Direct Measurement of Coronary Collateral Blood Flow in Conscious Dogs by an Electromagnetic Flowmeter

By Eric C. Elliot, Edward M. Khouri, Jerry A. Snow, and Donald E. Gregg

ABSTRACT

The development of collateral circulation has been indirectly monitored by determining peripheral coronary pressure and $^{133}$Xe clearance. In the present experiments, the development of collateral blood flow following gradual (three dogs) and rapid (three dogs) complete occlusion of the central circumflex branch, during partial central occlusion, and in parallel with a rise in peripheral coronary pressure was directly demonstrated by finding phasic coronary flow patterns in distal branches of the circumflex coronary artery. Collateral blood flow patterns were not consistently observed until a flow of 4–6 ml/min developed. In five dogs branch flow before central occlusion averaged 20 ml/min, but peak collateral blood flow after occlusion averaged 11.6 ml/min (range 7 to 17 ml/min). To circumvent central occlusion collateral blood flow must enter the circumflex bed retrograde; we proved this fact by finding collateral blood flow patterns that were mirror images of normal antegrade patterns and flow values that were negative with respect to mechanical reference zeros. Unexpectedly, collateral blood flow was antegrade in some branches indicating a redistribution of retrograde collateral blood flow within the circumflex bed. In a terminal open-chest experiment, there was indirect evidence of alternate collateral blood flow opposing measured collateral blood flow; this finding could explain some of the small reactive hyperemias observed after the release of a temporary occlusion of a distal branch. In summary, phasic collateral blood flow patterns in distal epicardial branches that developed rapidly in response to occlusion of the central circumflex branch and in conjunction with a rise in peripheral coronary pressure were demonstrated, and reversal from antegrade to retrograde collateral blood flow was documented.

KEY WORDS central circumflex branch peripheral coronary pressure distal or peripheral circumflex branch reactive hyperemia

Serial measurements of peripheral coronary pressure and $^{133}$Xe clearance have provided indirect evidence of the development of a collateral circulation to ischemic myocardium following coronary artery occlusion in conscious dogs. These indexes of collateral flow are very low during a brief temporary coronary occlusion, but they rise progressively following sustained partial or total coronary artery occlusion (1, 2).

In the present experiments, an attempt was made to measure directly the development of collateral blood flow in a peripheral branch of the circumflex coronary artery with an electromagnetic flowmeter. The experiments were performed on unanesthetized dogs.1

Methods

Experiments were conducted on six trained mongrel dogs (24.5–35.4 kg). An initial operation was performed through a left thoracotomy under sodium pentobarbital anesthesia (30 mg/kg, iv) for implantation of devices and intra-arterial tubes. Surgical procedures were carried out under sterile conditions. The lungs were ventilated with a Harvard respiratory pump connected to auffed endotracheal tube. In each dog, an electromagnetic flowmeter and a pneumatic cuff were implanted on a distal circumflex branch (Table 1). On the central circumflex branch, a mercury-filled cuff was implanted to produce gradual or rapid permanent occlusion, and a pneumatic cuff was implanted to produce temporary central circumflex occlusion (2). A Silastic tube was implanted in the first portion of the descending aorta, and...
**FLOWMETER MEASUREMENT OF COLLATERAL FLOW**

*Phasic records of the development of retrograde collateral blood flow at representative times during and after the period of gradual central circumflex occlusion (dog 2). The heavy horizontal bar above the day scale at the top of the figure represents the period of occlusion and vertical arrows denote times of increments of gradual occlusion. The top trace in each record is aortic pressure; the bottom trace is blood flow in the first marginal branch. Numerical values on each record were determined on 2-3 beats and are, from top to bottom, heart rate (beats/min), mean aortic pressure (mm Hg), and mean blood flow (ml/min) in the first marginal branch. The first record for each dog was taken before temporary central cuff occlusion (CCO); the center record was taken 15-20 seconds after central cuff occlusion and just before distal cuff occlusion (DCO); the third record is peak reactive hyperemia following distal cuff release (DCR). Horizontal lines represent mechanical zeros derived from distal cuff occlusions. Aortic pressure curves in each record were adjusted to atmospheric zero on scale at either side.*

**FIGURE 1**

**Graph of day-to-day changes for dog 2, before, during, and after central circumflex occlusion. Insert shows the placement of devices: mercury-filled cuff and pneumatic cuff on the central circumflex branch, and flowmeter and pneumatic cuff on the first marginal branch (circled area). HR = heart rate (beats/min) before central cuff occlusion, MAP = mean arterial pressure (mm Hg) before central cuff occlusion, PRH = peak reactive hyperemia (ml/min) (open circles joined by solid lines denote peak reactive hyperemia following release of central cuff occlusion, and open circles joined by broken lines indicate peak reactive hyperemia following release of distal cuff occlusion in the presence of complete central occlusion either from a temporary central cuff occlusion or from permanent occlusion with the mercury-filled cuff). CF = blood flow (ml/min) in the first marginal branch in the absence of a temporary central cuff occlusion, CoF = retrograde collateral blood flow in first marginal branch (ml/min) graphed below the zero line and denoted by crosses joined by solid lines. Reprinted with permission from Plenum Publishing Corp., New York, New York. The above figure appeared as Figure 5 in an article by Elliot, E.C.: Hemodynamic evidence of the development of coronary collateral circulation in conscious dogs. In Current Topics in Coronary Research, vol. 39, edited by C. M. Bloor and R. A. Olsson. New York, Plenum Press, 1973, pp 173-190.*
in dogs 4, 5, and 6 a Silastic tube was also implanted in the circumflex branch (2, 3); these tubes were used to measure aortic and coronary pressures. The dogs were given procaine penicillin (3.0 × 10^6 units, im) and streptomycin (1 g, im) before and after surgery; penicillin (1.5 × 10^6 units) and streptomycin (0.5 g) were given each day for 7-10 days after surgery.

Records were taken on an Electronics for Medicine DR8 recorder. Arterial blood pressures were measured with Statham transducers. In dogs 4, 5, and 6 the sensitivities of the two transducers were set at the same level and zero pressure references were superimposed to facilitate the comparison of aortic and coronary pressure traces. Electrocardiograms were made on a Sanborn recorder or on the Electronics for Medicine recorder. Before surgery the flowmeters were calibrated on arteries of appropriate size with normal saline and blood (hematocrit 40-45%) with a gravity-fed system. For the experiments in this study, flowmeters with known low imbalance and small phase shift were selected.

Experimental observations were commenced 5-10 days after surgery. In dogs 1, 2, and 3 central circumflex blood flow was gradually reduced to zero over 4-6 days, but in dogs 4, 5, and 6 central inflow was rapidly reduced to zero in 6-12 hours (Table 1). Intervals before, during, and after central occlusion are referred to as the preoclusional period, the occlusion period or the postocclusion period, respectively.

Collateral blood flow in these experiments is defined as the residual blood flow measured in a distal circumflex branch during complete central circumflex occlusion. To determine collateral blood flow in the postocclusion period, the distal branch beyond the flowmeter was occluded with the pneumatic cuff for 4-8 seconds; this procedure is hereafter called a distal cuff occlusion. With reference to the mechanical flow zero, the mean collateral blood flow was calculated using standard planimetric procedures and the calibration factor for that particular flowmeter. It was of particular interest, however, to establish whether collateral blood flow developed during partial occlusion. To make this measurement, the central circumflex branch was first temporarily occluded by inflating the central pneumatic cuff; then in 15-20 seconds a distal cuff occlusion of this branch was carried out. Collateral blood flow was then calculated as described. When the value of the planimetrically calculated mean collateral blood flow was positive in relation to the mechanical zero, the flow was antegrade (antegrade collateral flow), but a negative value indicated that the flow was in the opposite direction to normal antegrade flow (retrograde collateral flow). Complete permanent central occlusion by the mercury-filled cuff was assumed to have occurred, when, following a temporary central cuff occlusion, there was no change in branch flow, no change in the peripheral coronary pressure as in the case of dogs 4, 5, and 6, or no change in either flow or pressure.

In the presence of complete central occlusion, the evidence for collateral blood flow in a distal circumflex branch was a phasic coronary flow pattern that was clearly distinguishable from a flat mechanical reference zero of the distal cuff occlusion. These criteria were not consistently found until a collateral blood flow of 4-6 ml/min was observed. The most extreme situation prevailed.

### Summary of Salient Data for the Six Dogs

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>Dog wt (kg)</th>
<th>Branch with flowmeter</th>
<th>Time to complete central circumflex branch occlusion</th>
<th>Hematocrit (%)</th>
<th>Heart rate (beats/min)</th>
<th>Mean arterial pressure (mm Hg)</th>
<th>Branch flow (ml/min)</th>
<th>Mean peripheral coronary pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>35.4</td>
<td>Dorsal circumflex (2.0 mm)</td>
<td>4 days</td>
<td>40</td>
<td>82-142</td>
<td>20-25</td>
<td>N = 2</td>
<td>6-12 hours</td>
</tr>
<tr>
<td>2</td>
<td>34.0</td>
<td>First marginal (2.5 mm)*</td>
<td>6 days</td>
<td>45</td>
<td>118</td>
<td>82</td>
<td>N = 1</td>
<td>6-12 hours</td>
</tr>
<tr>
<td>3</td>
<td>24.5</td>
<td>Dorsal circumflex (2.5 mm)†</td>
<td>6 days</td>
<td>38</td>
<td>76-82</td>
<td>87-106</td>
<td>N = 2</td>
<td>6-12 hours</td>
</tr>
<tr>
<td>4</td>
<td>27.3</td>
<td>First marginal (2.0 mm)</td>
<td>6 hours</td>
<td>48</td>
<td>90</td>
<td>81-85</td>
<td>N = 2</td>
<td>6-12 hours</td>
</tr>
<tr>
<td>5</td>
<td>25.5</td>
<td>Second marginal (2.0 mm)</td>
<td>12 hours</td>
<td>44</td>
<td>60-70</td>
<td>86</td>
<td>N = 2</td>
<td>11-15 hours</td>
</tr>
<tr>
<td>6</td>
<td>29.0</td>
<td>Second marginal (2.5 mm)†</td>
<td>9 hours</td>
<td>59-64</td>
<td>96</td>
<td>9-14</td>
<td>N = 2</td>
<td>21 hours</td>
</tr>
</tbody>
</table>

*Flowmeter failed; a new one was applied 8 days after surgery.
†Dog damaged skin connector; a new flowmeter was applied 11 days after surgery.
‡Flowmeter failed; a new one was applied 6 days after surgery.
in the preocclusion period: collateral flow at this time was low, and poor electrode contact resulting from a sudden drop in coronary pressure was possible although encapsulation of the flowmeter was expected to be well advanced 6-10 days after surgery. Thus, in some dogs, although a small collateral flow appeared in the control period, values were considered to be unreliable and were disregarded.

In dogs 1, 3, 5, and 6 the location of the flowmeter and its relationship to other peripheral vessels was confirmed by gross observation following postmortem removal of the hearts and by injection of neoprene latex (Dupont No. 842A) into the hearts through the left anterior descending branch and subsequent digestion of the myocardial tissue with concentrated HCL (4). In dogs 2 and 4, the position of the flowmeter was only checked by gross observation at autopsy.

**Results**

**EFFECTS OF GRADUAL OCCLUSION OF THE CIRCUMFLEX BRANCH**

In dog 2 the flowmeter was placed on the first marginal branch. Phasic records at representative intervals are presented in Figure 1, overall changes are presented in Figure 2, and summary data are presented in Table 1. There was complete occlusion of the circumflex branch 17 days after surgery (Fig. 1, bottom row). The first two records in Figure 1 (bottom row) show that 14-17 ml/min of retrograde collateral flow had developed; the third record indicates that after release of the distal cuff occlusion, a peak reactive hyperemia of 31 ml/min occurred. Collateral flow was retrograde because the curves were below the mechanical zero of the distal cuff occlusion and were the mirror images of normal coronary flow patterns.

In dog 2, the transition of blood flow in the first marginal branch from normal antegrade flow to fully developed retrograde collateral flow occurred during partial occlusion (Fig. 1). Preocclusion branch flow was 31 ml/min (tenth day, first record); no collateral flow was detectable at this time (second record) or after two days of constriction (twelfth day, second record). But 14 days after surgery when antegrade branch flow had been reduced to 11 ml/min (first record), 5 ml/min of retrograde collateral flow was observed following central cuff occlusion (second record). The initial record taken before central cuff occlusion 15 days after surgery showed essentially no flow through the marginal branch. The central circumflex antegrade flow was apparently just balanced by distal collateral flow, because, when the former flow was removed by a central cuff occlusion, a retrograde collateral flow of 13 ml/min became evident (second record). This value of retrograde collateral flow represents the potential available crossover during complete central circumflex occlusion. The first record taken 16 days after surgery, however,

<table>
<thead>
<tr>
<th>Maximum heart rate during period of occlusion (beats/min)</th>
<th>Peak collateral blood flow in postocclusion period (ml/min)</th>
<th>Ratio of peak collateral flow to preocclusion control branch flow</th>
<th>Collateral blood flow at end of experimental period (ml/min)</th>
<th>Mean peripheral coronary pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>139</td>
<td>14</td>
<td>0.60</td>
<td>7</td>
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<tr>
<td>88</td>
<td>17</td>
<td>0.55</td>
<td>5</td>
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<td>97</td>
<td>11</td>
<td>0.50</td>
<td>9</td>
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<tr>
<td>150</td>
<td>3</td>
<td>0.17</td>
<td>3</td>
<td>19</td>
</tr>
<tr>
<td>127</td>
<td>9</td>
<td>0.70</td>
<td>6</td>
<td>35</td>
</tr>
<tr>
<td>72</td>
<td>7</td>
<td>0.58</td>
<td>4</td>
<td>52</td>
</tr>
</tbody>
</table>

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illustrated a retrograde collateral flow of 11 ml/min before central cuff occlusion. Because the retrograde flow increased to 17 ml/min (second record) after central cuff occlusion, the flow of 11 ml/min before central cuff occlusion represents crossover of retrograde collateral flow that was occurring during partial central circumflex occlusion.

The overall data for dog 2 (Fig 2) showed that the appearance of collateral flow during temporary central cuff occlusion coincided with the severe reduction in antegrade branch flow and the disappearance of reactive hyperemia following release of the temporary central cuff occlusion. The data also showed that, in the postocclusion period, the values of both collateral flow and reactive hyperemia following the release of the distal cuff occlusion progressively decreased during the balance of the experimental period. No significant alterations in heart rate or systemic arterial blood pressure occurred secondary to gradual occlusion of the central circumflex branch.

The phasic records for dog 3 are presented in Figure 3, and the summary data are presented in Table 1. The flowmeter was placed on the dorsal circumflex branch (Fig. 3). Following complete central circumflex occlusion, antegrade collateral flow was present 10 and 13 days after surgery; this flow contrasts with the retrograde collateral flow demonstrated in dog 2. At the third day of partial occlusion (9 days after surgery) the branch flow was reduced from 24 ml/min to 16 ml/min (first record); following central cuff occlusion 8 ml/min of antegrade collateral flow was measured (second record), but no reactive hyperemia was present at this time (third record). Collateral flow appeared in dog 3 when branch flow was severely reduced below that observed in the control period and reactive hyperemia following the release of temporary central cuff occlusion was abolished. In the postocclusion period, collateral flow stabilized at approximately 8-10 ml/min of antegrade flow; small reactive hyperemias were observed following the release of the distal cuff occlusions.

In dog 3, an open-chest experiment was performed under sodium pentobarbital anesthesia 20 days after surgery. The schematic insert in Figure 3 depicts four circumferential sutures on the epicardial vessels. When suture no. 1 was tightened, antegrade collateral flow increased from 10 ml/min to 14 ml/min; when suture no. 3 was tightened antegrade flow increased from 8 ml/min to 18 ml/min. Closure of either suture no. 2 or suture no. 4 caused the collateral flow pattern to disappear, confirming that the source of collateral blood flow in the first marginal branch was the left anterior descending branch.

In dog 1, the flowmeter was located on the dorsal circumflex branch, as it was in dog 3. A peak antegrade collateral flow of 14 ml/min was observed in the postocclusion period (Table 1). The double cuff occlusion technique (central cuff occlusion and distal cuff occlusion) was not performed during partial occlusion; therefore, no information was obtained from dog 1 about the development of collateral flow during partial occlusion.

**EFFECTS OF RAPID OCCLUSION OF THE CENTRAL CIRCUMFLEX BRANCH**

In dog 5, the flowmeter was placed on the second marginal branch. Representative phasic records are presented in Figure 4, overall changes are shown in Figure 5, and summary data are presented in Table 1. Eight days after surgery the central circumflex branch was rapidly constricted to cause a reduction in branch flow of approximately 50%. This reduction resulted in the elimination of the reactive hyperemia following temporary central cuff occlusion and an aortic-coronary pressure gradient of approximately 40 mm Hg (Fig. 5). Ventricular tachycardia predominated 5-30 hours after the commencement of central occlusion or up until 9 days 15 hours (Fig. 5). During this interval, however, there were sufficient sinus beats to permit measurements of collateral flow during sinus rhythm (Fig. 4). Antegrade collateral flow was observed 7 hours after constriction began (Fig. 5).

Figure 4 (top row) shows the phasic preocclusion records taken 8 days after surgery. The first record shows a branch flow of 14 ml/min but no aortic-coronary pressure gradient (curves superimposed); the second record taken 15-20 seconds after central cuff occlusion shows a peripheral coronary pressure of 19 mm Hg but no collateral flow. No significant changes were noted in the third and fourth records Figure 4 (middle row) shows records taken at 14 hours; by this time central occlusion was complete. The collateral flow was 4-5 ml/min (first and second records), and peripheral coronary pressure had risen from the control level of 19 mm Hg to 34-38 mm Hg (mean). The third record illustrates that, during the distal cuff occlusion, there was a further rise in peripheral coronary pressure to 43 mm Hg; this pressure fell to 39 mm Hg after the release of the distal cuff occlusion in conjunction with reactive hyperemia of 8 ml/min (fourth record). Figure 4 (bottom row) shows...
Phasic records of antegrade collateral blood flow in the dorsal circumflex branch developing secondary to gradual occlusion of the central circumflex branch (dog 3). Schematic insert at top left shows the flowmeter on the dorsal circumflex branch; the four circumferential sutures shown in the insert relate to the open-chest experiment performed 20 days after surgery (see text for details). The procedure is the same as in Figure 1.

records taken in the postocclusion period 10 days after surgery or 48 hours after the onset of constriction. The collateral flow had risen to 7-9 ml/min, and the collateral flow pattern was not different from the control branch flow pattern (compare with Fig 4 top row, first record). The peripheral coronary pressure had increased to 46 mm Hg (first and second records); a further rise to 52 mm Hg was exhibited during distal cuff occlusion, but a fall to 40 mm Hg occurred after the release of the distal cuff occlusion coincident with a reactive hyperemia of 11 ml/min. The values of heart rate and aortic pressure in these four records were similar. Peripheral coronary pressure and collateral flow continued to rise in the postocclusion period; however, the latter appeared to stabilize about 10 days after surgery (Fig. 5).

In dog 6, the flowmeter was placed on the second marginal branch. Representative phasic records appear in Figure 6, overall changes are shown in Figure 7, and summary data are presented in Table 1. Figure 6 (top row) shows preocclusion records taken 12 days after surgery; after central cuff occlusion no collateral flow was detectable, and peripheral coronary pressure was 21 mm Hg (second record). Following 6 hours of constriction, retrograde collateral flow was measured (Fig. 7). The phasic records in Figure 6 (middle row) were recorded at 11 hours when central occlusion was complete: collateral flow was 4-5 ml/min, and peripheral coronary pressure had increased to 33-43 mm Hg (first and second records). During distal cuff occlusion (third record) peripheral coronary pressure fell to 30 mm Hg and then rose slightly during the reactive hyperemia of 8 ml/min (fourth record). Figure 6 (bottom row) illustrates records taken during postocclusion 14 days after surgery or 27 hours after the onset of constriction that show a small increase in collateral flow to 6 ml/min, an elevation in peripheral coronary pressure to 65-66 mm Hg, and a diastolic run-off in peripheral coronary pressure. During distal cuff occlusion, peripheral coronary pressure was reduced to 51 mm Hg; it rose to 57 mm Hg during reactive hyperemia (fourth record).

The overall changes in dog 6 are shown in Figure 7. We were not as successful in dog 6 as we were in dog 5 in maintaining a constant aortic-coronary pressure gradient during the period of rapid occlusion. No changes in heart rate from control occurred, except for the elevated rate that appeared 14 days after surgery due to ventricular tachycardia which persisted during the measurement of collateral flow. A parallel rise in peripheral coronary pressure and collateral flow occurred following constriction up until 15 days after surgery when the occlusion of the central circumflex branch was released. Following the release of this occlusion there was an immediate return of antegrade flow in the branch; also a gradual return of reactive hyperemia occurred following temporary central cuff occlusion. In conjunction with these changes, a rapid fall in peripheral coronary pressure occurred, which by 24 hours had reached preocclusion levels. The collateral flow remained unchanged during the first 4 hours of release, but by 24 hours it was essentially negligible. Following reocclusion of the central circumflex branch 17 days after surgery, there was a rise in collateral flow and peripheral coronary pressure within 30 minutes.

In dog 4, the flowmeter was placed on the first marginal branch. The central occlusion performed in this dog was too severe and the dog died 6 hours later, presumably from ventricular fibrillation; however, shortly before death a small retrograde collateral flow of 3 ml/min appeared to be present (Table 1).
Phasic records of antegrade collateral blood flow developing secondary to rapid central circumflex occlusion, which was commenced 8 days after surgery at time zero (heavy bar on day scale) (dog 5). Flowmeter was placed on the second marginal branch (see schematic insert in Fig. 5). The phasic records during distal cuff occlusion are included (third record in each section). An intracoronary tube was implanted in the circumflex branch, and the coronary pressure curve is third from the top in each record, except in the first record of the top section in which aortic and coronary pressure curves are superimposed. The top trace is the lead II electrocardiogram recording. See text for the description of peripheral coronary pressure changes. The procedure is the same as in Figure 1.

**ELECTROCARDIOGRAPHIC FINDINGS**

In dog 1 progressive ischemic changes were observed in leads II, III, and aVF 9–12 days after surgery, which reverted to normal by 23 days after surgery. In dog 2 ischemic changes were evident in leads II, III, and aVF beginning 13 days after surgery; these changes were most marked 16 days after surgery. Persistent changes probably reflected subendocardial infarction. In dog 3 progressive ischemia was observed in leads II, III, and aVF 7–12 days after surgery. Improvement occurred by 13 days after surgery, and no evidence of infarction was observed. In dog 4 there was evidence of subendocardial infarction with probable transmural infarction. In dogs 5 and 6, there was evidence of inferior subendocardial infarction. These results were not histologically confirmed.

**Discussion**

By demonstrating phasic coronary flow patterns in distal branches of the central circumflex coronary artery, we have provided direct evidence of the development of collateral blood flow in conscious dogs following either rapid or gradual occlusion of the central circumflex branch. We also concluded that phasic coronary flow patterns in epicardial branches during the interval before the central circumflex branch was completely obstructed, indicating that collateral flow developed during partial occlusion. In the latter period, however, collateral blood flow only became ap-
Flowmeter measurement of collateral flow

Overall changes for dog 5. Abbreviations are the same as in Figure 2. Additional variables are MCP = mean coronary pressure (mm Hg) before central cuff occlusion, MPCP = mean peripheral coronary pressure (mm Hg) after central cuff occlusion and before distal cuff occlusion, and 2h, 4h, etc. = 2 hours, 4 hours, etc. after the start of rapid occlusion at time zero.

Parent when central constriction was performed to the point where reactive hyperemia to central cuff occlusion was eliminated, values of distal branch flow were reduced below control values, or both. This finding agrees with earlier reports (1, 2, 5) that the greatest increments in the collateral indexes of retrograde flow, peripheral coronary pressure, and $^{133}$Xe clearance occur after central inflow is severely compromised. The observation that collateral flow rose essentially in parallel with peripheral coronary pressure was particularly interesting because it supports our previous contention that peripheral coronary pressure, in spite of being an indirect measurement, can be used as an index of collateral circulatory development.

Since collateral flow was only monitored in one distal branch in each dog, no value of total collateral flow was attainable. Thus, the only value that could be compared with peak collateral flow was the value of branch flow in the control period. The ratio for five dogs (excluding dog 4) was 0.58 (range 0.50 to 0.70, Table 1).

During central circumflex occlusion, collateral flow must crossover retrograde via intercoronary connections into the circumflex bed. Irrefutable proof that antegrade flow in a marginal branch reverses to retrograde collateral flow during the postocclusion period and during partial occlusion was the altered flow pattern, namely, a pattern that was a mirror image of normal antegrade flow with a negative mean value with respect to the mechanical zero of the distal cuff occlusion. The finding that collateral flow could be antegrade was unexpected, but explainable. Epicardial collateral flow that reached the dorsal circumflex branch retrograde from the first marginal branch could only flow antegrade in the presence of central circumflex occlusion, and, if this flow then entered the second marginal branch, the collateral flow in that branch would also be antegrade, e.g., dog 2.

In these experiments, collateral flow could only be measured when the central circumflex branch was completely occluded. However, an exception to this circumstance was noted in the phasic records of dog 2 late during partial central occlusion: crossover of retrograde collateral flow was concurrently observed while a residual antegrade flow remained in the central circumflex branch, i.e., in the absence of a temporary central cuff occlusion.

The existence of antegrade and retrograde collateral flows in dogs 5 and 6, respectively, explains
the opposite changes observed in peripheral coronary pressure during the short distal cuff occlusion interval. In dog 5, peripheral coronary pressure rose during distal cuff occlusion (Fig. 4), because this occlusion raised the resistance of the portion of the circumflex bed in which peripheral coronary pressure was being measured. Conversely, in dog 6, since collateral flow in the second marginal branch was retrograde, blocking this flow during distal cuff occlusion lowered resistance in the circumflex bed, and peripheral coronary pressure fell (Fig. 6). In dog 6, the partial fall in peripheral coronary pressure during distal cuff occlusion indicates that there were other sources of collateral circulation. The hyperemic responses following the release of the distal cuff occlusion (fourth records, Figs. 4 and 6) explain the delayed return of peripheral coronary pressure to levels observed before distal cuff occlusion.

Undoubtedly, other collateral pathways functioned in these hearts, as suggested above and by the results of the open-chest experiment in dog 2. When suture no. 1 was tightened (Fig. 3), antegrade collateral flow increased by 4 ml/min, which presumably compensated for retrograde flow that would have otherwise entered at site no. 1. An even larger increase in antegrade flow occurred when suture no. 3 was tightened (10 ml/min). This observation is understandable, because retrograde flow entering at site no. 3 supplied a large part of the circumflex bed. Thus, it is conjectured that the collateral flows that we measured might, in some instances, have represented net values resulting from opposing flows. Alternate pathways developing at different rates could also explain, in dog 2, the progressive decrements in collateral flow and reactive hyperemia during the 17–30-day interval after surgery. A misalignment of the flowmeter in dog 2 was suspected at autopsy, but, because of encapsulation, a progressive misalignment would be unlikely after 17 days.

In dog 6, following the release of permanent
central occlusion, the return of antegrade flow and reactive hyperemia caused by temporary central cuff occlusion substantiated patency of the second marginal branch; however, concomitant decrements in collateral flow and peripheral coronary pressure indicated regression of the collateral circulation (Fig. 7). The rapid rise in the latter two variables following permanent reocclusion contrasted with the slower response after the first occlusion. Changes in peripheral coronary pressure essentially agreed with earlier findings of Khouri et al. (3).

Acknowledgment

We are grateful to Mr. Thomas M. Mouer for his technical assistance throughout these experiments and to Mr. Robert E. Jones for the fabrication of the electromagnetic flowmeters and pneumatic cuffs.

References

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