Coronary Collateral Quantitation in Patients With Coronary Artery Disease Using Intravascular Flow Velocity or Pressure Measurements

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Objectives. This study evaluated two methods for the quantitative measurement of collaterals using intracoronary (IC) blood flow velocity or pressure measurements.

Background. The extent of myocardial necrosis after coronary artery occlusion is substantially influenced by the collateral circulation. So far, qualitative methods have been available to assess the human coronary collateral circulation, thus restraining the conclusive investigation of, for example, therapies to promote collateral development.

Methods. Fifty-one patients with a coronary artery stenosis to be treated by percutaneous transluminal coronary angioplasty (PTCA) were investigated using IC PTCA guidewire-based Doppler and pressure sensors positioned distal to the stenosis. Simultaneous measurements of aortic pressure, IC velocity and pressure distal to the stenosis during and after PTCA provided the variables for calculating collateral flow indices (CFIv and CFIp) that express collateral flow as a fraction of flow via the patent vessel. Both CFIv and CFIp were compared with conventional methods for collateral assessment, among them ST-segment changes >1 mm on IC and surface electrocardiogram (ECG) at PTCA. Also, CFIv and CFIp were compared with each other.

Results. In 11 patients without ECG signs of ischemia during PTCA (sufficient collaterals), relative collateral flow amounted to 46% as determined by Doppler and pressure wire. Patients with insufficient collaterals (n = 40) had relative collateral flow values of 18%. Using a threshold of CFI = 30%, sufficient and insufficient collaterals could be diagnosed with 100% sensitivity and 93% specificity by IC Doppler, and 75% sensitivity and 92% specificity by IC pressure measurements. The agreement between Doppler and pressure measurements was good: CFIv = 0.08 + 0.8 CFIp, r = 0.80, p = 0.0001.

Conclusions. Intracoronary flow velocity or pressure measurements during routine PTCA represent an accurate and, at last, quantitative method for assessing the coronary collateral circulation in humans.

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The coronary collateral circulation is an alternative source of blood supply to a myocardial area jeopardized by the failure of the stenotic or occluded vessel to provide enough blood flow to this region. There have been numerous studies demonstrating a protective role of collaterals in hearts with coronary artery disease (CAD) showing smaller infarcts (1), less ventricular aneurysm formation, improved ventricular function (1,2) and better survival (3) compared with patients in whom collaterals were not visualized. However, the functional relevance of coronary collaterals in humans has also been a matter of debate for many years (4–6). Much of the controversy is probably due to inadequate means available in the past to assess human collaterals. In animal models, quantitative determination of coronary collateral flow has been performed by radiolabeled microspheres (7) or by direct measurement of retrograde flow and pressure (8). In patients, only a semiquantitative angiographic grading method to estimate the extent of spontaneously visible collateral channels (4) or acutely recruitable collaterals during angioplasty has been accomplished (9,10). The recent advent of ultrathin Doppler and pressure angioplasty guidewires has made it possible to measure flow velocity or pressure in remote vascular areas and, thus, to obtain functional variables for studying the pathophysiology of the human coronary circulation, including the hemodynamic relevance of coronary stenoses (11–13) and collaterals (14–16).

The theoretical basis for the use of intracoronary (IC) flow velocity or pressure measurements to determine collateral flow relates to the fact that velocity or perfusion pressure signals (greater than the central venous pressure [CVP]) obtained from collaterals (Fig. 1). The measurement of such signals provides the variables for the calculation of a flow velocity- (17) and pressure-derived collateral flow index (CFIv and CFIp) (18), both of which express the amount of flow via collaterals to the vascular region of interest as a fraction of the flow via the normally patent vessel (Fig. 1). Such a CFI could be regarded as the standard for the assessment of human coronary collat.
erals provided that its usefulness is demonstrated. Therefore, the purpose of this study was to compare Doppler-wire– and pressure-wire– obtained collateral indices with each other and with three traditional methods for the assessment of the collateral circulation, that is, IC electrocardiogram (ECG) signs of myocardial ischemia (19) and angina pectoris during balloon occlusion and the angiographic degree of collateralization (4,9).

Methods

Patients. Fifty-one patients (age 58 ± 10 years, 36 men, 15 women) with one- to two-vessel CAD and without Q-wave myocardial infarction were included in the study. All underwent percutaneous transluminal coronary angioplasty (PTCA) because of CAD-related symptoms of at least one stenotic lesion.

This investigation was approved by the institutional ethics committee, and the patients gave informed consent to participate in the study.

The study population was divided into two groups, namely, patients with coronary collaterals sufficient (n = 11) and patients with collaterals insufficient (n = 40) to prevent ECG signs of myocardial ischemia during the first balloon occlusion of the stenosis to be revascularized. Myocardial ischemia was defined as ST-segment changes (>1 mm) present on any of three surface leads or on an IC ECG lead, the latter obtained from the angioplasty guidewire (19) (Fig. 2).

Cardiac catheterization and coronary angiography. Patients underwent left heart catheterization for diagnostic purposes. Aortic pressure was measured using the PTCA guiding catheter. Biplane left ventricular angiography was performed followed by coronary angiography. Coronary artery stenoses were estimated qualitatively as percent diameter reduction. Angiographic collateral degrees (0 to 3) were determined independently by two observers according to the extent of epicardial coronary artery filling via collaterals with contrast medium from the contralateral side before PTCA: 0 = no filling of the distal vessel via collaterals, 1 = small side branches filled, 2 = major side branches of the main epicardial

*Figure 1.* Schematic illustration of the coronary collateral circulation with an inflated angioplasty balloon and a Doppler and pressure guidewire located distal to the stenosis. A coronary flow velocity spectrum obtained distal to the stenosis is shown on the right side (horizontal axis: time, second; vertical axis: average peak velocity [APV], cm/sc, i.e., time average value of instantaneous peak velocity samples over two cardiac cycles). The flow velocity trend depicts a graphic illustration of the concept of the velocity-derived collateral flow index (CFIv, no unit) showing that APV during occlusion amounts to more than half of APV during vessel patency. For the actual calculation of CFIv, ratios of distal velocity time integrals during (Vioccl or PVI, cm) to that after occlusion and following cessation of reactive hyperemia (Viø-occl, cm) are used (see equations above). Abbreviations: CFIv (no unit); pressure-derived collateral flow index; CVP: central venous pressure (mm Hg); Pao: mean aortic pressure (mm Hg); Poccl: intracoronary distal occlusive pressure (mm Hg).
vessel filled, \(3\) = main epicardial vessel filled by collaterals (4,9).

**IC Doppler flow velocity measurements.** Intracoronary Doppler flow velocity measurements were performed using a 0.014 in. (1/3 mm in diameter) PTCA Doppler guidewire with a 12-MHz piezoelectric crystal at its tip (FloWire; EndoSonics, Rancho Cordova, California). The validation of this Doppler guidewire has been described previously (11).

Coronary flow velocity reserve (CFVR) distal to the stenosis was determined by dividing hyperemic peak flow velocity averaged over two cardiac cycles (APV, cm/s) by APV at rest. Hyperemia was induced using an IC bolus of 18 \(\mu\)g adenosine for the left and 12 \(\mu\)g adenosine for the right coronary artery (20).

The velocity-derived index of collateral flow to the balloon-occluded vascular region relative to normal resting flow during vessel patency (CFI, no unit) was determined as the ratio of flow velocity time integral distal to the occluded stenosis (\(V_{i\text{occl}}\), cm) divided by that obtained at an identical location after PTCA (i.e., not occluded, \(V_{i\text{o-occl}}\), cm): 

\[ \text{CFI} = \frac{V_{i\text{occl}}}{V_{i\text{o-occl}}} \]

**IC pressure measurements.** A 0.014 in. fiberoptic pressure monitoring guidewire (Pressureguide; Radi Medical, Uppsala, Sweden) was set at 0, calibrated, advanced through the guiding catheter and positioned distal to the stenosis to be dilated (21,22). The IC pressure-derived CFI (no unit) was determined by simultaneous measurement of mean aortic pressure (\(P_{\text{aor}}, \text{mm Hg}\), obtained from the angioplasty guiding catheter) and the distal coronary artery pressure during balloon occlusion (\(P_{\text{occl}}, \text{mm Hg}\); Figs. 1 and 2). Central venous pressure was estimated to be equal to 5 mm Hg. Pressure-derived collateral flow index was calculated as (\(P_{\text{occl}}-\text{CVP}\)) divided by (\(P_{\text{aor}}-\text{CVP}\)).
(18) (Fig. 1). Pressure-derived collateral flow index expresses collateral flow relative to normal flow through the patent vessel, an index that conceptually corresponds to CFIv.

Intracoronary distal flow velocity and pressure measurements during balloon occlusion and vessel patency after PTCA were performed simultaneously.

**Study protocol.** After diagnostic coronary angiography, an interval of at least 10 min was allowed for dissipation of the effect of the nonionic contrast medium (iopamidol 755 mg/ml) on coronary flow velocity and vasomotion. Several IC boluses of 0.2 mg of nitroglycerin were given to maintain epicardial coronary artery calibers constant, and thus to prevent the influence of changing epicardial vessel diameters on flow indices (CFVR or CFIv) (23). The Doppler and pressure guidewires were positioned distal to the stenosis to be dilated, and CFVR measurements were obtained. The Doppler guidewire was used to transport the PTCA balloon. During the entire protocol, an IC ECG obtained from the Doppler guidewire and a three-lead surface ECG were recorded. After CFVR measurements, distal Viø-occl, Pao, and Poccl were determined simultaneously and repetitively during balloon occlusion (measurements obtained at the beginning of occlusions and averaged). After balloon deflation and cessation of reactive hyperemia, distal nonocclusive Vio-occl, distal IC pressure and Pao were determined simultaneously.

**Statistical analysis.** Between group comparison of continuous data was performed by an unpaired two-sided, student t test. A chi-square test was used for comparison of categorical variables among the two study groups. The diagnostic accuracy of traditional tests to detect sufficient collaterals and that of a specific CFI threshold to distinguish between sufficient and insufficient collaterals were compared, respectively. The CFI threshold was determined as the velocity- and pressure-derived values distinguishing most accurately between the study groups. Accuracies were determined for CFI values between 0.20 and 0.40 (intervals of 0.1). Linear regression analysis and a Bland-Altman (24) analysis were applied to analyze the agreement between IC Doppler- and pressure-derived collateral flow indices. Statistical significance was defined as a p value of <0.05.

**Results**

**Patient characteristics.** There were no statistically significant differences among the two groups with sufficient and insufficient collaterals regarding age of patients, gender, hemodynamic variables during cardiac catheterization, such as heart rate and blood pressure, number of coronary arteries with stenotic lesions, location of the stenotic lesion to be dilated and left ventricular angiographic ejection fraction (Table 1). Approximately one third of the patients had non-Q-wave myocardial infarction (10% in the region of interest) whereby no regional wall motion abnormalities were detected by left ventricular angiography (Table 1). Structural and hemodynamic (CFVR) severities of the stenotic lesion were more pronounced in the group with sufficient collaterals than in the group with insufficient collaterals. Angina pectoris during PTCA occurred less often and the angiographic collateral grade was higher in patients with sufficient collaterals (Table 1).

**IC Doppler flow velocity and pressure data.** Table 2 shows that Viø-occl (cm) and Poccl (mm Hg) obtained distal to the stenosis were significantly higher in patients with sufficient than in those with insufficient collaterals, whereas both nonocclusive Vi (Vio-occl, cm) and Pao (mm Hg) were not different among the groups. This resulted in a velocity- and pressure-derived collateral flow relative to the flow during vessel patency of around 46% in patients with sufficient collaterals, and of approximately 18% in those with insufficient collaterals (Table 2). In about one-fifth of the patients, the Doppler

### Table 1. Clinical, Hemodynamic, Angiographic and Conventional Coronary Collateral Data

<table>
<thead>
<tr>
<th></th>
<th>Sufficient Collaters (no ECG ST-segment changes during occlusion)</th>
<th>Insufficient Collaters (ECG ST-segment changes during occlusion)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>11</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>60 ± 12</td>
<td>57 ± 9</td>
<td>NS</td>
</tr>
<tr>
<td>Male sex (%)</td>
<td>10/11 (91)</td>
<td>26/40 (65)</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>71 ± 12</td>
<td>73 ± 12</td>
<td>NS</td>
</tr>
<tr>
<td>Mean blood pressure (mm Hg)</td>
<td>98 ± 8</td>
<td>101 ± 19</td>
<td>NS</td>
</tr>
<tr>
<td>1-2/vessel CAD (%)</td>
<td>55/45</td>
<td>54/46</td>
<td>NS</td>
</tr>
<tr>
<td>LAD/LCX/RCA stenosis (%)</td>
<td>25/23/62</td>
<td>39/21/40</td>
<td>NS</td>
</tr>
<tr>
<td>Non-Q-wave myocardial infarction (%)</td>
<td>4/11 (36)</td>
<td>13/40 (33)</td>
<td>NS</td>
</tr>
<tr>
<td>LV ejection fraction (%)</td>
<td>67 ± 10</td>
<td>69 ± 12</td>
<td>NS</td>
</tr>
<tr>
<td>Stenosis severity (% diameter stenosis)</td>
<td>92 ± 12</td>
<td>76 ± 15</td>
<td>0.003</td>
</tr>
<tr>
<td>CFVR distal to the stenosis to be dilated</td>
<td>1.45 ± 0.6</td>
<td>1.77 ± 0.7</td>
<td>0.2</td>
</tr>
<tr>
<td>Angina pectoris during stenosis occlusion (%)</td>
<td>3/11 (27)</td>
<td>35/40 (88)</td>
<td>0.0002</td>
</tr>
<tr>
<td>Angiographic collateral grade (0–3)</td>
<td>2.4 ± 0.8</td>
<td>0.8 ± 0.7</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

CAD = coronary artery disease; CFVR = Doppler-derived coronary flow velocity reserve; LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; LV = left ventricular; n = number of patients; NS = not significant; RCA = right coronary artery.
guidewire had to be rotated slightly to obtain an optimal flow velocity signal. The reproducibility of Doppler flow velocity signals obtained during subsequent occlusions was 94%. Thirty-five of the patients were treated by PTCA alone, whereas 19 patients were revascularized using a coronary stent or stents.

**Comparison of traditional and IC tests to assess coronary collaterals.** Table 3 illustrates that a CFIp of >0.30 or 30% (Fig. 3) detected sufficient collaterals most accurately when compared with CFIv > 0.30 (second most accurate test), with an angiographic collateral degree of ≥2, and with the presence of angina pectoris during PTCA (least accurate test). In Fig. 3, the accuracy of velocity- and pressure-derived collateral indices to differentiate between sufficient and insufficient collaterals is also graphically shown; the agreement between an angiographic collateral degree of ≥2 and velocity- and pressure-derived collateral indices >0.30 was much weaker. An angiographic collateral degree ≥2 as cutoff value defining well-developed collaterals was chosen on the basis of data in the literature using such a definition (10). The CFI threshold for velocity- and pressure-derived values was determined as the cutoff value with the highest accuracy for distinguishing between the study groups. Linear regression analysis between CFI, and angiographic collateral degree was as follows: angiographic collateral degree = 3.3 CFI, + 0.3, r = 0.53, p = 0.0001, SEE = 0.82. The correlation among velocity- and pressure-derived collateral flow indices was statistically significant with an underestimation of velocity data using pressure-derived collateral indices (Fig. 4).

**Discussion**

This study of 51 patients revealed that IC flow velocity or pressure measurements distal to a coronary artery stenosis during and after PTCA detect well-developed collaterals more accurately than traditional methods for collateral assessment. Those methods, that is, ECG ST-segment changes during PTCA, chest pain during PTCA and the angiographic collateral degree before PTCA, provide only a qualitative assessment of the collateral circulation, whereas IC Doppler and pressure determinations in this study showed without much discrepancy that approximately one third of blood flow via collaterals to the occluded vascular area relative to the flow through the patent vessel is needed to prevent myocardial ischemia at rest.

**Relevance of coronary collaterals.** The extent of cellular injury and necrosis after coronary artery occlusion depends on the time of occlusion, the size of the regional myocardial area at risk and the collateral circulation (25). How to effectively enhance the coronary collateral circulation has been studied in

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**Table 2. Intracoronary Distal Flow Velocity and Pressure Data During PTCA and the Respective Collateral Flow Indices**

<table>
<thead>
<tr>
<th>n</th>
<th>Sufficient Collaters (no ECG ST-segment changes during occlusion)</th>
<th>Insufficient Collaters (ECG ST-segment changes during occlusion)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(CFIp, no unit) 0.48 ± 0.15</td>
<td>(CFIv, no unit) 0.44 ± 0.16</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>Mean aortic pressure (Pao, mm Hg) 95</td>
<td>CVP = central venous pressure = 5 mm Hg</td>
<td>PTCA = percutaneous transluminal coronary angioplasty.</td>
</tr>
</tbody>
</table>

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**Table 3. Diagnostic Accuracy of Different Tests to Assess the Human Coronary Collateral Circulation**

<table>
<thead>
<tr>
<th>Test for the Detection of Sufficient Coronary Collaterals (no ECG changes during PTCA)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Positive Predictive Accuracy (%)</th>
<th>Negative Predictive Accuracy (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No angina pectoris during PTCA</td>
<td>64</td>
<td>89</td>
<td>50</td>
<td>89</td>
</tr>
<tr>
<td>Angiographic collateral degree ≥2*</td>
<td>82</td>
<td>90</td>
<td>69</td>
<td>95</td>
</tr>
<tr>
<td>Doppler guidewire, CFIp &gt; 0.30</td>
<td>100</td>
<td>93</td>
<td>79</td>
<td>100</td>
</tr>
<tr>
<td>Pressure guidewire, CFIp &gt; 0.30</td>
<td>75</td>
<td>92</td>
<td>75</td>
<td>92</td>
</tr>
</tbody>
</table>

*Before balloon inflation in the stenotic lesion to be dilated. CFIp = intracoronary distal pressure-derived collateral flow index; CFIv = intracoronary distal velocity-derived collateral flow index (>0.30) is equal to a collateral flow of the occluded vascular region relative to normal flow through the patent vessel >30%; CFVR = coronary flow velocity reserve; PTCA = percutaneous transluminal coronary angioplasty.
depth experimentally (26–28), but is not yet known in humans because the standard variable for the assessment of collaterals, volume flow rate, cannot be determined, and because conventional methods to characterize the human coronary collaterals are too blunt to discern subtle effects of a potentially beneficial therapy for the promotion of collaterals. Until very recently when 0.3 mm angioplasty guidewire-based Doppler- and pressure-sensors became available, substitutes for volume flow rate could not even be measured. Volume flow rate is the product of the cross-sectional vascular area through which the flow takes place and the mean velocity of the fluid. Doppler-derived flow velocity can be used as an index for volume flow rate provided that the vascular caliber is kept constant by, for example nitroglycerin. Under these conditions, the ratio between IC flow velocity time integral obtained distal to a coronary stenotic lesion during vessel occlusion to that after vessel occlusion provides relative collateral flow normalized for flow through the patent vessel (17), a parameter directly comparable with the IC pressure-derived collateral flow index introduced and evaluated by Pijls et al. (18,29). The present study simultaneously validated for the first time both of those theoretically favorable parameters for collateral assessment and the methods to obtain them.

Methods to assess the human coronary collateral circulation. Conventional methods for qualitatively assessing human coronary collaterals include postmortem coronary angiography (5), patient’s history of a walking-through angina pectoris (30), nuclear cardiology techniques (31), recording of IC and surface ECG (19) and coronary wedge pressure (32) during PTCA and angiographic grading during vessel patency or occlusion (9). Positron emission tomography has been demonstrated to provide quantitative data of collateral flow to a vascular region of interest (31). However, this can be achieved only in the presence of a naturally occurring coronary artery occlusion of the ipsilateral vessel. The PTCA model for collateral assessment can be used in every patient with a stenotic lesion to be dilated. The presence of ECG ST-segment changes as a sign of myocardial ischemia is regarded to be quite sensitive in studies on collaterals (30). An ECG obtained during PTCA from an IC guidewire with its tip distal to the occluded stenosis can be used even more effectively. For that reason, we chose IC or surface ECG ST-segment changes.

Figure 3. Coronary collaterals assessed by angiography (squares and circles, thick short lines: standard deviation) and intracoronary (IC) ECG (triangles and diamonds) compared with the IC velocity- and pressure-derived collateral flow index (CFI, vertical axis). Well-developed collaterals were defined as those with an angiographic collateral degree ≥2 (0 to 3). Patients with sufficient collaterals were defined as those without ST-segment changes (>1 mm) on IC or surface ECG during balloon occlusion (IC guidewire distal to the stenosis). Velocity- and pressure-derived collateral flow indices do not accurately differentiate between well and poorly developed collaterals, whereas they accurately predict sufficient and insufficient collaterals at a threshold of 0.30 (broken line).

Figure 4. (A) Comparison between simultaneously obtained velocity- and pressure-derived collateral flow indices (CFI_v, vertical axis; CFI_p, horizontal axis). There is a direct, significant correlation between CFI_v and CFI_p. (B) Bland-Altman analysis of the average between CFI_v and CFI_p (horizontal axis) and the difference between CFI_v and CFI_p (vertical axis).
during PTCA to be the primary conventional method for comparison with two other traditional methods (i.e., chest pain during PTCA and coronary angiography) and the IC methods to be tested. Since coronary angiography with contrast media injection from the collateral supplying artery is probably the most widely used semiquantitative method for assessment (grade 0 to 3), it served as a secondary method to be tested with IC parameters (Fig. 3).

The flow velocity-derived index was the most accurate tool for the detection of collateral flow sufficient to prevent myocardial ischemia during PTCA. Surprisingly, IC pressure-derived collateral indices were only slightly more accurate as an angiographic collateral degree (≥2). The problem inherent to a mere detector of well or poorly developed collaterals such as angiography is its inability to provide continuous data on the variable of interest, collateral flow. However, Doppler and pressure wires both measure quantities of relative collateral flow normalized for flow during vessel patency. In this context, it could be demonstrated that it is a collateral flow of 30% or more that prevents myocardial ischemia during PTCA, and that the overlap between such quantitative measures and qualitative angiographic degrees of collateral flow is quite large (Fig. 3). The simultaneously obtained IC velocity and pressure data in this study revealed furthermore that both devices can be used interchangeably to obtain collateral flow indices. The Doppler wire appeared to be more sensitive to measure very low collateral flow indices than the pressure wire (Fig. 4A). However, this interpretation of the data may be incorrect and the seemingly better detectability of flow signals using IC Doppler versus pressure sensors may be related to the recording of wall motion artifacts by the former.

Previous investigations using Doppler guidewires for collateral assessment compared absolute flow velocities during occlusion with the traditional angiographic standard (15,16,33,34). However, it is not conceptually feasible to quantify collateral flow by absolute IC occlusive flow velocities because they are co-determined by the measurement site within the coronary artery tree (35,36). Therefore, the present study computed a ratio of occlusive to patent flow velocities to circumvent the problem of the measurement site. This relative collateral flow is furthermore directly comparable with the IC parameters (Fig. 3).

**Study limitations.** One major source of error using the Doppler guidewire to calculate collateral flow indices is that the wire position may vary between the measurement of the occlusive flow velocity and the measurement during vessel patency. Because the situation present in every branching tree structure of the total cross-sectional area of the branches increasing toward the periphery, the velocity of the fluid transported in such a tree must decrease markedly at every branch point by a factor of 1.25 (36). We cannot entirely exclude that the location of the Doppler wire was altered in some of the cases to obtain good flow velocity signals. Varying heart rate and aortic pressure during IC Doppler measurements cause flow velocity changes. There was no significant alteration of those variables during the procedure. Apart from flow due to collateral flow, slow velocity signals recorded during coronary occlusion may represent wall motion artifacts, and fast velocity signals may be due to squeezing of blood into the recipient coronary artery during myocardial contraction. Both phenomena can potentially lead to an overestimation of the CFI, but they are both easily recognizable by their appearance as background noise (wall motion artifacts), and by their high-pitch, high-velocity, short-duration character. The fact that the denominator in the Doppler-derived CFI is the velocity integral during vessel patency after PTCA carries the risk of underestimating CFI due to still prevailing postocclusion hyperemia. This could explain the consistently lower CFI value for a given CFIp value >0.20 (Fig. 4). Theoretically and conversely, a residual stenosis after PTCA could lead to a still decreased flow velocity in the denominator, resulting in an underestimation of CFI. Because the distal flow velocity during vessel patency is mostly obtained at resting conditions, the residual stenosis would have to be >80% in diameter to be hemodynamically relevant. In the case of pressure-derived collateral flow indices, the mentioned variables of altered guidewire-location, changes of heart rate and aortic pressure do not influence the study results because the CFI is calculated under consideration of the occlusive and aortic pressure. However, the assumption of a CVP instead of a central venous measurement may introduce a substantial error because of its value, which may be relatively close to that of the occlusive pressure.

**Conclusion.** Intracoronary flow velocity or pressure measurements during routine PTCA represent an accurate and, at last, quantitative method for assessing the coronary collateral circulation in humans.

**References**

Intracoronary Pressure and Flow Measurement for Assessing Functional Coronary Artery Stenosis