The Relationship between Myocardial Blood Flow and Contraction by Myocardial Layer in the Canine Left Ventricle during Ischemia

WILLIAM  S. WEINTRAUB, SHIGEHIKO HATTORI, JAI B. AGARWAL, MONTY M. BODENHEIMER, VIDYA S. BANKA, AND RICHARD H. HELFANT

SUMMARY We investigated the relationship between regional myocardial blood flow (MBF) and segmental shortening in 17 open-chest dogs. The left anterior descending coronary artery was cannulated and perfused from the left carotid while measuring perfusion pressure. Graded occlusion was produced by a screw clamp. Extent of occlusion was monitored by perfusion pressure. Percent systolic shortening (%AL) was measured using ultrasonic crystals implanted in normal and ischemic endocardium and ischemic epicardium. MBF was measured in ischemic and normal endocardium and epicardium with tracer microspheres. Dogs underwent 1 to 4 grades of coronary stenosis. Myocardial blood flow both to endocardium and epicardium was found to be linearly related to diastolic perfusion pressure below 50 mmHg (r = 0.803 and 0.748, respectively). Normalized %AL (N%AL) was best related to fraction normal zone MBF in the endocardium by the sigmoidal equation, 

$$N\%AL = e^{-0.01MBF^{4.2}/1 + e^{0.01MBF^{4.2}}}.$$

%AL was only weakly related to myocardial blood flow in the epicardium (r = 0.584), and, in four dogs, %AL was zero in the epicardium despite normal regional blood flow. However, %AL in the epicardium was linearly related to %AL in the endocardium (r = 0.78). Thus, endocardial wall motion is related to MBF by a sigmoidal relationship while epicardial wall motion is tethered to endocardial wall motion.


CORONARY artery obstruction sufficiently severe to reduce regional myocardial blood flow results in contraction abnormalities in the subserved zone (Tennant and Wiggers, 1935; Case, 1954; Herman et al., 1967; Theroux et al., 1974). However, the precise relationship between myocardial blood flow and contraction in different myocardial layers is less clear. Within a relatively wide range of perfusion pressures, autoregulation can maintain coronary flow (Rubio and Berne, 1975), but below a diastolic perfusion pressure of approximately 50 mmHg, flow becomes pressure dependent Griggs and Nalsamura, 1968; Rubio and Berne, 1975. In addition, several studies have shown that blood flow to the subendocardium falls to a greater extent during coronary occlusion than that to the subepicardium (Buckberg et al., 1972; Rubio and Berne, 1975; Rivas et al., 1976; Hoffman and Beckberg, 1977).

Several studies have shown that myocardial contractile force or shortening is related to blood flow (Wyatt et al., 1975; Banka et al., 1977; Vatner et al., 1979); thus, myocardial contraction is initially maintained until coronary flow is reduced significantly, after which there is a progressive decrease in transmural myocardial contraction with progressive decreases in coronary flow. Forman et al. (1973) and Downey (1976), using strain gauges, have suggested that epicardial contractile force is maintained to a greater extent during partial coronary occlusion than endocardial force. However, recent preliminary studies have suggested that epicardial shortening is determined primarily by endocardial shortening or that the epicardium is "tethered" to the endocardium (Gallagher et al., 1978; Genain, et al., 1979).

To define better the relationship between myocardial blood flow and contraction by myocardial layer, we simultaneously measured myocardial blood flow with radioactive microspheres and myocardial contraction with ultrasonic crystals during graded coronary occlusion.

Methods

Seventeen mongrel dogs weighing 25-32 kg were anesthetized with intravenous sodium pentobarbital (50 mg/kg) and ventilated with room air using a Harvard respirator. Blood gases were monitored intermittently, and blood pH maintained between 7.35 and 7.45. Standard electrocardiographic lead II was monitored continuously throughout each experiment. Pressures were monitored with Statham P23Db transducers.

Catheters (#8F) were placed in the right femoral vein and right femoral artery, the latter advanced to the aorta for monitoring arterial pressure. An-
other stiff catheter with a 2-mm internal diameter was placed in the left femoral artery for blood withdrawal. A thoracotomy was performed in the 5th left intercostal space, and the heart was supported in a pericardial cradle. A segment of the left anterior descending (LAD) about 1 cm long was isolated. A sidehole catheter (#7 French) was inserted into the left atrial appendage for the injection of microspheres. A stiff 14-gauge catheter 10 cm in length was placed in the left ventricle through the apex for measurement of left ventricular pressure and dP/dt.

Three pairs of 2-mm ultrasonic crystals were placed one pair each in the nonischemic endocardium and ischemic endocardium and epicardium (Fig. 1). Endocardial crystals were inserted into the inner third of the myocardium through small stab wounds perpendicular to the long axis of the left ventricle. Epicardial crystals were inserted through stab wounds parallel to the direction of epicardial fibers at an angle of approximately 90° to the short axis of the ventricle.

The motion of the ultrasonic crystals was monitored with a circuit using the design of Patrick et al. (1974). Hemodynamic data and myocardial shortening were recorded on an Electronics for Medicine VR-16 recorder.

The left anterior descending coronary artery was ligated proximally, an arteriotomy performed and a 14-gauge blunt-tipped metal cannula introduced and tied securely in place. Care was taken to avoid occluding any branch vessels and to avoid kinking the left anterior descending. The left anterior descending was perfused continuously from the left common carotid artery through a plastic cannula-tion system (Fig. 1) from which all bubbles had been carefully removed. The time from ligation of the left anterior descending until reperfusion through the cannula averaged about 1 minute and was always less than 2 minutes. Perfusion pressure was measured in the cannulation system between the point of partial occlusion (see below) and the cannula tip. Initial pressure monitored in the cannulation system was identical to pressure in the aorta in all experiments, with only minor differences in waveform and no phase lag.

In five dogs, a 2-mm cannulating type flow probe (MIC 4020) was included in the cannulation tubing and flow monitored with an electromagnetic flow meter (Micron Instruments). In these five dogs, a 10-second period of total occlusion of the cannulation tubing was followed by more than 100% hyperemia, indicating that the cannulation system did not produce a hemodynamically significant stenosis and that some autoregulation could be maintained with this preparation. In addition, in one dog the pressure was measured at the cannula tip and aortic, cannula system, and cannula tip pressures were identical at flows up to 100 ml/min. The cannula system was further checked in vitro with a Harvard pump producing graded flows. Pressure was monitored in the proximal cannulation system and at the cannula tip. We were unable to demonstrate any pressure drop with flows from 1.1 ml/min up to 46 ml/min.

Microspheres were sonicated in an ultrasonic bath for at least 15 minutes and shaken in a vortex whirler. The microspheres (1.5 to 3 million) were diluted to a volume of 10 ml and injected into the left atrium over a period of 20–30 seconds, followed by a 10-ml saline flush over 20–30 seconds. Blood was collected from the aortic catheter with a Harvard pump at a rate of 7.75 ml/min beginning before microsphere injection and continuing for 1 minute after completion of the flush. $^{125}$I, $^{153}$Ce, $^{51}$Cr, $^{85}$Sr, $^{90}$Nb, and $^{57}$Co 9-μm microspheres (3M) were used.

After the dog had been instrumented, but before insertion of the cannula into the left anterior descending, hemodynamics and myocardial contraction patterns were recorded. In all dogs, segment shortening returned to precannulation values in less than 3 minutes. After cannulation, a control set of microspheres was injected. Microspheres were given during one to four sequentially increasing grades of coronary occlusion, each lasting 10 minutes. Occlusion was produced by partially occluding the cannulation tubing with a screw clamp and monitoring perfusion pressure. During the first grade of occlusion, minimum diastolic perfusion pressure was approximately 55 mm Hg. Perfusion pressure was lowered by approximately 10 mm Hg.
at each subsequent grade of occlusion. The last set of recordings was made and a last set of microspheres given when end-systolic shortening was replaced by end-systolic bulging.

After the final microsphere injection, crystal motion was calibrated, the crystals removed, and before the dog was killed Evans blue was injected by hand with a syringe through the coronary cannula with sufficient pressure to stain the ischemic myocardium but insufficient to fill the epicardial collaterals. The heart then was excised, inspected for the position of the crystal holes, washed with cold water, dried, and stuffed with gauze. The heart was wrapped in industrial strength aluminum foil (to prevent freezer burn) and frozen for 24-72 hours. The heart was cut while still frozen to facilitate sectioning. The right ventricle was removed and a 2- to 3-cm ring of left ventricle was cut out with the path of the cut perpendicular to the blue line separating normal from ischemic tissue. Before each cut the knife was cleaned to prevent contamination of ischemic tissue with microspheres from normal tissue. A sample of normal left ventricle was cut from the ring at a wide distance from the blue line. The ischemic tissue was separated from normal tissue at the blue line, care being taken to include all or almost all blue tissue on the ischemic side. A 1.0-cm sample was taken on the normal side of the blue line which was labeled “border normal.” Sequential 1.0-cm samples were then taken on the ischemic side and labeled “border ischemic” and “central ischemic.” All of the ischemic zone crystals were located in the central blue zone.

All samples were divided into subendocardial, mid-myocardial, and subepicardial zones. Samples were weighed and counted along with the blood and pure isotopes in standards in a Beckman Gamma 8000 well counter for 10 minutes each. Myocardial blood flow in ml per gram was calculated using the equation

$$MBF = \frac{AX7.75/BA}{W}$$

where A is specific tissue activity in counts per minute, 7.75 is the withdrawal rate in ml/min, BA is blood activity in counts/min, and W is the sample weight in grams (Heymann, et al., 1977). For purposes of comparison, blood flow was normalized by dividing each blood flow measurement by the transmural average blood flow to the normal zone for each isolate. Unless otherwise noted, MBF refers to normalized blood flow.

End-diastolic length was identified at the initial increase in $dP/dt$ (Fig. 2) and end-systolic length at the nadir of $dP/dt$ (Theroux et al., 1977). Percent shortening ($\%AL$) was calculated as the difference between the end-diastolic length and end-systolic length divided by the end-diastolic length multiplied by 100. As shown in Figure 2, percent shortening was normalized to the post-cannulation control period ($N\%AL$). For all points with end-systolic bulging, $\%AL$ was set equal to zero.

Data are expressed as mean ± SEM. Paired groups of data were compared for significant differences by the paired t-test. Groups of paired data were analyzed for a linear relationship by linear regression by the method of least squares. For data with multiple points in each experiment, a repeated measures analysis was performed to test for significance of linearity (Edwards, 1976). Data were analyzed for a sigmoidal relationship by the general equation

$$y = \frac{e^{bx+a}}{1 + e^{bx+a}}.$$  

We define $y' = \ln(y/1-y)$ (logit transformation). Then it can be shown that $y' = bx + a$. By performing linear regression of $y'$ on $x$, we can solve for “a” and “b” and obtain a correlation coefficient (Colton 1974). To test for a difference between two correlation coefficients, a normal curve approximation test as described by Croxton (1953) was used. For all statistical tests, $P < 0.05$ was considered significant.

Methodological Limitations

In this study, tracer microspheres were used to measure myocardial blood flow and ultrasonic crystals to assess myocardial shortening. Microspheres have become standard experimental technique for measuring regional blood flow. However, the technique is potentially subject to errors, the most important of which are the lack of even mixing of microspheres and of sampling tissue with an insufficient number of microspheres particularly in the ischemic zone. Microspheres are distributed in a Poissonian fashion and a tissue sample must have 1576 microspheres for an accuracy of 5% with 95% confidence levels (Buckberg et al., 1971; Heyman et al., 1977). This is of great importance in the central ischemic zone where flow is severely reduced. This problem can be minimized by taking tissue samples of adequate size such as was done in the present study. In addition, it has been suggested that there
is a 1–3% non-entrapment rate in the myocardium with the use of nine micron microspheres (Buckberg et al., 1971, Fan et al. 1979). With 15-μm microspheres, fewer microspheres are injected, although nonentrapment should no longer be a problem. Evenness of mixing can be assured with adequate sonication and careful left atrial injections, as was done in this study. The problems therefore were minimized in this study.

The accuracy of ultrasonic crystals is harder to assess in large part because there is no independent method of sufficient accuracy to compare to crystal motion (Theroux et al., 1974). Insertion of the crystals is traumatic to the heart, but crystal motion is repeatable and generally has been accepted as the best measure of segmental wall motion. In our laboratory, the epicardial crystals are placed near the surface of the heart and the top of the crystals can be seen during the experiment. When the heart is cut at the end of the experiment, the endocardial crystal hole tracts reach the inner one-third of myocardium. The ischemic zone tissue sampled in the present study was at least 1 full cm inside the ischemic zone as assessed by Evans blue, and this tissue sample contained the crystal tracts. Thus, ischemic zone microspheres counts and crystal motion should reflect ischemic zone blood flow and shortening, respectively.

This study was performed in an open-chest, open-pericardium model, and this may have some effect on blood flow and shortening, but the preparation permitted a more accurate creation of and monitoring of the partial occlusion. A carotid-to-left anterior descending cannulation technique and a screw clamp were used to create partial occlusion to facilitate an accurate measurement of perfusion pressure reduction. An in-line electromagnetic flow probe was used since it is more accurate and less "noisy" than a flow probe placed on a coronary artery. The adequacy of our cannulation system was demonstrated by the rapid return of segmental shortening to precannulation values and by greater than 100% reactive hyperemia after a 10-second total occlusion. Although hyperemic responses of 300–400% are reported, 100% reactive hyperemia demonstrates some autoregulatory reserve.

Results

Hemodynamics

Aortic pressure initially was 108 ± 4 mm Hg (n = 17) systolic and 86 ± 4 mm Hg (n = 17) diastolic, while coronary perfusion pressure measured in the cannulation system was 108 ± 4 mm Hg (n = 17) systolic and 85 ± 4 mm Hg (n = 17) diastolic. At the end of the experiment, aortic pressure was 92 ± 4 mm Hg (n = 17) systolic and 70 ± 4 mm Hg (n = 17) diastolic. Left ventricular end-diastolic pressure was 4.2 ± 0.4 mm Hg (n = 17) initially and 5.2 ± 0.4 mm Hg (n = 17) at the end of the study. Heart rate was initially 152 ± 5 (n = 17) and 140 ± 7 (n = 17) at the time of the final blood flow measurement.

Absolute Blood Flow and Myocardial Shortening

As seen in Table 1, after cannulation, pre-occlusion %AL in the normal zone endocardium was 18.4 ± 1.4 (n = 17) and was 20.2 ± 1.9 (n = 17) by the end of the study (not significant). Initial myocardial blood flow to normal zone endocardium was 1.16 ± 0.14 ml/g per min (n = 17) and to the epicardium 1.11 ± 0.15 ml/g per min (n = 17, not significant). Similarly, during the most severe grade of occlusion, blood flow to the normal zone endocardium was 1.14 ± 0.15 ml/g per min (n = 17) and to the epicardium 1.27 ± 0.28 ml/g per min (n = 17, not significant). %AL after cannulation, pre-occlusion in the ischemic zone endocardium was 19.8 ± 1.9 (n = 17) and was significantly greater than initial %AL in the ischemic zone epicardium 12.3 ± 1.9 (n = 17, P < 0.05). Control ischemic zone shortening was not significantly different from that in the normal zone.

The Relationship between Perfusion Pressure and Flow

Regional blood flow (expressed as a fraction of normal zone flow, MBF) to the subendocardium and subepicardium were correlated with diastolic perfusion pressure. Figure 3 shows the relationship between perfusion pressure and subendocardial flow in each experiment. Figure 4 displays the regression line for endocardial flow for all 17 experiments. There was a linear relationship between diastolic perfusion pressure and MBF in the subendocardium (n = 57) (r = 0.083, P < 0.0001).

Figure 5 displays the relationship between diastolic perfusion pressure and flow in each experiment. Figure 6 shows the regression line for MBF to the subepicardium. Subepicardial flow is linearly related to perfusion pressure (n = 56) (r = 0.748, P < 0.0001). However, above 30 mm Hg, epicardial flow is essentially normal and there is a considerable scatter. The intercept of zero blood flow as 8 mm Hg in the endocardium and 3 mm Hg in the epicardium.

Relationship between Myocardial Perfusion and Shortening

Figure 7 shows a set of recordings from a typical experiment illustrating the effect of progressive re-

<table>
<thead>
<tr>
<th>TABLE 1 Myocardial Function and Blood Flow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonischemic</td>
</tr>
<tr>
<td>zone %AL</td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td>Final</td>
</tr>
</tbody>
</table>
duction in diastolic perfusion pressure on MBF and segment shortening normalized to the preocclusion value. Grade 1 partial occlusion reduced perfusion pressure from 130/105 to 105/60 mm Hg. In the endocardium, MBF fell from 0.962 to 0.831 while N%ΔL was unchanged. At grade 2, perfusion pressure was 95/45 mm Hg endocardial MBF fell to 0.735 and N%ΔL 0.061. Thus, the decrease in endocardial perfusion pressure caused a correspond-

FIGURE 3 Myocardial blood flow (fraction normal zone) to the ischemic subendocardium plotted as a function of perfusion pressure in each experiment.

ing and progressive fall in endocardial perfusion and shortening with most of the decrease in shortening occurring on changing from grade 3 to grade 4 occlusion, whereas the fall in MBF between grades 3 and 4 was small.

In contrast, during grade 1 occlusion in the epicardium MBF was essentially unchanged, but N%ΔL fell to 0.531 of preocclusion. At grades 2 and 3, epicardial MBF was still unchanged; however, N%ΔL fell to 0.284 at grade 2 and shortening was abolished at grade 3. Finally, at grade 4, epicardial MBF fell to 0.842. Thus, in the epicardium, perfusion changed little until the final grade of occlusion while shortening progressively decreased. During each grade of occlusion, MBF to the epicardium was larger than MBF to the endocardium. In contrast, shortening in the epicardium actually fell faster than shortening in the endocardium.

In the nonischemic zone, endocardial N%ΔL varied from 1 to 1.19, while MBF, which was 0.979 preocclusion, similarly varied insignificantly from 0.845 to 0.985. MBF to the normal epicardium varied from 1.09 to 0.994 during the control period and the four grades of occlusion. Heart rates and aortic pressure remained constant. Left ventricular end-diastolic pressure remained constant at 4 mm Hg until, with the final grade of occlusion, it rose to 5 mm Hg.
MYOCARDIAL BLOOD FLOW AND CONTRACTION RELATIONSHIPS/Weintraub et al. 435

Figure 6 shows the relationship between myocardial blood flow and perfusion pressure in the subepicardium. As seen in Figure 6, regression line of myocardial blood flow on perfusion pressure in the subepicardium.

Figure 7 depicts tracings from a typical experiment. Panel one is control. Panels two through five show increasing grades of coronary occlusion. The next three tracings represent ultrasonic crystal motion in the ischemic zone endocardium, ischemic epicardium, and normal endocardium. Then dP/dt, LVEDP, LV pressure, aortic pressure and perfusion pressure. Blood flow and shortening in the ischemic endocardium both fell with increasing degrees of ischemia. Shortening decreased sequentially in the ischemic epicardium but blood flow did not fall until partial occlusion 4 (panel 5). Normal zone shortening and blood flow were unchanged during the course of the experiment. See text for details. ENDO = endocardium; EPI = epicardium, dP/dt = first derivative of left ventricular pressure; LVEDP = left ventricular end diastolic pressure; LVP = left ventricular pressure; AoP = aortic pressure.

Figure 8 demonstrates the relationship between endocardial myocardial flow and segmental shortening in each experiment, and Figure 9 shows the relationship between endocardial shortening and flow for all 17 experiments. As seen in Figure 9, endocardial shortening did not change until flow had fallen significantly. Further decrease in flow, shortening decreased rapidly such that %AL was frequently zero or paradoxical when myocardial flow, although reduced, was still measurable. This relationship can be viewed as sigmoidal and is described by the equation:

\[
N\%AL = e^{0.01 \text{MBF} - 4.03} / 1 + e^{0.01 \text{MBF} - 4.03}
\]

(n = 67) (r = 0.801, P < 0.0001).

In contrast, the relationship between epicardial contraction (Fig. 10) and flow was linearly correlated (n = 69) (r = 0.584, P < 0.0001) demonstrated considerable scatter. The scatter in the epicardium, was greater than in the endocardium (P < 0.001). Furthermore, contraction was abolished in the epicardium when regional blood flow was normal in four dogs and in eight dogs with a 50% flow reduction. This was not observed in the endocardium (Fig. 8).

The Relationship of Epicardial to Endocardial Flow and Contraction

As seen in Table 2, during each range of partial coronary occlusion, epicardial blood flow was greater than endocardial blood flow but there was no significant difference between endocardial and epicardial shortening. Furthermore, as shown in Figure 11, subepicardial wall motion was linearly related to subendocardial wall motion (n = 70) (r = 0.780, P = 0.001). However, the scatter is considerable, suggesting that there are multiple factors determining epicardial wall motion.

Discussion

Since the classic study of Tennant and Wiggers (1935), the intimate relationship between coronary
Myocardial blood flow and contraction has been recognized. During partial coronary occlusion, regional myocardial contraction deteriorates as blood flow is reduced. Wyatt et al. (1975) used mercury in Silastic segment length gauges and perfusion pressure to construct pressure-length loops and measured flow with an electromagnetic flow probe. They demonstrated a graded fall in ischemic zone function with more significant grades of occlusion. Banka et al. (1977) assessed epicardial tension with Walton-Brodie strain gauge arches and coronary flow with an electromagnetic flow probe and demonstrated a sigmoidal relationship between coronary flow and epicardial tension. However, the precise relationship between regional myocardial blood flow and contraction by myocardial layer has not been clearly defined previously. This is particularly important in view of the observation that, during graded coronary occlusion, endocardial flow is decreased to a greater extent than epicardial flow (Griggs and Nakamura, 1968; Hoffman and Buckberg, 1977). This was confirmed in the present study (Table 1), as was the finding that, as coronary perfusion pressure is reduced, flow initially is maintained; however, below a threshold of about 50 mm Hg in the endocardium and 30 mm Hg in the epicardium, flow becomes pressure dependent (Fig. 3–6) (Rubio and Berne, 1975; Guyton et al., 1977). Rouleau et al. (1979) similarly have shown that at any perfusion pressure epicardial flow was greater than endocardial flow. In addition, the finding that flow approaches zero at a higher pressure in the endocardium than in the epicardium suggests a difference in intramyocardial pressures. Our findings are also similar to those of Rouleau et al. (1979).

### The Relationship between Endocardial Contraction and Flow

Several studies have suggested that endocardial contraction is closely related to endocardial blood flow. Forman et al. (1973) measured myocardial tensions by layer with deeply and shallowly sutured strain gauges and correlated these measurements with myocardial blood flow measured with rubidium 86 and potassium 42. They found that endocardial tension and blood flow both decreased with partial coronary occlusion. Downey (1976) measured endocardial force with strain gauges soldered to pins impaled in the heart and noted a linear relationship between coronary blood flow measured with an electromagnetic flow meter and endocardial tension. Recent preliminary reports have measured flow with tracer microspheres and wall motion with ultrasonic crystals (Gallagher et al., 1978; Genain et al., 1979; Vatner et al., 1979).

In the present study, multiple levels of myocardial perfusion during graded coronary occlusion were evaluated and correlated with contraction by myocardial layer. The relationship between endocardial blood flow and shortening was found to be sigmoidal, being best described by Equation 1 (see Fig. 9).

Given the limitations of these techniques, this equation best describes the relationship between endocardial shortening and blood flow. Thus, a

### Table 2 Myocardial Blood Flow and Shortening

<table>
<thead>
<tr>
<th>Pressure range</th>
<th>n</th>
<th>Endo</th>
<th>Epi</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–19</td>
<td>15</td>
<td>0.10 ± 0.03</td>
<td>0.27 ± 0.07</td>
</tr>
<tr>
<td>20–29</td>
<td>10</td>
<td>0.28 ± 0.08</td>
<td>0.65 ± 0.16</td>
</tr>
<tr>
<td>30–39</td>
<td>10</td>
<td>0.45 ± 0.06</td>
<td>1.06 ± 0.09</td>
</tr>
<tr>
<td>40–50</td>
<td>14</td>
<td>0.69 ± 0.07</td>
<td>1.29 ± 0.06</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Normalized percent ΔL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pressure range</td>
</tr>
<tr>
<td>0–19</td>
</tr>
<tr>
<td>20–29</td>
</tr>
<tr>
<td>30–39</td>
</tr>
<tr>
<td>40–50</td>
</tr>
</tbody>
</table>
small decrease in myocardial blood flow did not influence contraction, but as flow fell by more than approximately 30%, segmental shortening decreased rapidly and was abolished at a finite flow greater than 0. For the purposes of this study, normalized %ΔL ranged from 0 to 1. If there was end-systolic bulging, %ΔL was set equal to 0. This approach focuses attention on the data points with graded reductions in segmental shortening rather than on points with end-systolic bulging.

The Relationship between Epicardial Contraction and Flow

The relationship between contraction and flow in the epicardium has been controversial. Forman et al. (1973) found that epicardial blood flow is related to epicardial tension, whereas Downey (1976) demonstrated a linear relationship between epicardial tension and flow meter-determined coronary blood flow. In contrast, the preliminary reports of Gallagher et al. (1978) and Genain et al. (1979) suggest a poor relationship between epicardial blood flow and myocardial shortening. In the present study, whereas a statistically significant correlation was found between shortening in the epicardium and the regional blood flow, there was considerable scatter (Fig. 10). Epicardial shortening was found to decrease significantly and sometimes to be abolished during partial occlusion despite normal regional epicardial flow. In addition, epicardial shortening decreased in tandem with endocardial shortening during coronary occlusion.

Implications

Thus, during graded coronary occlusion, the results of the present study demonstrate that shortening of the endocardium is related to endocardial myocardial blood flow. However, epicardial contraction appears more closely related to endocardial contraction than to epicardial blood flow. It is logical to hypothesize that motion of the epicardial layer will be determined by a complex series of variables. One of these would be the amount of tension it must generate to overcome the stress imposed on it during the course of systole. Thus, with normal inward endocardial contraction, epicardial stress is relatively low and a given amount of its own tension development will produce normal inward epicardial motion. Conversely, with reduced or absent inward endocardial movement or actual endocardial paradoxical expansion, the total amount of epicardial tension development will not result in inward epicardial motion. In addition, the intrinsic ability of the epicardial layer to generate tension or force will depend on the severity of epicardial ischemia. This in turn will depend in part on epicardial blood flow and perhaps other factors such as transmural metabolic abnormalities. Additional studies will be required to ascertain the precise nature of the relationship between regional blood flow and shortening to determine the relative importance of the various factors influencing epicardial shortening.

Acknowledgments

We wish to thank Joseph Lewandowski, Robert Krumm, and Janice Phillips for technical support and Kathleen McNair, Regina Harrison, and Catherine Breslin for secretarial assistance.

References

Genain C, Theroux P, Thouless C, Bouressa MG, Waters DD (1979) The interrelationships between function and flow in
the subendocardial and subepicardial regions of the left ventricle (abstr). Circulation 60 (suppl II): 28
Vatner S, Manders T, Baig H (1979) Correlation between ischemic induced reductions in regional myocardial blood flow and function in conscious dogs (abstr). Circulation 60: (suppl II): 29
The relationship between myocardial blood flow and contraction by myocardial layer in the canine left ventricle during ischemia.

W S Weintraub, S Hattori, J B Agarwal, M M Bodenheimer, V S Banka and R H Helfant

doi: 10.1161/01.RES.48.3.430

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 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/48/3/430

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/