volume. Since total ventricular and atrial myocardial blood volumes which resulted from the various hemodynamic interventions of this study are unknown, no accurate calculations can be made of the degree of contribution of luminal blood to the total under these circumstances. Nonetheless, the magnitude of accumulation of luminal blood in both atria and the right ventricle suggests that luminal collateral circulation might be functionally significant for these structures.

Before further discussing the possibility of luminal coronary flow as the source of the labeled blood appearing in the myocardium, the role of collateral flow via known extracardiac channels, the retrocardiac and transepipcardial vessels, must be considered. These vessels are small anastomoses between the coronary arteries and extracardiac vessels such as the vasa vasorum of the aorta and pulmonary artery, and the bronchial and pericardial arterial circulation (1). Post-mortem injection studies have shown the presence of these communications in the human heart (14); however, their functional role in the beating dog heart is uncertain. Although the capacity of these vessels to serve as collateral coronary circulation has been demonstrated, this has been true only in the presence of chronic coronary artery constriction coupled with pulmonary artery ligation and cardiopneumonopexy (15, 16). Similarly, the possibility of the internal mammary artery as an extracardiac coronary collateral source must be considered. Although there are internal mammary arterial communications with the pericardial circulation, there is no evidence that they are a significant source of coronary collateral circulation, even with the combination of peripheral internal mammary and coronary artery ligation (17-19). Nonetheless, in our study the internal mammary artery was ligated as close as possible to the subclavian artery to interrupt connections with the pericardiophrenic circulation. It is unlikely that the extracardiac circulation was a source of the labeled myocardial blood in our study, although elimination of this possibility would require autotransplantation of the heart.

Within the limits of the foregoing discussion, the results of our study are interpreted as showing the movement of systemic blood from the cardiac lumen into the myocardium. Except in the case of total coronary occlusion, the amounts of blood were not increased by the experimental attempts to increase the transfer of blood from the left ventricular lumen to the myocardium. Under all circumstances, however, the right ventricle and atria showed greater amounts of labeled blood than the left ventricle. When right ventricular hypertension was imposed, the septum did not accumulate greater amounts of labeled blood than the left ventricle. When right ventricular hypertension was imposed, the septum did not accumulate greater amounts of labeled blood than the left ventricular free wall. There was generally no difference in distribution of labeled blood between the inner and outer halves of either ventricle; where differences were found, the epicardial halves of the ventricles contained more labeled blood than the endocardial halves, except in the hypertensive right ventricle.

On the basis of this information, what types of vascular communications might be responsible for this blood flow from cardiac lumen to myocardium? First consideration should be given to channels connecting the coronary vasculature with the cardiac cavities and having a proved functional role in the beating heart. The coronary sinus, the anterior cardiac veins, the septal branch of the left coronary artery, and the left anterior atrial artery fulfill this criterion.

For the coronary sinus and anterior cardiac veins to function as retrograde myocardial perfusion channels, a filling gradient from the right atrial cavity to the coronary arterial or venous system is necessary. Because of the small increase in right atrial pressure associated with complete coronary occlusion in our study, and the low peripheral coronary arterial
pressure, the possibility of retrograde flow through the coronary sinus as the source of the luminal blood in ventricles, particularly the left, had to be considered. However, occlusion of the coronary sinus ostium did not eliminate this, and it is unlikely that retrograde coronary sinus perfusion was responsible for the appearance of myocardial luminal blood in this or the other groups. The role of the anterior cardiac veins is more difficult to interpret, since the right coronary artery was ligated in all studies except those with right ventricular hypertension, and studies with occlusion of these veins were not attempted, since isolation of all these vessels is virtually impossible. (20).

It seems probable that the anterior cardiac veins served as retrograde myocardial perfusion channels for the right atrium and ventricle in those animals with ligated right coronary arteries, even with a normal right atrial pressure, since the peripheral coronary pressure of this vessel would be expected to be low. (21). However, in the normal and right ventricular hypertensive groups where the right coronary artery was perfused, the peripheral right coronary artery pressure would be higher than the right atrial pressure and retrograde flow from anterior cardiac veins to coronary arteries would not be expected. Even in this case, however, retrograde anterior cardiac vein perfusion cannot be excluded, since filling gradients between the right atrium and the anterior cardiac venous system might have been present in all these studies and the types of vessels in which the labeled blood accumulated are not known.

The role of left anterior atrial artery, a branch of the left circumflex having an average of 50% of its drainage directly into the left atrium (5), must also be considered. This drainage is predominantly venous (22), so that retrograde perfusion through these channels as the source of left atrial accumulation of luminal blood is subject to the same conditions discussed in connection with the anterior cardiac veins and the right atrium and ventricle. The hemodynamic conditions for retrograde left atrial perfusion differ, however, in that the left coronary artery was perfused during all of the studies except those with total coronary occlusion so that a favorable filling gradient from left atrium to left anterior atrial artery was not possible even with coronary perfusion pressure lowered to the point of producing myocardial ischemia. Therefore, in all animals except those with total coronary occlusion, it is probable that luminal blood accumulated in the left atrium via left atrial venous drainage channels. Is, therefore, the left atrial venous system also the source of the luminal blood which appeared in the left ventricular myocardium of these studies? This seems likely, at least as the explanation for the increased amounts of left ventricular luminal blood found with total coronary occlusion. Here there is clearly a filling gradient between the left atrial cavity and the left coronary artery as a result of the low peripheral coronary pressure and the high left atrial pressure.

The last vessel with proved luminal drainage in the beating heart is the septal branch of the left coronary artery (4). Approximately 80% of the drainage of this vessel is into the right ventricular cavity and is probably venous. Consequently, the amount of luminal blood appearing in the septum was separated from that of the free wall of the left ventricle to determine whether these transventricular channels can function as a source of retrograde myocardial perfusion. As noted (Table 2) there was no difference between the septum and the left ventricular free wall in any of the groups studied. Additionally, enhancement of retrograde filling gradients by right ventricular hypertension coupled with lowering of left coronary artery perfusion pressure and left ventricular pressure did not cause a greater accumulation of luminal blood in the septum over that in the free wall.

The types of luminal vessels which have been discussed are drainage channels, and those associated with the left anterior atrial artery and the septal artery would be classified as thebesian veins. Other types of luminal communications with the cardiac cavities have been classified as arterioluminal and arterio-sinusoidal vessels (2), but no functional role
in the normally beating heart has been shown. Although post-mortem perfusion studies show direct communication of the coronary arteries with the ventricular cavities (23), presumably via these types of channels, it is not known whether this occurs in the normally beating heart. More important to our study, however, is the possibility of retrograde perfusion from the ventricular cavities via such communication. On the basis of hydraulic principles pertinent to both the normal and partially occluded coronary circulation, Wiggers has reasoned that blood flow from the ventricular lumen to the myocardium is unlikely (24). However, the possibility of accumulation of luminal blood in the inner layers of the ventricular myocardium by an ebb-and-flow into sinusoids has been suggested by Myers and Honig (9). Distribution of luminal blood between the endocardial and epicardial halves of the ventricular myocardium was studied in our experiments to determine if a concentration gradient decreasing from endocardium to epicardium was present under normal conditions, and whether it could be modified by different hemodynamic interventions. These studies showed no increase in luminal blood in the inner half of the myocardium over that in the epicardium except in the case of the hypertensive right ventricle; in fact, in the only animals in which the left ventricle showed significantly more luminal blood than normal—the coronary occlusion group—the endocardium contained less than the epicardium. These data are at variance with the findings of Myers and Honig (9). However, their study is complicated by the fact that the right coronary artery was patent and perfused with radioactive blood by phasic aortic pressure while the left coronary artery was perfused with non-radioactive blood at a constant pressure. Their assumption that right coronary flow contributed nothing to the amount and distribution of labeled systemic blood in the left ventricular myocardium under these circumstances remains unsupported. We chose to ligate or cannulate the right coronary artery to obviate this problem.

These studies were all performed in dogs with normal coronary arteries. The effect of chronic occlusive coronary disease on the magnitude, type, and effect of luminal collateral coronary flow remains to be determined.

Acknowledgments

The author is happy to acknowledge the assistance of A. Lawrence Gould and Thomas D. Downs, Department of Biometry, Case Western Reserve University, who designed the computer program and carried out the statistical analysis of the data of this study. The expert technical help of John Dattilo and Eugenia Bobo is gratefully acknowledged.

References

12. MYERS, W. W., AND HONIG, C. R.: Number and


Study of Luminal Coronary Collateral Circulation in the Beating Canine Heart
THOMAS W. MOIR

Circ Res. 1969;24:735-744
doi: 10.1161/01.RES.24.5.735

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1969 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circres.ahajournals.org/content/24/5/735

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

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

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