Direct Assessment of Coronary Steal and Associated Changes of Collateral Hemodynamics in Chronic Total Coronary Occlusions

Gerald S. Werner, MD; Hans R. Figulla, MD

Background—Coronary steal can occur in collateral-dependent myocardium during pharmacologically induced vasodilation. This study assessed coronary steal invasively in chronic total coronary occlusions (TCOs).

Methods and Results—In 35 consecutive patients with a percutaneous transluminal coronary angioplasty of a TCO (duration >4 weeks), coronary flow velocity (APV) by a Doppler wire and distal pressure (Pd) by a pressure wire were assessed in the collateral-dependent vascular bed before dilatation. Indexes of peripheral resistance (Rp) and for the collateral pathway, including the donor artery segment (Rcp), were calculated. Changes of these parameters were assessed during intravenous adenosine (140 μg·kg⁻¹·min⁻¹). Adenosine caused a decrease of APV, ie, coronary steal, in 13 patients (37%; group S), an increase in 11 patients (group R), and no change in 11 patients (group N). Angiographic analysis of collateral pathways showed no difference between the groups, except that in group S all collateral connections were continuously visible but no large collaterals (>0.5 mm) were found. In group N, collaterals were least developed. The increase of APV in group R was associated with a decrease of Rp, whereas Rcp remained unchanged. In contrast, group S showed no change in Rp but a significant increase of Rcp, indicating an increased resistance of the donor segment.

Conclusions—Coronary steal is observed in about one third of TCOs and is associated with specific hemodynamic changes of Rp and Rcp. Steal occurred only with well-developed angiographically visible collaterals but not with very large collaterals. (Circulation. 2002;106:435-440.)

Key Words: collateral circulation • coronary disease • vasodilation
The PTCA was done as previously described. After the lesion was crossed by a 0.014-inch guide wire, an over-the-wire exchange catheter (Transit, Cordis) or low-profile balloon catheter (Ranger, Scimed) was advanced distal to the occlusion. The guide wire was exchanged for a pressure recording wire (PressureWire, RADI Medical Systems). The distal coronary pressure ($P_{P_r}$) was recorded together with the aortic pressure ($P_{Ao}$). The fractional collateral flow $Q_{CP}/Q_T$ during hyperemia was calculated $(P_{P_r}−P_{RA})/(P_{Ao}−P_{RA})^{2,23}$ where $P_{RA}$ as the right atrial pressure was substituted for by 5 mm Hg.

The pressure wire was then exchanged for the Doppler wire (FloWire, JoMed). An unaccounted contribution of antegrade flow along the exchange catheter was ruled out in all patients by lack of contrast passage along the over-the-wire catheter during proximal contrast injection into the recanalized artery and no effect on the distal Doppler signal. All Doppler flow signals were measured manually, as previously described. The velocity integral during systole and diastole and the duration of systole and diastole were measured to calculate the average peak velocity (APV). A collateral flow reserve (CFI) was calculated before recanalization and during the final balloon occlusion as the ratio of distal APV/antegrade APV. The latter obtained at the same location after PTCA. A peripheral resistance index was calculated as $R_{PA}=(P_{P_r}/P_{Ao})/APV$ (mm Hg · cm$^2$ · s$^{-1}$). The resistance index of the collateral supply pathway was defined as $R_{CP}=(P_{Ao}−P_{RA})/APV$ (mm Hg · cm$^2$ · s$^{-1}$), incorporating both the resistance of the collateral vessel and of the donor segment proximal to the collateral takeoff (Figure 1).

**Study Protocol**

The baseline recordings started with distal pressure, followed by the Doppler flow velocity. These measurements were repeated during intravenous adenosine infusion (140 μg · kg$^{-1}$ · min$^{-1}$), but because pressure recordings are less affected by the exact wire position than the Doppler flow signal, adenosine was started with the Doppler wire kept in a constant position after the baseline measurement. The APV was recorded until 3 minutes after the start of the adenosine infusion. During continuing infusion, the Doppler wire was exchanged for the pressure wire and $P_{P_r}$ and $P_{Ao}$ were obtained. Adenosine infusion was stopped after another 2 minutes, and $P_{P_r}$ and $P_{Ao}$ were recorded until they had returned to their baseline values. From the measurements during adenosine infusion, CFI and derived indexes were again calculated.

**Study Groups**

The collateral flow reserve is the ratio of APV during adenosine infusion and APV at baseline. The spontaneous variability of the Doppler collateral signal was 15%, as determined by continuous...
analysis in 7 patients. Therefore, a change of the collateral flow reserve was considered significant from 1.0 by \(0.15\). A collateral flow reserve \(0.85\), or coronary steal, was observed in 13 patients (group S; Figure 3). A collateral flow reserve \(1.15\) was observed in 11 patients (31%) (group R), and no significant change was observed in 11 patients (31%) (group N).

**Statistics**

Data are given as mean ± SD. Comparisons of continuous variables among the 3 groups were done by ANOVA and a post-hoc Scheffé test. Categorical variables were compared by a Fisher’s exact test. Repeated-measures ANOVA was used to compare parameter changes during adenosine infusion. The correlation between 2 parameters was assessed by linear regression analysis. \(P<0.05\) was considered significant. All calculations were done on a personal computer with SPSS for Windows (Version 10.05, SPSS Inc).

**Results**

**Clinical Variables**

Patients had a right (60%) or left (40%) coronary occlusion similarly distributed within the study groups. There was no difference in age, sex, history of prior myocardial infarction, extent of coronary artery disease, regional dysfunction, and clinical symptoms, but there was a trend toward more diabetic patients in group N (Table 1). No differences in medication, such as \(\beta\)-blocker, were observed. A circumscript stenosis in the donor artery proximal to the takeoff of a collateral was observed in only 5 patients, 1 in group S and 2 each in the other groups.

**Collateral Flow Pattern and Flow Duration**

Patients of group S and group R showed a biphasic diastolic/systolic collateral flow pattern in 23 of 24 cases (96%) (Figure 3). Patients of group N showed this pattern in only 4 of 11 cases (36%) \((P<0.001)\). Collateral flow occurred during the complete cardiac cycle in 77% in group S and in 73% in group R but in only 27% in group N \((P=0.027)\). In group R the diastolic/systolic APV ratio tended to be higher than in group S (Table 2).

**Collateral Function During Adenosine Infusion**

Adenosine increased the heart rate in all groups \((67 ± 11 \text{min}^{-1} \text{to } 78 ± 17 \text{min}^{-1}; P<0.001)\). The collateral flow reserve was \(0.65 ± 0.17\) in group S, \(1.40 ± 0.16\) in group R, and \(1.02 ± 0.10\) in group N. Both baseline Doppler and pressure parameters and their changes during adenosine infusion are summarized in Table 2. Group N showed a lower diastolic APV and diastolic/systolic velocity ratio and a lower CFI compared with the other groups. The \(Q_S/Q_N\) was highest in group N. Flow and pressure indexes were not correlated \((r=0.16; P=0.38)\). Collateral flow reserve was independent of the effect of adenosine on \(P_D\), which decreased even in group R while CFI increased (Figure 4).

There was a distinct difference in the effect of adenosine on \(R_P\) in groups R and S (Figure 5). \(R_P\) was slightly higher at baseline in group S, and it decreased significantly after adenosine only in group R. In contrast, \(R_{SP}\) remained unchanged in group R but increased in group S (Figure 5). No significant changes of \(R_P\) and \(R_{SP}\) were observed in group N (Table 2).

**Collateral Anatomy and Flow Reserve**

An average of 2.1 ± 0.6 collateral pathways was observed in each patient, similar between groups. The principal pathways
were septal in 46%, atrial in 29%, branch-branch in 17%, and bridging in 9%, equally distributed between the study groups (x², P=0.94). In group S, no large collaterals or small discontinuous connections were observed, and all had continuous collateral connections of size 1 (77%) and 2 (23%) (ie, between 0.1 and 0.5 mm). In group R, all collateral pathways and sizes were found, and only one had discontinuous connections. Of the 3 patients with very large collaterals (size 3), all were in group R; these were the collaterals with the lowest RCP (3.7±0.1 mm Hg · cm⁻¹ · s⁻¹) compared with smaller collaterals (8.5±4.8 mm Hg · cm⁻¹ · s⁻¹). In group N, most patients had discontinuous or size 1 connections (82%).

Discussion

Previous Studies on the Effect of Adenosine on Collateral Function

Two groups studied the effect of adenosine on collaterals during balloon occlusion. They reported an increase of collateral flow, but a few patients also showed a decrease, ie, coronary steal. Another study assessed Doppler coronary collateral flow, but a few patients also showed a decrease, ie, most patients had discontinuous or size 1 connections (82%).

Comparison With Scintigraphic Studies

The present study was done under baseline conditions similar to those in scintigraphic studies. Coronary steal was observed in one third of patients. Another one third had a collateral flow reserve >1. When group S is compared with quantitative PET studies, the values of collateral flow reserve (0.65±0.17) are in a similar range.

TABLE 1. Clinical Characteristics of Patients With TCOs

<table>
<thead>
<tr>
<th></th>
<th>Group S</th>
<th>Group R</th>
<th>Group N</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>60.7±12.8</td>
<td>59.6±9.6</td>
<td>63.8±10.9</td>
<td>0.66</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>8 (62)</td>
<td>10 (91)</td>
<td>8 (73)</td>
<td>0.39</td>
</tr>
<tr>
<td>Angina pectoris (CCS 0–4)</td>
<td>0/1/5/70</td>
<td>0/0/3/80</td>
<td>0/0/5/1</td>
<td>0.53</td>
</tr>
<tr>
<td>Heart failure (NYHA 0–4)</td>
<td>1/5/6/10</td>
<td>0/6/5/0/0</td>
<td>0/2/6/3/0</td>
<td>0.27</td>
</tr>
<tr>
<td>Diabetes, n (%)</td>
<td>3 (23)</td>
<td>1 (10)</td>
<td>5 (45)</td>
<td>0.15</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>11 (85)</td>
<td>9 (82)</td>
<td>9 (82)</td>
<td>0.98</td>
</tr>
<tr>
<td>No. of diseased arteries, 1/2/3</td>
<td>4/63</td>
<td>7/2/2</td>
<td>5/5/1</td>
<td>0.46</td>
</tr>
<tr>
<td>Duration of occlusion≥3 months, n (%)</td>
<td>5 (38)</td>
<td>6 (55)</td>
<td>6 (55)</td>
<td>0.66</td>
</tr>
<tr>
<td>Prior Q-wave myocardial infarction, n (%)</td>
<td>7 (54)</td>
<td>7 (64)</td>
<td>8 (73)</td>
<td>0.63</td>
</tr>
<tr>
<td>Regional dysfunction, n (%)</td>
<td>10 (77)</td>
<td>7 (64)</td>
<td>8 (73)</td>
<td>0.77</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>62.5±11.2</td>
<td>68.6±18.8</td>
<td>60.1±18.9</td>
<td>0.47</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
<td>18.0±8.0</td>
<td>15.8±6.0</td>
<td>20.1±10.2</td>
<td>0.55</td>
</tr>
</tbody>
</table>

CCS indicates Canadian Cardiovascular Society classification of chest pain; LVEDP, left ventricular end diastolic pressure; and NYHA, New York Heart Association classification of heart failure.

TABLE 2. Collateral Flow, Pressure, and Resistance Indexes in TCO Before and After Adenosine Infusion

<table>
<thead>
<tr>
<th></th>
<th>Group S Before Adenosine</th>
<th>Group R Before Adenosine</th>
<th>Group N Before Adenosine</th>
<th>P</th>
<th>Group S After Adenosine</th>
<th>Group R After Adenosine</th>
<th>Group N After Adenosine</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>APV Occl, cm/s</td>
<td>11.3±6.0</td>
<td>7.6±4.8</td>
<td>&lt;0.001</td>
<td></td>
<td>8.4±3.9</td>
<td>11.8±5.3</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Diastolic/systolic APV ratio</td>
<td>0.82±0.42</td>
<td>0.71±0.62</td>
<td>0.72</td>
<td></td>
<td>1.40±0.72</td>
<td>1.39±1.24</td>
<td>0.97</td>
<td></td>
</tr>
<tr>
<td>Pw, mean, mm Hg</td>
<td>109±16</td>
<td>94±11</td>
<td>0.006</td>
<td></td>
<td>107±19</td>
<td>97±21</td>
<td>0.13</td>
<td></td>
</tr>
<tr>
<td>Ps, mean, mm Hg</td>
<td>42±11</td>
<td>29±6</td>
<td>&lt;0.001</td>
<td></td>
<td>53±15</td>
<td>36±14</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>CFI</td>
<td>0.36±0.21</td>
<td>0.24±0.16‡</td>
<td>&lt;0.001</td>
<td></td>
<td>0.35±0.23</td>
<td>0.49±0.36</td>
<td>0.009</td>
<td></td>
</tr>
<tr>
<td>Qp/Qs</td>
<td>0.25±0.07</td>
<td>0.30±0.08</td>
<td>0.38±0.14*</td>
<td></td>
<td>0.25±0.07</td>
<td>0.30±0.08</td>
<td>0.38±0.14*</td>
<td></td>
</tr>
<tr>
<td>Rp, mm Hg · cm⁻¹ · s⁻¹</td>
<td>8.35±5.15</td>
<td>17.63±17.17</td>
<td>0.02</td>
<td></td>
<td>8.36±4.86</td>
<td>8.07±5.04</td>
<td>0.57</td>
<td></td>
</tr>
<tr>
<td>Rn, mm Hg · cm⁻¹ · s⁻¹</td>
<td>5.75±4.54</td>
<td>7.42±5.76</td>
<td>0.18</td>
<td></td>
<td>7.92±4.04</td>
<td>4.39±2.36</td>
<td>0.001</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations as in Figure 1. P values for changes within groups; difference between group N and S, *P<0.05; between group N and R, †P<0.05; between group S and R, ‡P<0.05.
steal, flow increased >4 times in selected patients without myocardial infarction and regional dysfunction,1,2 but in patients with regional dysfunction2 and multivessel disease,4,5,13 the flow reserve was similar to our study (1.40±0.16). Even though we measured collateral-dependent flow velocity in an epicardial artery and scintigraphy assesses myocardial perfusion, this quantitative agreement supports the validity of the invasive approach.

Direct Confirmation of the Collateral Network Model in Man
Gould and colleagues9,25 specified the following 3 assumptions required for the occurrence of steal: (1) the collateral resistance is not negligible; (2) the microvasculature distal to the occlusion, being already maximally dilated, lacks a vasodilation reserve, and (3) the epicardial resistance of the supply artery causes a pressure drop proximal to the collateral origin during adenosine-induced hyperemic flow. All 3 assumptions are supported by our data. First, coronary steal required well-developed collaterals,14,16,24 whereas collaterals in group N had a lower CFI, higher RCP, and predominantly a systolic collateral flow pattern, as evidence of reduced collateral function.18,26

As shown previously, collateral resistance is not negligible.14,16,18 The assumption that large collaterals, because of a low resistance, would not show steal25 was confirmed by our study, because large collaterals were only observed in group R but not in group S. The lack of a vasodilatory reserve is demonstrated by the unchanged RP in group S, whereas it was decreased significantly in group R. On the other hand, the higher RP in group N and its lack of response to adenosine could be related to the higher proportion of diabetics with a known prevalence of microvascular dysfunction.27

We did not record directly the coronary pressure in the donor artery at the collateral origin. The multiple coexisting collateral pathways make such an approach difficult and unreliable; in only 2 of 13 patients with steal we observed a singular collateral takeoff. The increase of RCP in patients with steal during adenosine infusion represented an increased resistance of the complete collateral supply pathway, which includes the donor artery segment proximal to the collateral takeoff (Figure 1). Assuming a constant collateral resistance, the increase of RCP indicated an increase of the donor segment resistance or, stated another way, decrease of distal coronary donor pressure. This does not require a circumscript epicardial stenosis, as recently demonstrated by a pressure drop during hyperemia in angiographically normal segments of patients with coronary artery disease attributable to diffuse atherosclerosis.28,29

Collateral Pathway and Coronary Steal
Angiography is of limited value for assessing function of collaterals,23,30 but it illustrates their anatomy.31 Angiographic assessment of collaterals is mostly semiquantitative.32 A refined quantitative approach was recently suggested but requires high-quality cine films.20 With the limitation of a lower spatial resolution of digital media, we analyzed the collateral anatomy and size relative to the capillary bed to test an extension of the network model of coronary steal. It was suggested that prearteriolar collaterals would be required for coronary steal.25 This was confirmed in group S, where all collaterals showed continuous connections between donor and recipient segments. Another assumption was that an increase of flow would occur in barely visible postarteriolar collaterals that pass through the microvascular bed of the donor artery. Their flow capacity should increase with peripheral vasodilation of the donor microcirculation. This could not be confirmed in our study, because these postarteriolar collaterals were predominantly observed in group N and indicated a low collateral function. Visible connections were the principal requisite for the incidence of coronary steal but also for a positive collateral flow reserve. However, there was no anatomic pathway that predicted the response to adenosine.
Study Limitations
Flow and pressure recorded distal to an occlusion are approximations of collateral function, because they assess only that part of collateral perfusion that reaches the occluded epicardial artery. Collateral perfusion through intramyocardial pathways may be underestimated. An increase of collateral flow velocity during adenosine might lead to a shear stress–related vasodilatation and, consequently, an underestimation of true volume flow. However, the flow velocities involved were far below those evoked during the observation of flow-mediated epicardial vasodilatation.33 Nitroglycerine application would have avoided the influence of minor diameter changes, but because nitroglycerine itself influences collateral hemodynamics,14 we did not apply it during collateral flow measurements. Right atrial pressure (PRA) was not directly measured. This influences the calculation of absolute values of pressure indexes, but because PRA does not considerably change during adenosine infusion,22,34 it would not decisively affect the assessment of the intraindividual response to adenosine.

Clinical Implications
In patients with coronary artery disease, coronary steal can occur without a circumscript donor artery lesion but requires the presence of diffuse atherosclerosis that causes a pressure gradient in the donor artery during hyperemia. We could not detect clinical discriminators of coronary steal in TCOs. Steal but also a collateral flow reserve >1 occurred irrespective of regional myocardial function. Some angiographic characteristics of collaterals, such as visible but not very large collateral connections (0.1 to 0.5 mm), are mandatory for the coronary steal phenomenon, but it cannot be predicted whether steal or a positive collateral flow reserve would occur.

References
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