Phasic and Mean Blood Flow in the Canine Septal Artery and an Estimate of Systolic Resistance in Deep Myocardial Vessels

By Richard W. Eckstein, M.D., Thomas W. Moir, M.D., and Thomas E. Driscol, M.D.

Gregg and Green concluded that phasic patterns of coronary flow recorded from meters placed at the ostia of coronary arteries fail to indicate actual blood flow within the myocardium except during late systole and late diastole. They interpret the superficial arteries as in essence, elastic volume buffers imposed between flow meters and the myocardium. Three deviations of recorded flow curves from true myocardial flow may be predicted on theoretical grounds. 1. The magnitude of the sharp reversal of flow usually registered in early systole is underestimated because the blood expressed from deep layers of myocardium distends superficial vessels and is not metered. 2. The generally recorded mid-systolic forward flow is too high since the rise in aortic pressure results in further distention of large surface vessels and of vessels in superficial layers of myocardium where systolic extravascular compression is less than in deeper layers. This is recorded, however, as a forward flow through the meter. 3. Finally, the metered forward flow during early diastole is less than the true intramyocardial flow since blood from the distended superficial vessels flows into the relaxing myocardium without being metered.

In dogs the septal artery passes almost immediately into the muscular substance of the interventricular septum and therefore offers opportunity to evaluate these predictions critically. However, careful exploration of the exact course of this vessel revealed that it also has a superficial component, variable in length, which passes just beneath the endocardium of the right ventricular surface of the interventricular septum. Elevation of right ventricular pressure by pulmonary arterial constriction was chosen as a means of reducing the phasic flow characteristics which are postulated to result from the superficial component of coronary arteries.

The initial section of this report describes the normal phasic flow pattern in the canine septal artery and the alterations which result from elevation of right ventricular pressure. Because the results indicated that mid-systolic septal flow was essentially blocked by this maneuver, experiments were designed to measure the mid-systolic septal resistance during elevation of right ventricular systolic pressure as a possible estimate of intramyocardial pressure.

The concluding section presents experiments which aim to derive the fraction of common left coronary arterial inflow which enters the septal artery as a preliminary step to the definition of the septal drainage pathways as described in a separate paper.

Methods

SURGICAL PREPARATION

Mongrel dogs weighing from 15 to 22 kg were anesthetized with 30 mg of morphine and 25 to 30 mg/kg of sodium pentobarbital. Respiration was provided through an intratracheal tube with room air under intermittent positive pressure. The left chest was opened between the 4th and 5th ribs and the septal artery was isolated. In three in-
MID-SYSTOLIC SEPTAL ARTERIAL RESISTANCE

Phasic septal arterial inflow was recorded with an orifice meter as previously described, but in these experiments the perfusion source was a chamber in which pressure was manually controlled and which was filled with blood from the aorta by means of a Sigma motor pump. The exact perfusion pressure was recorded from a Statham gauge connected between the meter and the septal artery. In order to attain the highest possible right ventricular pressures, the right coronary artery was cannulated and perfused at above aortic pressures directly from the pressure chamber. Right and left ventricular or aortic pressures were also recorded. Continuous phasic flow and pressure records were made while perfusion pressure was slowly lowered in order to produce zero mid-systolic septal arterial flow under conditions of normal and elevated right ventricular pressures. The simultaneous pressures in the right and left ventricles and septal artery were measured in mid-systole in the particular canine cycles in which mid-systolic septal flow was zero. The hearts were removed after death and after brief flushing with saline the septal arteries were injected with dilute black India ink at a pressure of 100 mm Hg. The right and left walls of the interventricular septa were observed through incisions in the ventricles. The right and left superficial veins were also inspected for the appearance of ink.

MEAN SEPTAL ARTERIAL INFLOW

The septal artery was cannulated as previously described. The common left artery was cannulated via the subclavian artery either with the special compression cannula or with a cannula ligated in place. Both arteries were perfused with blood brought from the aorta through a metal sound introduced via the left common carotid artery or from a pressure chamber as described above. Flow was metered with a Shipley rotameter designed to measure 200 cc/minute. A three-way stopcock was placed distal to the rotameter and arranged so that a one-quarter turn of the rotating element diverted the septal inflow from the rotameter through a bypass. The rotameter then metered common left flow minus septal flow. Perfusion pressures in the common left and septal arteries were not altered by the bypass. During continuous records the stopcock was rotated from the original position at five- to six-second intervals. The rotameter was calibrated during each experiment with the animal’s own blood. Flows with and without the bypass were averaged over each period and the differences (septal flow) calculated. A total of 189 determinations was made in seven dogs. In two of the animals septal and common left flows were measured before and after elevation of right ventricular pressure. Septal flows were calculated as a percent of total common left flow.

At the end of these experiments the common left
FLOW IN SEPTAL ARTERY

Figure 1 shows a normal phasic inflow pattern and the changes which result from a progressive rise in right ventricular pressure. The normal pattern in (A) shows a decline in forward flow at the onset of isometric ventricular systole which is converted into a reversal of flow. The maximum rate of backflow of 32 cc/min is attained just after aortic pressure begins to rise. There is a definite mid-systolic forward flow of 25 cc/min which decreases slightly during protodiastole. Forward flow increases quickly to 101 cc/min during isometric relaxation. This is followed by a rapid decline to reach a rate of 55 cc/min at the end of diastole. Figure 1B shows the flow changes induced by elevation of systolic right ventricular pressure to 45 mm Hg. The exaggeration of early systolic backflow and early diastolic forward flow and the decrease in mid-systolic forward flow is clear. The effects of further increases in right ventricular systolic pressure are evident in figure 1 (C and D). It should be noted that mid-systolic forward flow is absent in (D) in the presence of a lower aortic pressure than in the control. Other records (not shown) display similar patterns even though aortic pressures were maintained by constriction of the thoracic aorta. Figure 1 (E, F, G, H) shows records from a different animal in which a pulsus alternans developed at high right ventricular pressure (H). Although the alterations in systolic flow in (F) and (G) are minor, those in diastole are apparent. The weak beat in (H) shows an absence of systolic forward flow. The more forceful beat resulted in a brief forward flow during the early decline of right ventricular pressure.

Table 1 shows the volume of flow in systole, diastole, and during the backflow phase as
calculated from single heart beats. Elevation of right ventricular pressure results in a large increase in systolic backflow, accompanied by an increase in diastolic forward flow. The total stroke flow and flow per minute remain approximately the same.

The septal arterial pressure pulses registered after their decline to stable values following occlusion of the septal artery ranged from 37/3 mm Hg to 75/24 mm Hg with an average of 55/10 mm Hg. Elevation of right ventricular pressure resulted in a large increase in the peripheral septal arterial pressure particularly in systole. For example, in Exp. P. F. 43 (records not shown) the peripheral septal pressure was 72/8 mm Hg at a right ventricular pressure of 24/0 mm Hg and an aortic pressure of 133/104 mm Hg. Elevation of right ventricular pressure to 90/5 mm Hg raised the peripheral septal arterial pressure to 105/13 mm Hg even though aortic pressure fell to 127/98 mm Hg. In each of nine determinations systolic septal pressure exceeded the elevated systolic right ventricular pressure, but failed to equal systolic aortic pressure.

**TABLE 1**

*Effect of Elevation of Right Ventricular Pressure on Systolic and Diastolic Septal Flow*

<table>
<thead>
<tr>
<th>Exp. no.</th>
<th>Aortic pressure mm Hg</th>
<th>Right ventricular pressure mm Hg</th>
<th>Heart rate</th>
<th>Stroke* systolic flow cc</th>
<th>Stroke diastolic flow cc</th>
<th>Stroke backflow cc</th>
<th>Stroke total cc</th>
<th>Minute total cc</th>
</tr>
</thead>
<tbody>
<tr>
<td>P.F. 22A</td>
<td>118/91</td>
<td>23/5</td>
<td>176</td>
<td>-0.092</td>
<td>0.071</td>
<td>0.010</td>
<td>0.081</td>
<td>14.2</td>
</tr>
<tr>
<td>P.F. 22B</td>
<td>120/95</td>
<td>85/12</td>
<td>166</td>
<td>-0.548</td>
<td>0.144</td>
<td>0.0578</td>
<td>0.091</td>
<td>14.9</td>
</tr>
</tbody>
</table>

*Systolic forward flow minus backflow.*
systolic septal flow is slightly less than double the right systolic ventricular pressure. This same relationship does not apply to the determinations when right ventricular pressures were normal.

Observations of the right and left ventricular cavities during the postmortem septal arterial injections with black India ink showed copious flow from multiple exits on the right ventricular surface of the interventricular septum. Extremely small amounts drained from the left ventricular surface of the septum. After a few seconds small quantities of ink appeared in some superficial veins over the right and left ventricles.

**MEAN SEPTAL ARTERIAL INFLOW**

Table 3 shows the average values and the ranges of common left arterial and septal inflow as measured in 189 determinations on seven dogs. The table indicates that an average of 16.0% of common left inflow enters the septal artery with a range of 11.4% to 21.1%. The calculated average total common left flow is 73.8 cc/min/100 g in four dogs and is similar to the average septal flow of 83.2 cc/min/100 g in the total of seven dogs. Calculations show that in the four hearts in which the un.injected perfused septum was weighed the septum comprised 9.4% of the total heart weight and 11.8% of the weight of muscle perfused by the total common left coronary artery, while in the three hearts in which the injected perfused septum was weighed it accounted for 11.3% of the total heart weight. The average weight of the septum obtained by the two methods is 10.3% of the total heart weight.

In two experiments in which septal and
<table>
<thead>
<tr>
<th>Exp. no.</th>
<th>Heart wt g (g)</th>
<th>Common left &amp; perfused septum wt g (g)</th>
<th>Septal flow avg (range) cc/min</th>
<th>Septal flow % of common left</th>
<th>Aortic pressure (range) mm Hg</th>
<th>Perfusion source</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>166.5</td>
<td>231.5* (69-74)</td>
<td>14.5 (72-116)</td>
<td>15.8* (10-14)</td>
<td>119.5 (95-132)</td>
<td>Aorta</td>
</tr>
<tr>
<td>2</td>
<td>165.5</td>
<td>68.5 (54-89)</td>
<td>6.3 (6-20)</td>
<td>11.6* (10-14)</td>
<td>128.2 (90-108)</td>
<td>Aorta</td>
</tr>
<tr>
<td>3</td>
<td>165.5</td>
<td>143.5 (91-104)</td>
<td>9.6 (5-14)</td>
<td>9.9* (5-14)</td>
<td>102.6 (76-111)</td>
<td>Aorta</td>
</tr>
<tr>
<td>4</td>
<td>145.0</td>
<td>129.5* (72-116)</td>
<td>16.2 (7-16)</td>
<td>10.2 (7-16)</td>
<td>92 (69-98)</td>
<td>Aorta</td>
</tr>
<tr>
<td>5</td>
<td>166.2</td>
<td>63.3 (37-99)</td>
<td>12.7 (8-20)</td>
<td>10.4 (59-115)</td>
<td>89 (80-108)</td>
<td>Pump</td>
</tr>
<tr>
<td>6</td>
<td>173</td>
<td>119 (104-124)</td>
<td>11.8 (10-14)</td>
<td>11.4 (103-111)</td>
<td>115 (108-115)</td>
<td>Pump</td>
</tr>
<tr>
<td>7</td>
<td>185.5</td>
<td>20.5 (85-139)</td>
<td>11.0 (8-18)</td>
<td>15.6 (78-108)</td>
<td>129 (107-124)</td>
<td>Aorta</td>
</tr>
</tbody>
</table>

### Summary of Pertinent Data from Seven Experiments

- Unstained.
- *Stained.

Based on 7 expts. Expts. 7 and 7a are averaged together. Ranges are shown in parentheses.
common left perfusion and aortic pressures were maintained at control levels, elevation of right ventricular pressure from a control of 15/0 mm Hg to a high level of 90/5 mm Hg produced an average increase in common left inflow from 106 to 115 cc/min and an average increase in septal flow from 15 to 16.7 cc/min. Septal flow was 13.8% and 14.3% of common left flow in the two conditions respectively.

Discussion
The use of orifice meters for metering phasic flows has been criticized from two points of view. The first suggests that the pressure difference over the orifice during rapid flow alterations actually results from the flow alterations themselves and therefore gives a retarded record of changes in velocity. The second criticism applies to the use of orifice meters in large elastic vessels where a phase shift between pressure and flow has been found. It is believed that the first objection does not invalidate the conclusions to be drawn from the results of these experiments since minute delays in recorded velocity curves have little, if any, relationship to the large quantitative changes resulting from elevations of right ventricular pressure. The second objection requires consideration. If phase shifts in coronary arteries are as pronounced as they are within the aorta, large errors in coronary velocity curves as recorded from orifice meters might result. However, recent experiments with electromagnetic flow meters on coronary arteries have yielded coronary velocity curves essentially the same as those measured from orifice meters. Of particular importance is the finding that electromagnetically recorded left coronary inflow curves often reveal brief periods of early systolic backflow similar to that previously recorded with orifice meters. It is believed, however, on the basis of our own experience and that of Remington, that placement of the source cannula at the level of the coronary ostia is of critical importance in preventing errors in the flow curve resulting from the delays in transmission time which exist in more distal sites in the aorta.

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The results of these experiments on the pattern of phasic flow in the septal artery are believed to substantiate the hypothesis set forth as to the alterations in metered inflow which result from changes in distension of the superficial components of coronary arteries. When the septal artery was rendered functionally less superficial by elevation of right ventricular pressure, early systolic backflow increased, mid-systolic forward flow was almost abolished, and the early diastolic flow was augmented.

These experiments were originally designed in an attempt to define the flow pattern in deep myocardial layers commonly referred to as intramyocardial flow. This term requires more exact definition; it may be construed as intramyocardial arterial, arteriolar, or even capillary flow. It is clear that the patterns recorded in the septal artery in these experiments can reasonably be applied only to myocardial arterial flow. The large systolic backflows recorded in the present experiments occur during a marked systolic accentuation in venous outflow. This suggests that at some location there is a reversal of direction from which point forward flow continues distally and backward flow occurs centrally, at least during the early phase of ventricular systole. Experiments on models have shown that from 80% to 100% of externally applied pressure is transferred through the walls of distended elastic tubes and is added to the original distending pressure. The application of this observation suggests that the extravascular pressure in early systole may be transferred through the vessel walls and thus maintain intravascular pressure gradients favorable to forward flow into capillaries and/or veins although a backflow is recorded at coronary orifices at this time. In later systole, when tension in vascular walls is low due to displacement of blood forward and backward, intravascular pressure gradients would be so small that no systolic flow should occur.

It is believed that the large early diastolic flow recorded in these experiments after ele-
vation of right ventricular pressure chiefly represents filling of the main septal trunk during diminishing vascular compression and therefore probably is not representative of actual arteriolar flow. The actual stroke increase in diastolic septal flow after elevation of right ventricular pressure ranged from 0.01 to 0.07 cc while the increase in backflow ranged from 0.03 to 0.07 cc. These volumes could readily be accommodated by the main septal trunk and its larger branches and therefore no estimate of early diastolic arteriolar or capillary flow can be made.

The systolic peripheral septal arterial pressure of 55/10 mm Hg is in contrast to that of 30/20 mm Hg previously registered in other branches of the common left coronary artery. The higher systolic septal arterial pressure probably results from the more extensive systolic compression on the proximal vessel not only by the contracting interventricular septal musculature, but also from the force exerted by right ventricular systolic pressure. The striking increase in systolic septal arterial pressure induced by augmentation of right ventricular systolic pressure occurs even though the vessel is poorly filled as is indicated by the low diastolic pressure of 10 mm Hg. Presumably this elevation of septal arterial pressure is due to the decrease in the systolic expansion of the superficial component since the lower aortic pressure in the experiment cited would not be likely to provide additional collateral flow. The slight rise in diastolic septal pressure probably results from the similar rise in right ventricular diastolic pressure. Although no quantitative estimate of intramyocardial pressure can be made from these data they do suggest that the distortion which results from the interposition of superficial coronary arteries between the myocardium and metering devices must be large indeed.

The experiments in which measurements were made of the perfusion pressure required to abolish mid-systolic flow were attempted in an effort to make an estimate of intramyocardial pressure by a method which does not deform the architecture of the myocardium. In each case the measurements were made after the peak of backflow had occurred and when the contracting muscle should have reduced or abolished the tension in the vascular walls. Although the pressures recorded here are generally in excess of those recorded by Laszt, it is noteworthy that by both techniques they are considerably in excess of systolic aortic pressures.

The authors are unable to offer proof that the measured perfusion pressures represent true intramyocardial pressure. However, the evidence strongly suggests that resistance to coronary flow in deep myocardial layers in mid-systole is in excess of that which can be overcome by aortic systolic pressure.

The technique employed to determine the fraction of common left arterial inflow which enters the septal artery was chosen so as to maintain normal pressure relationships between the septal and surrounding arteries during septal flow measurements thereby excluding errors due to flow through interarterial collateral vessels. The average value of 16.0% of the common left flow which enters the septal artery is of importance in the identification of the exact venous channels through which the extra coronary sinus fraction of common left coronary flow drains, particularly in view of the previous indications of absence of substantial septal drainage via either the coronary sinus or anterior cardiac veins. The results of the injection studies reported here indicate the existence of anatomical pathways which permit considerable direct Thebesian drainage into the right ventricle. The identification of the drainage pathway of the septal artery in the beating heart is considered in a companion paper.

**Summary**

Phasic flow in the canine septal artery was recorded with an orifice meter supplied by pulsatile aortic pressure. Flow patterns were recorded when right ventricular pressure was normal and contrasted with those following elevation of right ventricular pressure in
FLOW IN SEPTAL ARTERY

order to minimize the dynamic effect of the superficial component of the artery. Results indicate that elevation of right ventricular pressure increases the early systolic backflow, largely prevents mid-systolic forward flow, and accentuates early diastolic flow. It is suggested that this pattern does not represent that in arterioles in deep layers of the myocardium.

In other experiments the septal artery was perfused through an orifice meter at variable pressures to determine the pressure required to negate mid-systolic flow at high right ventricular pressures. The results indicate that pressures considerably above aortic or right ventricular systolic pressures are required to generate forward mid-systolic septal flow. The mean septal flow was quantitated in terms of total common left inflow. It was found that an average of 16.0% of common left flow enters the septal artery. The significance of this has been discussed in terms of its relationship to the venous drainage of common left coronary arterial inflow.

Acknowledgment

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