Coronary Steal Mechanisms in Dogs with One-vessel Occlusion and Other Arteries Normal

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SUMMARY The objectives of the present study were to determine whether coronary steal can occur in one-vessel coronary occlusion when other arteries are normally patent, and to determine the quantitative effect on the magnitude of coronary steal of a graded partial stenosis in the coronary circulation proximal to the origin of the collaterals. In 10 dogs, occlusion of the left anterior descending coronary artery (LAD) was induced and perfusion of the left main coronary artery was maintained with a special servo pump at constant pressure (measured distal to the cannula tip) during intracoronary adenosine infusion. Adenosine increased left main coronary blood flow from 85 ± 15 to 229 ± 42 ml/min, while mean pressure in the left main coronary artery was held constant at 83 ± 3 mm Hg (mean ± SD). Systemic hemodynamics did not change, but three indexes of collateral function decreased: peripheral LAD coronary pressure, from 14.0 ± 2.7 to 12.5 ± 2.9 mm Hg (p < 0.005); retrograde LAD flow, from 3.2 ± 2.5 to 2.4 ± 2.6 ml/min (p < 0.001); and microsphere blood flow to ischemic myocardium, from 0.055 ± 0.029 to 0.040 ± 0.031 ml/min/g (p < 0.03). Next, we added the resistance of the Gregg cannula to the perfusion system, i.e., in the same dogs we regulated constant pressure in the tubing proximal to the cannula tip. This added resistance created a 4.5-mm Hg pressure gradient (multivessel occlusion) and a larger drop in retrograde LAD flow (23% vs 36%, p < 0.05). Because a screwclamp caused graded increments in resistance of the left main coronary perfusion system and the resting pressure gradient, there was a linear increase in the magnitude of coronary steal (r = 0.71). Thus, arteriolar vasodilators increased flow velocity, which led to decreased pressure at the origin of the collaterals because there was some resistance in the native coronary circulation between the left main coronary artery and the origin of the collaterals (12% of total resting resistance). Further increments in vascular resistance proximal to the origin of the collaterals caused a linear increase in the magnitude of coronary steal.

VASODILATOR-INDUCED increases in blood flow through a partially occluded coronary vessel, which decrease pressure distal to the partial occlusion, have been suggested as the cause of decreased collateral flow to a totally occluded vessel in dogs with coronary occlusions during dipyridamole administration.1, 2 Fam and McGregor1, 2 did not use the term "coronary steal;" others have used it to describe the effect of vasodilator drugs to decrease flow to ischemic myocardium by this specific mechanism.3-6

Although coronary steal has been demonstrated clearly where one artery is totally occluded and another artery is partially occluded (multivessel occlusion),1, 2, 5, 6 there is conflicting evidence concerning whether coronary steal can occur when only one artery is totally occluded and other arteries are normally patent. This question was raised, but not answered, in a recent symposium.7 In several studies, decreases in blood flow to ischemic myocardium have been reported and were suggested to result from coronary steal. These studies, however, did not demonstrate coronary steal, because they involved only changes in transmural distribution of blood flow to myocardium8, 9 supplied by a single partially occluded coronary artery.8-10 These studies and another study11 showed associated decreases in aortic pressures, however, so they could not demonstrate coronary steal in the absence of partial occlusions on other coronary arteries. If the coronary vasodilator agent decreases aortic pressure, this fall in coronary perfusion pressure, per se, could explain the decreased collateral blood flow without invoking the specific mechanism described by Fam and McGregor.1, 2 A previous study from this laboratory found that i.v. adenosine decreased collateral myocardial blood flow more than aortic pressure, yielding a decreased calculated value of conductance. The decreased value of conductance in that study did not prove the occurrence of coronary steal because the relationship between aortic pressure and collateral blood flow may not always be linear over the entire range of aortic pressures.14

Cohen et al.15 reported coronary steal after occlusion of the left anterior descending coronary artery (LAD) and infusion of either isoproterenol or adenosine. The conclusion that "coronary steal can be induced when the large coronary vessels are normal" except for the occluded LAD was incorrect because the other vessels were not, in fact, normal. That study failed to account for a potential pressure gradient across the tip of the Gregg cannula in the left main coronary artery, which could create a partial stenosis proximal to the collaterals.

In a recent review, Schaper and Wusten16 expressed skepticism about the occurrence of coronary steal in one-vessel occlusion: "Our own measurements of collateral blood flow in acute coronary occlusion during autoregulation and during maximal (adenosine-induced) vasodilation (unpublished) did not show sys-
tematic differences. We interpret these findings as indicative that a pure flow-induced steal does not play an important role in acute coronary occlusion. 13

In contrast to the demonstration that arteriolar vasodilators might cause coronary steal when given to dogs with two-vessel occlusion, 17 Becker 18 reported recently that dipyridamole actually increased collateral myocardial blood flow when methoxamine was used to maintain constant aortic pressure in dogs with LAD occlusion alone. These studies led Becker to conclude that coronary steal cannot occur unless there is a partial occlusion of one artery in addition to the total occlusion. 17

The objectives of the present study were to determine whether coronary steal can occur in the presence of a one-vessel occlusion with other arteries normally patent and with constant left main coronary perfusion pressure, and to determine the quantitative effects on coronary steal of graded stenoses of the coronary circulation proximal to the origin of the collaterals.

Methods

Experimental Preparation (fig. 1)

Ten mongrel dogs that weighed 19–26 kg were anesthetized with sodium pentobarbital, 30 mg/kg i.v., and received positive pressure respiration with 100% oxygen. A thoracotomy was performed to instrument the dogs (fig. 1).

After administration of heparin, 5000 U i.v., a large-bore (¾ inch i.d.) plastic T-connector cannula was inserted into the aorta and tied securely without occluding the aorta. Heparin doses of 3000 U i.v., were repeated every 30 minutes. The cannula was connected by polyvinyl tubing (½ inch i.d.) to a modified Gregg cannula (¼ inch i.d.) in the left main coronary artery (fig. 1). The perfusion system included a servo pump (Harvard Apparatus, Inc.), an extracorporeal flow probe (Biotronix), and side arms for infusion of drugs or measurement of perfusion pressure just proximal to the left coronary cannula. Another side arm was connected to perfuse the LAD independently with blood from a side arm of the left main coronary cannula. Additional side arms allowed measurement of LAD peripheral coronary pressure (PCP) and timed collections of retrograde flow (RF) after occlusion of the perfusion line that supplied the LAD.

Thin-walled polyvinyl tubing was inserted into the proximal end of the LAD incision and advanced retrogradely into the left main coronary artery, just distal to the tip of the cannula as determined by direct palpation. This catheter was connected to a P23Db pressure transducer to measure pressure in the left main coronary artery itself, distal to the tip of the Gregg cannula but proximal to the origin of collaterals or other coronary branches. This left main coronary pressure served as the input pressure, or set point, which was regulated by the servo pump to allow this preparation to serve as a model of one-vessel (LAD) occlusion with other coronary branches normally patent.

Microsphere Estimates of Myocardial Blood Flow

Standard carbonized microspheres, 9 ± 1 µm in diameter, labeled with the nuclides 125I, 141Ce, 51Cr, 85Nb and 46Sc (3M Company), were used to measure regional myocardial blood flow and cardiac output as described previously. 19, 21 After the dogs were killed, Evans Blue dye was injected into the LAD cannula to stain the region of myocardium supplied by the LAD. Biopsy samples (0.80–1.20 g) were taken 5–10 mm inside the blue-stained ischemic region supplied by the LAD to minimize contamination by normal tissue. 13, 18, 21

The potentially ischemic region supplied by the LAD was identified more precisely by a modification of a microsphere labeling method previously developed 19 and used 13, 19–22 in this laboratory. Briefly, the first set of microspheres was injected into a mixing chamber in the left main coronary perfusion tubing distal to the side arm that perfused the LAD so that the LAD branch did not receive these microspheres (fig. 1). Five minutes were allowed for complete washout of these microspheres into the myocardium supplied by
the left main coronary artery. Microspheres were excluded from the LAD because it was perfused separately with blood containing no microspheres, at the same pressure as the left main coronary to minimize flow through interarterial anastomoses. Counting myocardial radioactivity due to this first set of microspheres revealed that this label was present in the normal region of the left ventricle, while the potentially ischemic region was identified by its containing essentially no microspheres with this label.13 Our ischemic samples from the center of the blue-stained region contained little or no overlapping normal zone tissue; thus, we did not calculate a corrected blood flow to ischemic components of mixed samples.21, 22

**Experimental Protocols**

After injecting the first set of microspheres into the left main coronary perfusion line to identify the potentially ischemic myocardium, the LAD was occluded permanently and the LAD tubing disconnected from the main perfusion line (fig. 1). Eight to 9 minutes after LAD occlusion, control values of peripheral LAD pressure and retrograde LAD flow were measured, and microspheres were injected into the left atrium to estimate myocardial blood flow. Adenosine infusion (0.1–0.3 μmol/min) into the left main coronary perfusion line produced maximal measured flow rates by 18–20 minutes after LAD occlusion. The servo pump regulated the mean pressure at a constant value in the left main coronary artery distal to the cannula tip, to model the situation of a single coronary occlusion and a constant coronary perfusion pressure. When pressure and coronary flow had stabilized during adenosine infusion, all measurements were repeated.

Repeat measurements of pressures, coronary flow, peripheral LAD pressure, retrograde LAD flow and microsphere blood flow were made during a second control period, after allowing pressures and flows to return to normal after discontinuing adenosine.

Although left main coronary artery pressure did not change from the control before adenosine, the servo pump was used to regulate constant pressure in the tubing, proximal to the left coronary cannula, to model the situation of multivessel coronary disease, i.e., total LAD occlusion plus a mild constriction of the left main coronary artery due to the Gregg cannula. The cannula tip introduced a mild partial stenosis proximal to the origin of the collaterals as shown by the average pressure gradient across the Gregg cannula of 4.5 ± 4.1 mm Hg (mean ± SD). Adenosine was infused into the perfusion system at the same rate as before, regulating constant pressure in the tubing proximal to the left main coronary cannula. Left main coronary pressure distal to the tip of the cannula decreased during adenosine infusion with the regulation of pressure constant proximal to the cannula tip. Left main coronary flow increased to a lower peak value than during regulation of left main coronary pressure distal to the tip of the cannula (229 ± 42 vs 178 ± 56 ml/min, p < 0.05). We randomized the sequence of regulating constant pressure in either the left main coronary artery itself or the pressure in the cannula.

After measuring PCP, RF and microsphere blood flows under these conditions, adenosine was discontinued. A screw clamp was placed on the left coronary perfusion line, between the side arm used to measure left coronary "cannula" pressure and the brass cannula in the left main coronary artery (fig. 1). The screw clamp was tightened to add more resistance to the partial constriction of the brass cannula and tubing. The servo pump was used to maintain constant pressure in the tubing proximal to the cannula. Control measurements were made of pressure, coronary flow, peripheral LAD pressure, retrograde LAD flow and microsphere blood flows. Then, adenosine was infused at the same rate into the left main coronary perfusion line. Left main coronary pressure distal to the brass cannula decreased; left coronary flow increased less than it had previously. Measurements were repeated.

**Analysis of Data**

A paired t test was used to compare values obtained in the same dog before and during adenosine. A paired t test was also used to compare the effects in the same dog of regulating left main coronary arterial pressure constant regulating cannula pressure constant (to add the resistance of the tubing and cannula). A one-way analysis of variance was used to test whether the slope of a regression line differed from zero.23 A p value less than 0.05 was considered statistically significant.

We calculated the resistance added to the left main coronary artery by the cannula or constriction under control conditions as

\[ R_{\text{added}} = \frac{P_{\text{can}} - P_{\text{LM}}}{\text{CBF}_{\text{LM}}}, \]

where \( P_{\text{can}} \) = cannula pressure proximal to the brass cannula tip, \( P_{\text{LM}} \) = left main coronary pressure distal to the cannula tip, and \( \text{CBF}_{\text{LM}} \) = left main coronary blood flow (fig. 2).

To compare an index of coronary steal in different dogs with varying resting coronary collateral resistance and varying increases in left main coronary blood flow during adenosine, we calculated an index as shown in the Appendix.

**Results**

**Controlled Left Main Coronary Pressure Preparation**

When mean left main coronary pressure was regulated constant by the servo pump at 83 ± 3 mm Hg in 10 dogs, intracoronary adenosine produced no significant change from control in heart rate (137 ± 24 vs 130 ± 24 beats/min, NS), left ventricular peak systolic pressure (100 ± 18 vs 93 ± 18 mm Hg, NS) or end-diastolic pressure (10.0 ± 5.1 to 9.9 ± 7.2, mm Hg, NS). Left main coronary flow increased from 85 ± 15 to 229 ± 42 ml/min (p < 0.005) (fig. 2). Diastolic PCP decreased 9% from 14.0 ± 2.7 mm Hg (p < 0.005) and RF decreased 23% from 3.2 ± 2.5 ml/min
Coronary effects of intracoronary adenosine (a) with controlled left main coronary pressure (CP). The height of the clear bar in each pair indicates the mean value during the control (c) period after left anterior descending coronary artery (LAD) occlusion. The brackets indicate the SEM. The height of the hatched bar indicates the mean value during intracoronary adenosine infusion. The code above each pair of bars indicates the result of a paired t test testing the significance of any change. The first set of bars confirms that mean CP did not change. Left main coronary blood flow (CBF) increased, diastolic peripheral LAD coronary pressure (PCP) decreased, and retrograde LAD flow (RF) decreased. Not shown are several hemodynamic variables that did not change: heart rate, left ventricular peak systolic and diastolic pressures and cardiac output. The reduction in PCP and RF during adenosine despite no change in CP indicates coronary steal during regulation of mean left main coronary pressure constant to model one-vessel occlusion with other coronary arteries normally patent.

\[ \text{PCP} \] (mm Hg)

\[ \text{CBF} \] (ml/min)

\[ \text{CP} \] (mm Hg)

\[ \text{RF} \] (ml/min)

\( p < 0.01 \). Intracoronary adenosine infusion with mean left main coronary pressure held constant required an increase in the pressure gradient across the left coronary cannula from 4.4 ± 1.1 to 8.3 ± 2.2 mm Hg \( (p < 0.001) \), which was associated with an increase from 1.11 ± 0.30 to 3.26 ± 0.98 ml·min\(^{-1}\)·g\(^{-1}\) in blood flow to normal myocardium \( (p < 0.001) \), but a 27% decrease, from 0.055 ± 0.029 to 0.040 ± 0.031 ml·min\(^{-1}\)·g\(^{-1}\), in blood flow to ischemic myocardium \( (p < 0.03) \) (fig. 3). Thus, three indexes of collateral coronary flow decreased to demonstrate coronary steal despite no change in left main coronary pressure or other hemodynamic variables.

Next, the servo pump regulated pressure at a constant value in the tubing proximal to the Gregg cannula (cannula pressure) to test the effect of added resistance proximal to the origin of the collaterals, i.e., a model of multivessel coronary disease. Adenosine caused a decrease in mean left main coronary pressure distal to the cannula, from 76 ± 6 to 70 ± 9 mm Hg \( (p < 0.025) \). Again, adenosine caused no change in heart rate or left ventricular pressures. Left main coronary flow increased from 72 ± 15 to 178 ± 57 ml/min \( (p < 0.01) \). RF decreased from 3.8 ± 4.2 to 2.4 ± 2.7 ml/min \( (p < 0.001) \). The changes produced by adenosine in each dog without the added left main coronary resistance (i.e., one-vessel disease model) were compared with the changes caused by the same intracoronary dose of adenosine in the same dog with left coronary resistance added by regulating constant pressure in the cannula (i.e., multivessel disease model). When the brass cannula and tubing resistance were added to the left main coronary artery, an average 4.5-mm Hg resting pressure gradient was noted across the cannula. Adenosine caused a slightly smaller increase in left coronary flow (247% vs 269%) and a larger reduction in retrograde LAD flow (36% vs 23%, \( p < 0.05 \)) when the resistance was added to the left main coronary artery proximal to the origin of the collateral vessels.

The effect of added left coronary resistance is to increase the magnitude of the decrease in retrograde LAD flow (normalized change in RF) (fig. 4). As shown in the Appendix, the magnitude of coronary steal is expressed as normalized change in RF \( (\text{the change in retrograde flow caused by adenosine/the change in left coronary flow caused by adenosine}) \times \) the collateral resistance before adenosine). A coronary steal \( (\text{defined as a change in RF}) \) is present even when no resistance is added \( (y \text{ intercept} = 0.21 \text{ ml/min}) \) (fig. 4). In fact, the steal could not be abolished theoretically unless 0.12 resistance unit was subtracted from the native left main coronary artery \( (x \text{ intercept} = -0.12 \text{ mm Hg·ml·min}^{-1}·\text{g}^{-1}) \). These data imply that the normal left main coronary artery and circumflex system proximal to the origin of the collaterals have a vascular resistance of 0.23 unit, or 12% of total vascular resistance.

**Discussion**

The objectives of this study were to determine whether coronary steal could occur during adenosine-induced vasodilation when one coronary artery was

**Figure 2.**

*Coronary effects of intracoronary adenosine (a) with controlled left main coronary pressure (CP). The height of the clear bar in each pair indicates the mean value during the control (c) period after left anterior descending coronary artery (LAD) occlusion. The brackets indicate the SEM. The height of the hatched bar indicates the mean value during intracoronary adenosine infusion. The code above each pair of bars indicates the result of a paired t test testing the significance of any change. The first set of bars confirms that mean CP did not change. Left main coronary blood flow (CBF) increased, diastolic peripheral LAD coronary pressure (PCP) decreased, and retrograde LAD flow (RF) decreased. Not shown are several hemodynamic variables that did not change: heart rate, left ventricular peak systolic and diastolic pressures and cardiac output. The reduction in PCP and RF during adenosine despite no change in CP indicates coronary steal during regulation of mean left main coronary pressure constant to model one-vessel occlusion with other coronary arteries normally patent.*

**Figure 3.**

*Effects of intracoronary adenosine on regional myocardial blood flow (MBF) while mean left main coronary pressure (CP) was kept constant by the servo pump. Designations on the pairs of bar graphs are as in figure 2. Adenosine produced no change in CP, a 194% increase in flow to the normal zone (MBF\(_{NZ}\)), and a 36% decrease in flow to the ischemic zone (MBF\(_{IZ}\)) \( (p < 0.03) \).*
occluded and the other coronary branches were normally patent and to determine the relationship between coronary steal and varied partial coronary stenosis proximal to the origin of the collateral vessels. Despite evidence that collateral coronary blood flow varies as a linear function of aortic pressure, others have reported that this relationship may not always be linear. Thus, one cannot conclude that an agent causes coronary steal if it also decreases aortic blood pressure.

Our experimental model controlled main left coronary perfusion pressure at a constant value during intracoronary adenosine vasodilation. Later, we deliberately added a partial stenosis to the cannula proximal to the left main coronary artery to test the effect of resistance added proximal to the origin of collateral vessels. During these periods, we regulated pressure to maintain a constant value in the cannula proximal to the site of the partial stenosis. This model allowed quantitation of the resistance of the partial stenosis and its effect on collateral blood flow during adenosine vasodilation.

The results of these studies demonstrate the occurrence of coronary steal — a reduction in three indexes of coronary collateral function — during adenosine vasodilation even when there was one-vessel (LAD) occlusion and the remainder of the coronary circulation was normally patent.

The magnitude of decreased collateral blood flow in this model of one-vessel occlusion with constant left main coronary perfusion pressure was 23–27%, measured by two methods. This degree of decreased collateral flow is quantitatively related to further decreases in regional left ventricular contraction and to further increases in myocardial infarction size.

The mechanism of coronary steal when there are no stenoses proximal to the origin of the collaterals can be inferred to be the presence of some resistance in the large coronary arteries between the left main and the point of origin of the collaterals. Figure 4 interprets the x-axis at −0.12 mm Hg·ml·min, indicating that 12.5% of the total resting coronary vascular resistance value would have to be subtracted from the native coronary circulation between the left main and the origin of the collaterals to abolish the coronary steal. Partial constriction of the left main coronary artery caused larger decreases in collateral flow during adenosine (fig. 4).

The demonstration of coronary steal during vasodilation after one-vessel occlusion with the remainder of the coronary circulation normal differs importantly from reports by Becker. In dogs with LAD occlusion receiving methoxamine to maintain constant aortic pressure, dipyridamole caused a 71% increase in collateral blood flow measured by radioactive microspheres. In contrast, in dogs with LAD occlusion plus variable partial stenosis of the circumflex, dipyridamole decreased collateral blood flow despite methoxamine infusion to maintain constant aortic pressure. These results were interpreted to show that coronary steal cannot occur unless there is a partial stenosis in the coronary circulation proximal to the origin of the collaterals.

The differences between the conclusions of the present study and those by Becker may be related to differences between adenosine and dipyridamole or to the use of methoxamine, which has been reported to increase coronary collateral function. Most important, the study by Becker differed from the present study in the criteria used to identify myocardial samples as ischemic in order to measure collateral blood flow by the microsphere method. Another study from this laboratory indicated that samples selected as ischemic by the criterion used by Becker—blood flow less than 50% of the normal zone flow — produced misleading results during vasodilation because these criteria fail to exclude normal zone tissue that overlaps with ischemic
tissue. The difference between the conclusions about coronary steal in the present study and the studies by Becker may be that myocardial samples in those studies contained a mixture of normal tissue in supposedly ischemic samples, and the large increase in normal zone flow during dipyridamole may have obscured an actual decrease in collateral blood flow in mixed samples. These considerations suggest that the present study is correct in demonstrating that coronary steal can occur with one-vessel occlusion and other arteries normally patent.

Further, the present study expands on the report of Becker that indicates coronary steal during vasodilation with a circumflex partial stenosis after LAD occlusion. We have defined quantitatively the magnitude of intrinsic vascular resistance present in the coronary circulation between the left main coronary artery and the origin of the collateral vessels (12%) and the effects of increases in resistance in the coronary circulation proximal to the origin of the collaterals.

Mechanics of Coronary Steal

Coronary steal can be defined mechanistically in terms of pressure changes in the coronary circulation at the origin of the collateral vessels. Vasodilation of distal arterioles causes a large volume of blood per unit time to flow by the points of origin of collateral vessels, resulting in a higher flow velocity. The increased flow velocity is accompanied by a reduction in pressure at the origins of the collaterals. The effect of decreased pressure in the nonoccluded arteries at the origins of collateral vessels is determined by the relative changes in pressures in the distal vascular beds of normal vs ischemic myocardium. Because vascular resistance of collaterals and distal ischemic zone vessels is probably minimal due to the process of ischemia per se, drugs that cause vasodilatation of small arteries will decrease vascular resistance and pressure in normal myocardium, while causing little or no change in the ischemic zone vessels. Thus, during arteriolar vasodilatation, the driving pressure gradient between the unoccluded vessel at the origin of the collaterals and the normal zone arterioles will increase due to an increase in blood flow. In contrast, since arteriolar vasodilatation cannot decrease the low pressures in the collaterals and the ischemic arterioles, the fall in pressure at the origin of the collaterals leads to a decreased pressure gradient and a decreased collateral flow — coronary steal.

When there was a constriction of the coronary artery proximal to the origin of the collaterals, the same volume of blood per unit time had to pass through the narrowed vessel at a higher flow velocity. The higher velocity of flow would be associated with a lower pressure in the artery. Thus, a stenosis proximal to the origin of the collaterals (multivessel disease) would be expected to aggravate the decrease in distal pressure at the origin of the collaterals and, thus, increase the magnitude of steal in proportion to the decrease in coronary pressure.

Clinical Implications

Extrapolation from canine studies to man must always be approached with caution, but the ability to make controlled measurements with the best techniques in dogs provides valuable insights into the disturbed physiologic mechanisms found in human coronary disease. First, the magnitude of coronary steal appears to be small relative to the potential for reduction of collateral flow by interventions that reduce aortic pressure, emphasizing the value of careful monitoring of arterial blood pressure to maintain patient safety during administration of vasodilators. Second, coronary steal may occur in any patient with coronary disease, including those with one-vessel disease. Third, the potential magnitude of coronary steal is increased by the presence of a coronary stenosis proximal to the origin of collateral vessels (i.e., multivessel disease), the more proximal the location of a given lesion before the origins of more collaterals, and the magnitude of increase in blood flow velocity through normal vessels from which collateral vessels originate.

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Appendix

An Index of Coronary Steal

To compare an index of coronary steal in different dogs with varying coronary collateral resistances and varying responses of left main coronary blood flow to adenosine, we calculated the index, normalized change in RF:

\[
\frac{\text{RF}_{\text{ado}} - \text{RF}_{\text{con}}}{\text{CBF}_{\text{ado}} - \text{CBF}_{\text{con}}} \cdot \frac{\text{RF}_{\text{ado}}}{\text{RF}_{\text{con}}}
\]

where \(\text{RF}_{\text{con}}\) = control RF, \(\text{RF}_{\text{ado}}\) = RF during adenosine, \(\text{CBF}_{\text{ado}}\) = left main coronary blood flow during adenosine infusion or control periods (CBF_{con}), \(\text{R}_{\text{cc}}\) = control collateral resistance estimated by left main coronary pressure (LMCP)/control RF (LMCP/RF_{con}). The magnitude of steal (normalized change in RF) was plotted as a function of added left main resistance. We calculated a correlation coefficient and a best-fit, least-squares linear regression and the 95% confidence limits for the line.23
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