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# Absolute Quantitation of Coronary Steal Induced by Intravenous Dipyridamole

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**OBJECTIVES** The study was done to determine whether coronary steal (defined as an absolute decrease in perfusion from resting blood flow) is induced by intravenous (IV) dipyridamole in patients with severe coronary artery disease (CAD).

**BACKGROUND** Myocardial ischemia during coronary vasodilation is usually attributed to coronary steal. However, there is limited data on the absolute magnitude of coronary steal in humans.

**METHODS** Eighteen patients with multivessel CAD underwent dynamic positron emission tomography (PET) imaging with $^{13}NH_3$ at rest and after infusion of IV dipyridamole. Eight myocardial sectors were analyzed per short axis slice and myocardial blood flow calculated with a two-compartment model in absolute terms.

**RESULTS** Coronary steal occurred in 8 of the 18 patients. In the 8 patients with coronary steal, myocardial blood flow decreased from 90 ± 18 ml/100 g/min at rest to 68 ± 27 ml/100 g/min following dipyridamole in the segments with steal, and increased from 87 ± 19 to 138 ± 16 ml/100 g/min following dipyridamole in the segments without steal. Significant clinical correlates of coronary steal were either ST elevation or the combination of ST depression and angina.

**CONCLUSIONS** Coronary vasodilation with IV dipyridamole is associated with significant reductions in blood flow to collateral-dependent myocardium consistent with coronary steal in about 45% of patients with severe CAD. (J Am Coll Cardiol 2001;37:109–16) © 2001 by the American College of Cardiology

Perfusion imaging during coronary vasodilation with either adenosine or dipyridamole is widely used for the diagnosis of coronary artery disease (CAD) (1–3). Dipyridamole, by increasing endogenous adenosine levels, induces coronary vasodilation (4,5). In experimental studies, absolute flow is increased, even in territories distal to stenoses, when collateral circulation is not present (6,7). Nonetheless, myocardial ischemia (defined as an imbalance between myocardial perfusion and demand) is believed to occur in some patients with CAD following dipyridamole. Because dipyridamole does not usually increase myocardial oxygen demand, it causes ischemia, mainly by decreasing flow to collateral-dependent vascular beds through "coronary steal" (8–12).

Coronary steal is conventionally defined as an absolute decrease in perfusion, compared to resting flow, to collateralized myocardium following coronary vasodilation (12). It is caused by redistribution of flow away from collateral-dependent myocardium following coronary vasodilation because of a fall in resistance in the supply artery diverting flow away from the collateral bed, which is already near maximal vasodilation (13). Thus, the driving pressure at the head of the collateralized bed decreases and reduces distal perfusion.

Myocardial ischemia due to coronary steal is generally believed to be manifested clinically by ST segment depression following coronary vasodilation (13,14). Although coronary steal has been well described in canine studies (15,16), there are more limited observations of the absolute magnitude of coronary steal in humans and its relationship to clinical signs of myocardial ischemia. In addition, there is controversy whether chest pain and electrocardiographic (ECG) changes with coronary vasodilation represent true myocardial ischemia because of coronary steal. Thus, the goal of this study was to evaluate whether dipyridamole induces absolute flow reductions in collateralized myocardium in patients with multivessel disease, and to determine whether any clinical signs or symptoms correlate with the occurrence of myocardial ischemia due to steal.

## METHODS

The study was approved by the Institutional Review Board of the Columbia-Presbyterian Medical Center, and written informed consent was obtained. Eighteen patients (15 men, 3 women) with severe multivessel coronary artery disease (double vessel disease in 4 and triple vessel disease in 14 patients) as assessed by coronary angiography performed within six months of enrollment underwent quantitative positron emission tomography (PET) imaging at rest and again following induction of hyperemia with intravenous (IV) dipyridamole. No cardiac events occurred between the angiogram and the PET study. Absolute blood flow levels (ml/100 g/min) at rest and after vasodilation were correlated with angiography.

**Positron emission tomography.** Subjects were studied after an overnight fast. All caffeinated beverages and

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**Abbreviations and Acronyms**

| | |
|---|---|
| ACE | = angiotensin-converting enzyme |
| BP | = blood pressure |
| CABG | = coronary artery bypass grafting |
| CAD | = coronary artery disease |
| LAD | = left anterior descending artery |
| LCx | = left circumflex artery |
| MBF | = myocardial blood flow |
| $^{13}N$ | = nitrogen-13 |
| $NH_3$ | = ammonia |
| PET | = positron emission tomography |
| RCA | = right coronary artery |

theophylline-containing medications were withheld for at least 24 h. The studies were performed on an ECAT EXACT-47 whole-body scanner, which provides 47 contiguous transaxial slices. The localization of the heart within the axial field of view of the scanner was confirmed by performing a 2-min transmission scan using a rotating germanium-68/gallium-68 ($^{68}Ge/^{68}Ga$) rod source. Patient positioning within the field of view of the camera was indexed using laser cross beams and indelible marks on the patient's torso. The patients were instructed not to move during data acquisition, and the location of the ink marks relative to the laser cross beams on their torso was checked frequently to ensure compliance. A 20-min transmission scan with a rotating $^{68}Ge/^{68}Ga$ rod source was performed to correct for attenuation.

Between 12 to 20 mCi of nitrogen-13 ammonia ($^{13}NH_3$) was infused with a Harvard constant infusion pump over 30 s. Sequential dynamic frames were acquired for a total of 19 min (twelve 10-s frames, two 30-s frames, one 60-s frame, and one 900-s frame).

After completion of the rest study, we allowed 30 to 60 min for the decay of $^{13}NH_3$. Patients then received IV dipyridamole (0.56 mg/kg) via a Harvard pump over 4 min. Blood pressure and 12-lead ECGs were recorded every minute during dipyridamole infusion and for an additional 4 min thereafter. Patients were repeatedly questioned about symptoms following the onset of infusion of dipyridamole. Episodes of chest pain that tended to mimic the patient's usual angina were noted. At the 8th min following the onset of dipyridamole infusion, between 12 and 20 mCi of $^{13}NH_3$ was infused over 30 s. Sequential dynamic imaging was commenced simultaneously for 19 min using the framing protocol outlined above. All patients with chest pain and/or ST segment depression received IV aminophyline after the first 2 min of data acquisition.

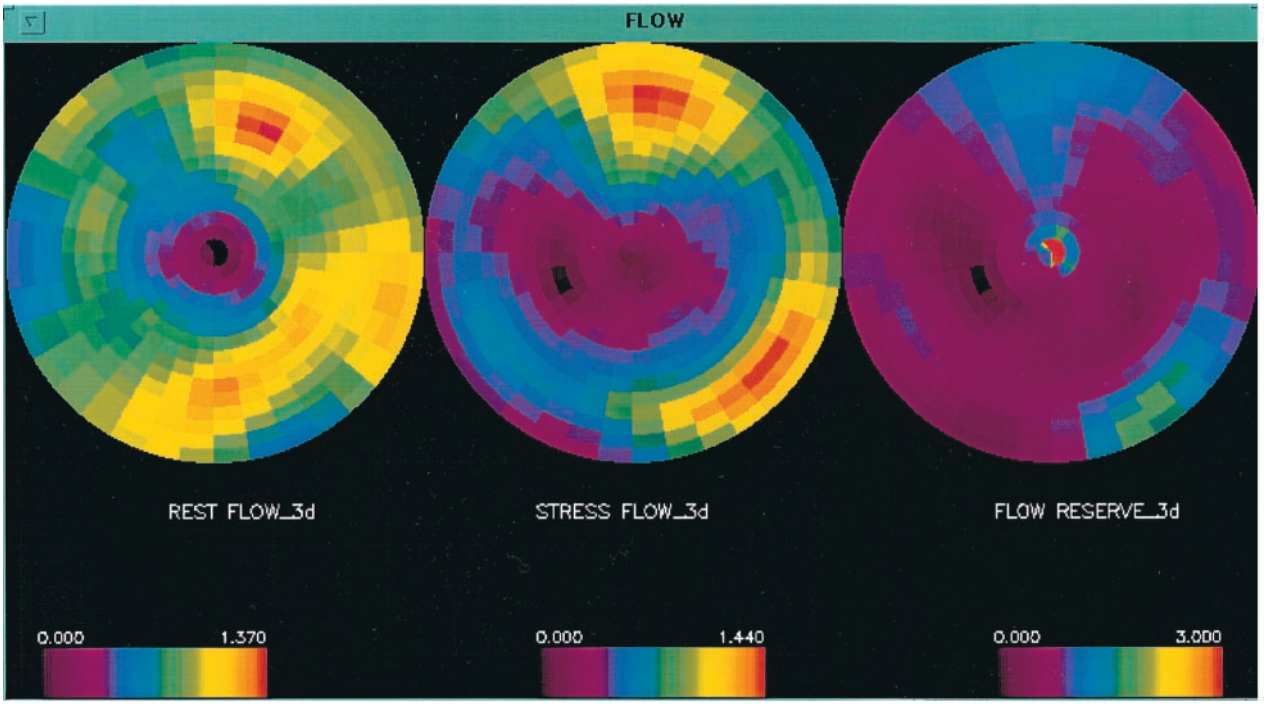
**PET image analysis.** Images were reconstructed with a Hann filter with critical frequency of 0.4 cycles per pixel, giving a spatial resolution of 10 mm in plane and 10 mm in the axial direction. The transaxial images were corrected for radionuclide decay and transferred to a Sun workstation for processing. The transaxial slices acquired in the last frame (900 s) were reoriented into eight short axis slices using standard system software. Each of the short axis slices was divided into eight myocardial sectors (each subtending 45 degrees). In addition, a 5 × 5-pixel region of interest (1.3 $cm^3$) was placed in the center of the left ventricular blood pool in several basal slices and averaged to comprise the input function. The sectors and the blood pool regions of interest were copied onto the initial 12 frames (120 s of data). Sectorial count data in these frames were used to generate myocardial time-activity curves. The arterial tracer input function was determined from the blood pool regions of interest in these initial 12 frames. The tissue time-activity curves were corrected for partial volume, assuming a wall thickness of 1 cm (17). Separate correction was made for blood-to-tissue activity spillover. Corrections were not made for metabolites from the breakdown of tracer because the quantitative analysis was limited to the data collected in the first 120 s, during which metabolite contamination is negligible (18). Myocardial blood flow quantitation (ml/100 g/min) was performed by fitting the decay-corrected time activity curves with a two-compartment model (19). Sectorial absolute flows were calculated in eight basal to midventricular short axis planes in each patient. Because variances in sectorial blood flow values down the planes were negligible, absolute blood flow in each sector was averaged deep through the planes.

For display purposes, functional polar maps were constructed to display absolute myocardial perfusion and flow reserve (Fig. 1). To construct these maps, which present myocardial perfusion in ml/100 g/min, dynamic polar map data were obtained from the system software, which uses both short axis and long axis slices. Tissue time-activity curves were then subjected to a nine-pixel averaging and were also averaged one plane above, and one plane below, the pixel of interest. This was repeated for all pixels. The input function was obtained from short axis slices as above.

**Definition of coronary steal.** Coronary steal was defined as an absolute decrease in blood flow from resting values in a segment following vasodilation.

**Analysis of the coronary angiograms.** All patients underwent coronary angiography using standard techniques within six months of enrollment into the study. Experienced observers who were blinded to the results of the PET studies reviewed coronary angiograms. Visual estimates of luminal narrowing of at least 50% in the left main artery and 75% in the other coronary vessels were considered significant. Both the origin and the recipient vascular beds of angiographic collaterals were noted. The vascular territories on the perfusion scans were divided, using standard criteria.

Collateral territories were defined as those supplied by collateral vessels in an area where the proximal vessel was occluded or severely narrowed. Collateral vessels were identified by late contrast filling. Careful correlations of the distribution and extent of noncollateralized and collateralized myocardium were then transferred to the PET images to co-localize these areas.

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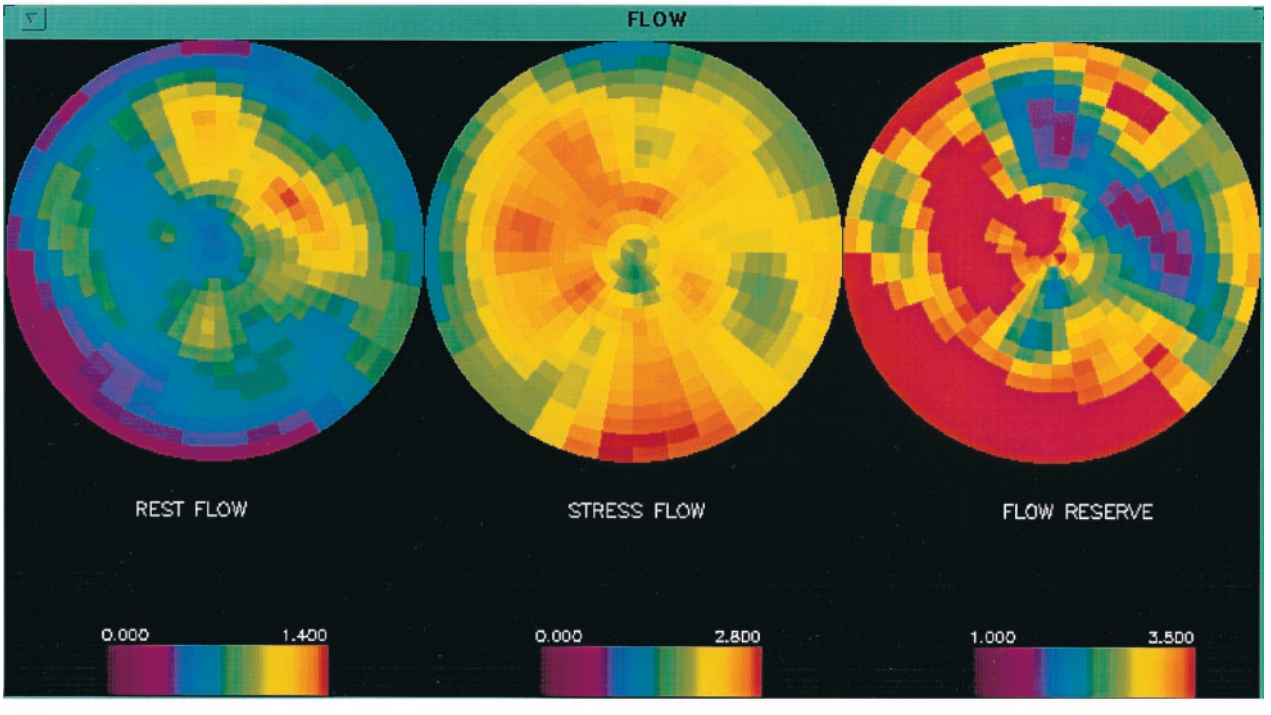


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**Figure 1.** Parametric polar map of myocardial perfusion at rest **(left)**, after coronary vasodilation with dipyridamole **(middle)**, and a map of myocardial perfusion reserve (produced by a pixel-by-pixel division of the post-dipyridamole map by the resting map) in a patient with coronary steal **(A)** and a patient without coronary steal **(B)**. The color scale represents absolute myocardial perfusion (ml/100 g/min) in the flow map, and a relative ratio in the map of myocardial perfusion reserve. Blood flow in the patient with steal decreases in absolute terms after dipyridamole, and myocardial perfusion reserve becomes <1. Polar maps represent myocardial perfusion with anterior to the top, lateral to the reader's right, inferior myocardium to the bottom, and septal myocardium to the reader's left. The apex is depicted in the center and the base at the periphery.

**Analysis of the ECG.** Observers who were blinded to the results of the PET studies performed ECG analysis. The occurrence of 0.1 mV horizontal or downsloping ST segment depression, 0.15 mV upsloping ST segment, 0.15 mV additional ST segment depression when there were resting ST segment depressions or the presence of 0.1 mV ST

**Table 1.** Clinical Characteristics in Patients With and Without Coronary Steal

| | Patients With Steal (n = 8) | Patients Without Steal (n = 10) | p Value |
|---|---|---|---|
| Age (yrs) | 63 ± 11 | 65 ± 11 | 0.72 |
| Male gender | 8 | 7 | 0.09 |
| Ejection fraction | 48 | 44 | 0.64 |
| Angina | 6 | 6 | 0.50 |
| Previous infarct | 2 | 5 | 0.28 |
| Previous CABG | 3 | 5 | 0.59 |
| Medications | | | |
| Beta-blockers | 5 | 2 | 0.07 |
| Calcium antagonists | 2 | 4 | 0.50 |
| Nitrates | 3 | 3 | 0.74 |
| ACE inhibitors | 1 | 1 | 0.87 |
| Aspirin | 4 | 4 | 0.67 |

ACE = angiotensin-converting enzyme; CABG = coronary artery bypass grafting.

segment elevation (in leads without Q-waves) were all considered significant. All measurements were taken at 60 ms after the J point.

**Statistical analysis.** Numerical data are presented as mean ± SD. Myocardial perfusion reserve and the hemodynamic and clinical variables were compared in patients with and without coronary steal using the Student *t*-test. The Fisher exact test and logistic regression analysis were performed to test for a relationship between coronary steal and the following variables: angina, ST depression, ST elevation, combination of ST depression and angina, baseline diastolic blood pressure (BP), hyperemic diastolic BP, change in diastolic BP, baseline systolic BP, hyperemic systolic BP, change in systolic BP, baseline rate-pressure product, hyperemic rate-pressure product, change in rate-pressure product, and change in heart rate.

## RESULTS

**Patient characteristics.** The study population comprised 18 patients with severe symptomatic multivessel CAD (Table 1). Seven patients had a prior history of myocardial infarction; of these, six were Q-wave infarcts. No patient had an infarct within six months of enrollment in the study. Eight patients had a history of prior coronary artery bypass grafting (CABG). All patients with bypass graft also had evidence of significant graft stenosis on angiograms.

Coronary steal was noted in at least one myocardial segment in 8 of the 18 patients studied. In these patients, by definition, absolute myocardial blood flow (MBF) was reduced in the segments defined as having coronary steal following coronary vasodilation compared with rest. Figure 1 shows representative polar maps of myocardial perfusion in two subjects—one with steal and one without evidence of steal. No significant difference was seen between patients with and without coronary steal in terms of age, gender, extent of CAD, history of previous myocardial infarction, history of previous CABG or use of anti-ischemia medications (Tables 1 and 2). The angiographic extent of CAD was comparable between patients with and without coronary steal with the exception of a higher prevalence of angiographic collaterals in the patients with coronary steal (Table 2).

**Table 2.** Angiographic Findings in Patients With and Without Coronary Steal

| | Patients With Steal (n = 8) | Patients Without Steal (n = 10) | p Value |
|---|---|---|---|
| Triple-vessel disease | 7 | 6 | NS |
| Double-vessel disease | 1 | 3 | NS |
| LAD stenosis (mean % stenosis) | 8 (92) | 9 (87) | NS |
| LCx stenosis (mean % stenosis) | 7 (82) | 9 (84) | NS |
| RCA stenosis (mean % stenosis) | 8 (78) | 8 (94) | NS |
| 100% occlusions | 4 | 6 | NS |
| Patients with angiographic evidence of collaterals | 8 | 4 | |

LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery.

**Hemodynamic data.** The results of the hemodynamic response to dipyridamole infusion are summarized in Table 3. Mean heart rate in all subjects increased from 61 ± 16 beats/min at baseline to 83 ± 15 following dipyridamole.

Mean arterial pressure did not change significantly following dipyridamole compared with baseline. Mean double product in all subjects increased from 9294 ± 3444 mm Hg·beats/min to 11704 ± 3144 mm Hg · beats/min (p = NS). Diastolic BP increased from 75 ± 12 mm Hg at baseline to 94 ± 13 mm Hg following dipyridamole (p = 0.05). No significant differences existed in hemodynamic variables studied between patients with and without evidence of coronary steal (Table 3).

**Clinical and ECG findings.** The ST segment depression was noted in eight patients. The mean number of ECG leads involved in the patients with ST depression was 2 ± 3; ST segment elevation in leads without Q waves was noted in five patients. The mean number of ECG leads with ST elevation was 2 ± 0.5. All patients with ST elevation had ST depressions. The ST segment depressions did not appear to be reciprocal to the elevations. Ten patients

**Table 3.** Hemodynamic Findings in Patients With and Without Coronary Steal

| | Patients With Steal (n = 8) | Patients Without Steal (n = 10) | p Value |
|---|---|---|---|
| Baseline diastolic BP | 79 ± 9 | 73 ± 16 | 0.36 |
| Hyperemic diastolic BP | 82 ± 10 | 75 ± 15 | 0.26 |
| Change in diastolic BP | 3 ± 9 | 2 ± 13 | 0.83 |
| Baseline rate-pressure product | 9615 ± 2626 | 9037 ± 4108 | 0.73 |
| Hyperemic rate-pressure product | 11456 ± 2016 | 11902 ± 3926 | 0.77 |
| Change in rate-pressure product | 1841 ± 2587 | 2865 ± 3731 | 0.52 |
| Baseline systolic BP | 151 ± 20 | 140 ± 31 | 0.41 |
| Hyperemic systolic BP | 143 ± 19 | 140 ± 36 | 0.83 |
| Change in systolic BP | −8 ± 20 | 0 ± 27 | 0.52 |
| Change in heart rate | 17 ± 9 | 19 ± 13 | 0.81 |

BP = blood pressure.

**Table 4.** Clinical and Electrocardiographic Findings During Coronary Vasodilation in Patients With and Without Coronary Steal

| | Patients With Steal (n = 8) | Patients Without Steal (n = 10) | p Value |
|---|---|---|---|
| Angina | 6 | 4 | 0.06 |
| Patients with ST depression | 5 | 3 | 0.16 |
| Patients with ST depression and angina | 4 | 1 | 0.04 |
| Mean No. of ECG leads with ST depression | 2 ± 3 | 1 ± 2 | 0.17 |
| No. of patients with ST elevation | 5 | 0 | < 0.001 |

ECG = electrocardiogram.

complained of angina following dipyridamole (Table 4). There was a significant difference in the occurrence of ST elevation (p = 0.002) and the combination of ST depression and angina (p = 0.04) between patients with and without coronary steal. There was no significant difference between the two groups in the prevalence of ST depression alone. The incidence of angina alone following dipyridamole infusion tended to be higher in the patients with coronary steal (p = 0.06).

**Quantitation of coronary steal.** Coronary steal was noted in 8 of the 18 patients studied. Of the total 64 myocardial regions (8 sectors per short axis plane averaged from base to apex) in these eight patients, coronary steal was noted in 24 segments. Mean resting flow in these 24 segments was 90 ± 18 ml/100 g/min. Following hyperemia, flow decreased significantly to 68 ± 27 ml/100 g/min (p < 0.001) instead of the expected increase. Thus, absolute MBF in segments with coronary steal decreased by 22 ± 10 ml/100 g/min (12 ± 5%) following dipyridamole compared with baseline. In the remaining 40 segments, flow increased from 87 ± 19 ml/100 g/min at rest to 138 ± 56 ml/100 g/min following dipyridamole (Fig. 2). Myocardial flow reserve, which is the ratio of hyperemic to rest flow, was 0.75 ± 0.1 in segments with steal and 1.6 ± 0.3 in segments without steal (p < 0.001).

A comparison of absolute MBF in patients with and without coronary steal in at least one segment revealed a higher resting flow in patients with coronary steal (89 ± 25 and 75 ± 21 ml/100 g/min in patients with and without steal, respectively (p = 0.01). Mean hyperemic flow was significantly lower in patients with steal than in patients without steal, 114 ± 67 vs. 144 ± 57 ml/100 g/min, respectively (p < 0.001; Fig. 2). Mean myocardial perfusion reserve in patients with coronary steal was 1.3 ± 0.2 compared with 1.9 ± 0.3 in patients without steal (p < 0.001).

**Angiographic correlation.** Twelve of 18 patients had angiographic evidence of collaterals. Coronary steal was identified only in patients with angiographic evidence of collaterals. Thus, of the eight patients who demonstrated coronary steal defined with PET, all had angiographic

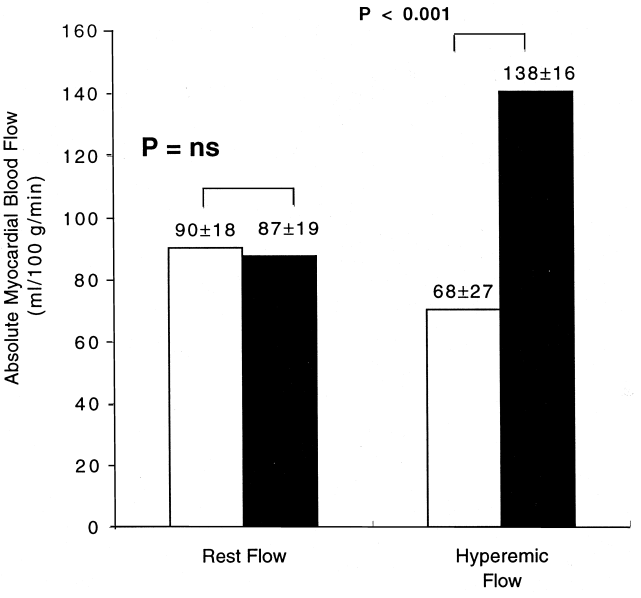


**Figure 2.** Histogram of myocardial perfusion in hearts from patients who exhibited steal in at least one segment. In these patients, as depicted, myocardial perfusion at rest in segments with and without steal was equivalent. However, after dipyridamole, segments with steal exhibited an absolute reduction in perfusion, whereas segments without steal vasodilated to similar levels as those observed in patients without steal. **Open bars**: segments with steal (n = 24); **solid bars**: segments without steal (n = 40).

evidence of collateral circulation. In comparison, in the 10 patients without coronary steal, only 4 had evidence of collateral circulation (p = 0.04) (Table 3). The collateral supply in six of eight patients with coronary steal and four of four patients without coronary steal were qualitatively assessed as "good." All myocardial segments with absolute decreases in flow following induction of hyperemia, compared with rest, had corresponding angiographic evidence of collaterals. Eight of these 24 segments were in territories supplied by vessels with total occlusions.

**Clinical correlates of coronary steal.** Table 5 shows the significant clinical correlates of coronary steal. The combination of ST segment depression with the patients' typical angina, and ST segment elevation, were the only significant clinical correlates of coronary steal on univariate analysis. The ST segment depression alone did not correlate with coronary steal. The relationship between angina alone and coronary steal was marginal (p = 0.06). There was no relationship between presence of coronary steal and any of the hemodynamic variables tested.

Of all the variables tested, ST segment elevation in any lead was most strongly correlated with steal (p = 0.006). On stepwise logistic regression, the addition of other variables did not significantly improve the relationship between ST elevation and coronary steal.

## DISCUSSION

Coronary steal has been well described in animal studies (15,16). It is believed to be due to a decrease in supply via collaterals to dependent myocardium following coronary

**Table 5.** Correlates of Coronary Steal

| | p Value | Sensitivity (%) | Specificity (%) | Positive Predictive Value (%) | Negative Predictive Value (%) |
|---|---|---|---|---|---|
| ST elevation | < 0.001 | 63 | 100 | 100 | 77 |
| Angina with ST depression | 0.04 | 71 | 90 | 80 | 82 |
| Angina | 0.06 | | | | |
| ST depression | NS | | | | |
| Baseline rate-pressure product | NS | | | | |
| Hyperemic rate-pressure product | NS | | | | |
| Change in rate-pressure product | NS | | | | |
| Baseline diastolic BP | NS | | | | |
| Hyperemic diastolic BP | NS | | | | |
| Change in diastolic BP | NS | | | | |
| Baseline heart rate | NS | | | | |
| Hyperemic heart rate | NS | | | | |
| Change in heart rate | NS | | | | |

BP = blood pressure.

vasodilation (12). The cause is diminished driving pressure at the proximal end of the collateral vessels due to decreased resistance in the supplying, conductance artery as well as a nominal or blunted fall in resistance in the collateral circuit (16). Consequently, we specifically selected patients with advanced multiple-vessel CAD in order to maximize our chances of eliciting the coronary steal phenomenon.

**Absolute quantification of coronary steal.** Our results in this population of patients with multivessel CAD show that the magnitude of coronary steal is quite significant. Absolute myocardial blood in the affected segments decreased by 22 ± 10 ml/100 g/min. Rest flow was also noted to be significantly higher in patients with coronary steal than in those without steal. The increased resting flow in patients with steal might be attributable to the higher double product in these patients (although not statistically significant). In addition, a difference in wall stress, which was not measured in this study, could account for the difference in resting flow between the two groups. Mean myocardial perfusion reserve in the segments with coronary steal in our study (0.75 ± 0.18) is consistent with the observation of a coronary flow reserve velocity ratio of 0.7 in collateralized myocardium, following infusion of adenosine, reported by Seiler et al. using invasive techniques (20). In addition, this magnitude is consistent with changes in myocardial perfusion following pharmacologic vasodilation reported by other investigators (21–23). Baliga et al. (21) demonstrated a 25% decrease in blood flow to regions supplied by stenotic coronary arteries in patients with postprandial angina. The reduction in flow was presumably due to redistribution of flow away from the diseased regions.

Other investigators have also demonstrated comparable magnitudes of coronary steal by PET measured semiquantitatively (12,24). In those studies, coronary steal was defined as a decrease in regional tracer counts following pharmacologic vasodilation compared with rest counts. Garza et al. (24) noted a hyperemia/rest count ratio of 0.8 in patients with coronary collaterals following infusion of dipyridamole, and Demer et al. (12) demonstrated a mean decrease of 15% in counts following induction of hyperemia compared with rest in collateralized myocardium. However, in these semiquantitative studies, absolute flow quantitation was not performed.

Skopicki et al. (23) demonstrated an absolute decline in myocardial perfusion in patients with collateralized myocardium, using PET and $^{13}NH_3$. In their study, flow in normal segments increased from 90 to 310 ml/100 g/min, whereas flow in segments with steal decreased from 92 to 50 ml/100 g/min. Severi et al. (22) similarly demonstrated that myocardial segments that develop a wall-motion abnormality in response to dobutamine have an absolute decrease in myocardial perfusion when assessed with oxygen-15–labeled water.

Our results contrast with the findings in the study by McFalls et al. (25), which showed increased flow relative to baseline in collateralized myocardium following infusion of dipyridamole. The absence of an absolute decrease in flow to collateralized myocardium in their study might have been due to the fact that only patients with single-vessel CAD were studied. As demonstrated in studies in animals by Becker (16), for coronary steal to occur, it is important to have some degree of stenosis in the donor vessel proximal to the origin of the collaterals in addition to the stenosis in the recipient vascular bed. Thus, coronary steal will tend to occur mainly in the presence of multivessel CAD.

It should be recognized that even in segments that do not exhibit coronary steal, vasodilator reserve is abnormal. This is consistent with findings in patients with ischemic heart disease.

**Clinical correlates of coronary steal.** Of all the variables tested, only the combination of ST segment depression and angina ($p = 0.040$) or ST elevation ($p = 0.006$) correlated with coronary steal. The ST segment depression alone did not correlate with coronary steal. Furthermore, the relationship between angina alone and coronary steal was marginal ($p = 0.06$). The other variables, including extent of CAD,

baseline rate-pressure product, hyperemic rate-pressure product, change in rate-pressure product, and baseline, hyperemic and change in diastolic pressure and heart rate did not correlate with coronary steal.

The ST segment elevation during pharmacologic stress testing is a highly specific but insensitive marker of severe CAD. The presence of ST elevation during exercise generally signifies transmural ischemia. Thus, the relatively high *incidence* of ST elevation in this study is most likely due to the fact that we studied only patients with severe multivessel CAD.

Our findings support the routine administration of IV aminophyline to reverse the effects of dipyridamole in all patients with either ST elevation or the combination of angina and ST depression, given their strong correlation with coronary steal.

Previous studies have shown a relationship between ST segment depression during vasodilator-induced hyperemia and the following variables: decrease in arterial BP (26), increase in rate-pressure product (27), triple-vessel disease (27), presence of collaterals (3,14,26), systolic BP at baseline (14), and angina (14). These studies used ST segment depression during hyperemia as a marker of ischemia and, consequently, coronary steal. However, using decreases in absolute flow from rest to define ischemia, we found a relationship between ST segment depression and coronary steal only when the former is accompanied by angina. Thus, our data suggest that either the combination of ST depression and angina or ST elevation should be used as clinical evidence of ischemia during dipyridamole infusion rather than ST depression alone.

All of the hemodynamic variables studied, including change in rate-pressure product, systolic BP at baseline and decrease in diastolic BP did not correlate with coronary steal. Thus, it is unlikely that the mechanism of the observed reduction in flow following induction of hyperemia is related to a decrease in perfusion pressure of the entire coronary circulation.

**Study limitations.** The limitations of this study should be recognized: First, the study size was small and selected for patients with severe CAD. Thus, the sample might not represent the average population of patients referred for pharmacologic stress testing. Second, matched normal controls were not studied.

**Conclusions.** In conclusion, coronary vasodilation with IV dipyridamole is associated with a significant absolute reduction in blood flow to collateral-dependent myocardium consistent with coronary steal. The combination of angina and ST depression and ST elevation were the only clinical correlates of coronary steal.

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