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Coronary Vasomotion After Percutaneous Transluminal Coronary Angioplasty Depends on the Severity of the Culprit Lesion

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Objectives. **This study sought to evaluate coronary vasomotor response to percutaneous transluminal coronary angioplasty (PTCA) and its influence on proximal and distal vessel diameters with regard to stenosis severity and coronary blood flow.**

Background. **Coronary vasoconstriction of the distal vessel segment has been reported after PTCA. This vasoconstrictive effect was thought to be due to balloon-induced injury of the vessel wall, with release of local vasoconstrictors or stimulation of the sympathetic system with release of catecholamines, or both.**

Methods. **Thirty-nine patients were prospectively studied before and after PTCA. Patients were classified into two groups according to the severity of the culprit lesion: *group 1* = ≥70% to ≤85% diameter stenosis (n = 23); and *group 2* = >85% to ≤95% diameter stenosis (n = 16). The coronary vessel diameter of the proximal and distal vessel segments as well as the minimal lumen diameter were determined by quantitative coronary angiography. In a subgroup of 16 patients, basal and maximal coronary flow velocity was measured before and after PTCA with the Doppler FloWire system.**

Results. **The groups were comparable with regard to age, gender, serum cholesterol levels and medical therapy. The proximal vessel segment remained unchanged after PTCA in group 1 ([mean ± SD] 0.9 ± 3.5%, p = 0.8) but showed vasodilation in group 2 (+13.7 ± 3.6%, p < 0.05). However, the distal segment showed vasoconstriction in group 1 (−6.7 ± 2.0%, p < 0.01) and vasodilation in group 2 (+31 ± 8.0%, p < 0.01). A significant correlation was found between the change in distal vessel diameter after PTCA and stenosis severity (r = 0.61, p < 0.0001). Changes in blood flow were directly correlated to stenosis severity (r = 0.85, p < 0.002); that is, rest flow increased after PTCA in narrow lesions but remained unchanged in moderate lesions. The diameter changes in the distal vessel segment after PTCA were significantly related to flow changes (r = 0.90, p < 0.0001). Coronary distending pressure of the distal vessel segment increased significantly in both groups; however, this increase was significantly greater in group 2 than in group 1 (55 ± 4 vs. 14 ± 3 mm Hg, p < 0.0001).**

Conclusions. **Coronary vasomotion of the proximal and distal vessel segments after PTCA depends on the severity of the culprit lesion; that is, vasoconstriction of the distal segment is found in patients with moderate lesions and vasodilation in those with severe lesions. Thus, vasomotion of the post-stenotic vessel segment depends on the severity of the culprit lesion and is influenced by changes in coronary flow or distending pressure, or both.**

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Coronary vasoconstriction of the distal vessel segment has been reported after successful percutaneous transluminal coronary angioplasty (PTCA) (1–6) and has been attributed to the release of local vasoconstricting factors, such as endothelin (7), serotonin (8) and thromboxane (9,10), which are released from the endothelium or platelets, or both. Fischell et al. (2) reported stretch-induced vasoconstriction of the distal vessel segment after balloon angioplasty that was independent of platelet aggregation but was probably mediated through endothelium-derived cyclooxygenase products. Neural mechanisms are also likely to play a vasoconstrictive role, especially in the absence of a normal endothelium (6). Local and systemic vasoconstricting factors may induce coronary vasoconstriction after PTCA that can be blocked by either alpha-adrenergic blocking agents (6), ketanserin (8,11), endothelin antagonists (12) or nitric oxide (endothelium-derived relaxing factor [EDRF]) (13,14). However, coronary flow may also influence coronary vasomotion of the distal vessel segment because an increase in flow after removal of the flow-limiting stenosis may lead to coronary vasodilation through release of relaxing factors from the intact coronary endothelium (15,16). In the presence of endothelial dysfunction, the increase of distending pressure early after PTCA may induce coronary vasodilation by passive distension of the distal vessel segment. Thus, the present study sought to evaluate the influence of stenosis severity on coronary vasomotion of the proximal and distal vessel segments before and after PTCA.

Methods

Patients. Thirty-nine patients with coronary artery disease were prospectively studied and classified into two groups according to the severity of the culprit lesion: *Group 1* included

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Abbreviations and Acronyms

PTCA = percutaneous transluminal coronary angioplasty
EDRF = endothelium-derived relaxing factor

23 patients with ≥70% to ≤85% diameter stenosis, and *group 2* included 16 patients with >85% to ≤95% diameter stenosis. All patients underwent coronary angiography for diagnostic purposes. Medication was stopped at least 24 h before cardiac catheterization. Biplane left ventricular angiography was performed before diagnostic coronary arteriography. PTCA was carried out according to the Judkins technique using 7F guiding catheters. No nitroglycerin was given during the study. Only a minority of patients received nitroglycerin at the completion of the examination, but these data were not quantitatively analyzed. Collateral grading (from 0 to 3) was visually assessed according to the technique of Rentrop et al. (17): *grade 0* = no visible collateral vessels; *grade 1* = filling of side branches of the artery; *grade 2* = partial filling of the epicardial segment; *grade 3* = complete filling of the vessel.

Patients with total occluded vessels or with mild lesions (<70% diameter stenosis) as well as those with a clearly visible dissection after PTCA or with stent implantation were excluded from the study. Patients with acute myocardial ischemia or infarction were also excluded from the analysis.

Quantitative coronary angiography. Selective arteriography of the culprit lesion was performed in multiple projections using nonionic contrast material. The precise angles of the optimal angiographic projection were recorded, and this projection was used for all subsequent coronary arteriograms. Cinefilm was used as a data carrier (filming rate 25 or 50 frames/s). Quantitative evaluation was performed with a semiautomatic computer system (18). The system is based on a 35-mm film projector (Tagarno A/S, Horsens, Denmark), a slow-scan charged-couple device camera (for image digitalization) developed at the Institute for Biomedical Engineering in Zurich and a computer workstation (Apollo DN 3000, Wangen, Switzerland) for image storage and processing. For calibration purposes the exact diameter of the catheter was used. Contour detection was carried out using a geometric densitometric edge detection algorithm. The methodology for computerized analysis of coronary arteriograms has been described elsewhere (18). Measurements were carried out before and 10 min after PTCA. Each segment was analyzed during late or end-diastole, and measurements from 2 to 4 consecutive beats were averaged. Quantitative evaluation was not blinded, but because the analysis was done automatically, it was not possible to influence the outcome of the data. The *proximal segment* was defined as a clearly identifiable smooth vessel segment proximal to the dilation site (n = 39). The *culprit lesion* was defined as the minimal lumen diameter between the proximal and distal vessel segment that was considered responsible for the clinical symptoms (n = 39). The *distal segment* was defined as a clearly identifiable smooth vessel segment distal to the dilation site (n = 39). The *control segment* (n = 26) was defined as a clearly visible smooth segment of the left coronary artery that was not reached by the guide wire or balloon catheter (ideally located in the left circumflex coronary artery when PTCA of the left anterior descending coronary artery was performed or vice versa). No control segment was obtained in patients with PTCA of the right coronary artery (n = 13). *Vasoconstriction* or *vasodilation* was defined as the percent change in vessel diameter with regard to the baseline value: Percent diameter change = {(Post-PTCA diameter − Pre-PTCA diameter)/Pre-PTCA diameter} × 100%.

Coronary flow measurements and calculation of coronary distending pressure. Flow velocity was measured in 16 patients using the Doppler FloWire system. The wire is a flexible 0.014-in (0.036 cm) angioplasty guide wire with a 12-MHz piezoelectric ultrasound transducer at its tip (Cardiometrics). Velocity data were processed on-line by a fast Fourier transformation with a real-time scrolling gray-scale spectral display. Velocities were recorded on videotape, and single images were printed for off-line analysis. Flow velocity measurements obtained with this system were validated in vitro and in an animal model using simultaneous electromagnetic flow measurements for comparison (19–21).

Basal and maximal coronary flow velocities distal to the stenosis were measured continuously with the Doppler guide wire before and after PTCA. Hyperemia was induced by intracoronary bolus injection of adenosine: 12 μg of adenosine for the right coronary artery and 18 μg of adenosine for the left coronary artery. Estimates of basal and maximal coronary flow were derived from the product of cross-sectional area and mean velocity at baseline and at peak response, respectively, and were expressed in ml/min. Mean velocity was estimated as 50% of the time-averaged peak velocity (APV) assuming a fully developed velocity profile. Coronary blood flow (CBF) was calculated from the corresponding peak blood flow velocity and vessel diameter (D) as $CBF = (D^2\pi/4) \times (APV \times 0.5) \times 0.6$, where 0.5 is a correction coefficient for parabolic flow, and 0.6 is a conversion factor from $mm^2 \times cm/s$ to ml/min (22). The vessel diameter at the tip of the guide wire was measured by quantitative coronary angiography.

The *distending pressure* was calculated from mean pressure gradient across the stenosis and mean aortic pressure. The *mean pressure gradient* was calculated from stenosis severity assuming constant flow (23).

Statistical analysis. Results are expressed as mean value ± SEM and clinical data as mean value ± SD. Comparisons between the two study groups were performed using an unpaired Student *t* test. Comparisons between coronary dimensions and flow before and after PTCA as well as under adenosine infusion were carried out by analysis of variance for repeated measures. If the analysis was significant, the Scheffé procedure was applied. The chi-square test was used for event rates and other discrete factors. Regression analysis was performed to evaluate relations between coronary flow, dimensional changes and stenosis severity; $p < 0.05$ was considered significant.

Table 1. Patient Characteristics

	Group 1 (n = 23)	Group 2 (n = 16)	p Value
Age (yr)	54.7 ± 8.4	58.1 ± 8.6	0.219
Men/women	23/0	15/1	0.224
Patient history			
Stable angina	11 (48%)	7 (44%)	0.802
Previous MI	12 (52%)	9 (56%)	0.802
NYHA functional class			
II	13 (57%)	7 (44%)	0.432
III	10 (43%)	9 (56%)	0.432
Laboratory findings			
Cholesterol (mmol/liter)	4.63 ± 2.5	4.31 ± 2.5	0.723
Triglycerides (mmol/liter)	2.52 ± 1.4	2.64 ± 2.5	0.874
Anti-ischemic therapy			
Beta-blocker	18 (78%)	14 (87%)	0.452
Ca antagonist	15 (65%)	11 (68%)	0.818
Nitrates	12 (52%)	10 (62%)	0.522

Data presented are mean value ± SD or number (%) of patients. Ca = calcium; MI = myocardial infarction; NYHA = New York Heart Association.

Results

Patient characteristics. Age, gender, medical treatment, lipid profile, other coronary risk factors, New York Heart Association functional class and hemodynamic data were equally distributed within the two study groups (Tables 1 and 2). Before PTCA, all patients had positive results on exercise testing. The maximal achieved work load was higher (155 ± 47 vs. 115 ± 29 W, $p < 0.01$) and the magnitude of ST segment depression lower in group 1 than group 2 (0.1 ± 0.1 vs. 0.2 ± 0.1 mV, respectively, $p < 0.02$). After PTCA no significant difference was found between maximal work load (164 ± 35 vs. 144 ± 35 W, $p = 0.096$) and ST segment depression (0.07 ± 0.08 vs. 0.1 ± 0.1 mV, $p = 0.52$) in group 1 versus group 2.

Angiographic data. Before PTCA the proximal vessel diameter was similar in groups 1 and 2 (3.0 ± 0.11 vs. 2.7 ± 0.14 mm, respectively, $p = 0.08$), whereas the distal vessel diameter was slightly larger in group 1 than group 2 (2.5 ± 0.1 vs. 2.1 ± 0.14 mm, $p < 0.02$). Minimal lumen diameter was significantly larger (0.67 ± 0.04 vs. 0.26 ± 0.02 mm, $p < 0.001$) and percent diameter stenosis significantly smaller (78 ± 5% vs. 90 ± 3%, $p < 0.001$) in group 1 than group 2. Collateralization according to Rentrop et al. (17) was similar in both groups (group 1: 0.4 ± 0.15 vs. group 2: 0.8 ± 0.28, $p = 0.119$).

After PTCA, the minimal lumen diameter (2.0 ± 0.1 vs. 2.0 ± 0.08 mm, $p = 0.9$) as well as the proximal (2.96 ± 0.11 vs. 2.96 ± 0.1 mm, $p = 0.97$) and distal (2.33 ± 0.10 vs. 2.65 ± 0.16 mm, $p = 0.072$) vessel diameters were comparable in the two groups. Accordingly, the percent diameter stenosis decreased from 78% to 26% ($p < 0.0001$) in group 1 and from 90% to 28% ($p < 0.0001$) in group 2.

Coronary artery response to PTCA. There was no significant change in proximal and distal vessel diameters before and after PTCA (Fig. 1). Minimal lumen vessel diameter increased significantly, from 0.5 ± 0.04 to 2.0 ± 0.06 mm ($p < 0.001$) after PTCA. The proximal vessel segment showed no change in group 1 (+0.9 ± 3.5%, $p = 0.8$) before and after PTCA, but there was slight vasodilation (+13.7 ± 3.6%, $p < 0.001$) in group 2 (Fig. 2). In contrast, the distal vessel segment elicited coronary vasoconstriction in group 1 (−6.7 ± 2.05%, $p < 0.005$) but vasodilation in group 2 (+31 ± 8.02%, $p < 0.002$). The control segment showed no significant change in the two groups after PTCA (Fig. 2).

Influence of stenosis severity on vasomotion of proximal and distal vessel segments and coronary blood flow after PTCA. Significant correlation (Fig. 3) was found between coronary vasomotion of the proximal vessel and severity of the culprit lesion ($r = 0.455$, $p < 0.01$) as well as between vasomotion of the distal vessel and severity of the culprit lesion ($r = 0.61$, $p < 0.0001$) (Fig. 3).

In patients (n = 10) with moderate lesions (Fig. 4), basal blood flow remained unchanged after PTCA (21.9 ± 4.1 ml/min before PTCA vs. 27.4 ± 8.0 ml/min after PTCA, $p = 0.314$), whereas in patients (n = 6) with severe lesions, basal flow increased significantly, from 8.4 ± 2.3 to 27.9 ± 5.8 ml/min ($p < 0.05$) after PTCA. Maximal blood flow increased significantly in group 1 (44.9 ± 7.6 ml/min before PTCA vs. 99.0 ± 18.3 ml/min after PTCA; $p < 0.02$) and group 2 (17.8 ± 3.1 ml/min before PTCA vs. 80.4 ± 17.6 ml/min after PTCA; $p < 0.02$). Significant correlation was observed between the change in basal flow after PTCA and stenosis severity ($r = 0.85$, $p < 0.001$). No significant change in basal flow occurred after PTCA in patients with mild lesions, but a significant increase was found in those with severe lesions (Fig. 5).

There was a high linear correlation between the change in distal vessel diameter and the increase in coronary blood flow after PTCA ($r = 0.90$, $p < 0.0001$). Other correlations are reported in Table 3.

Table 2. Angiographic and Hemodynamic Data

	Group 1 (n = 23)	Group 2 (n = 16)	p Value
CAD			
1 VD	10 (43%)	7 (44%)	0.987
2 VD	11 (48%)	7 (44%)	0.802
3 VD	2 (9%)	2 (12%)	0.700
Vessel treated			
LAD	13 (56%)	6 (38%)	0.242
LCx	2 (9%)	5 (31%)	0.071
RCA	8 (35%)	5 (31%)	0.818
Collateral grading	0.35 ± 0.7	0.8 ± 1.1	0.119
Mean aortic pressure (mm Hg)			
Before PTCA	85 ± 14.5	90 ± 9.6	0.205
After PTCA	86 ± 13.8	88 ± 8.4	0.566
Heart rate (beats/min)			
Before PTCA	64 ± 8.2	60 ± 9.5	0.200
After PTCA	70 ± 12.2	63 ± 11.6	0.079

Data presented are mean value ± SD or number (%) of patients. LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; PTCA = percutaneous transluminal coronary angioplasty; RCA = right coronary artery.

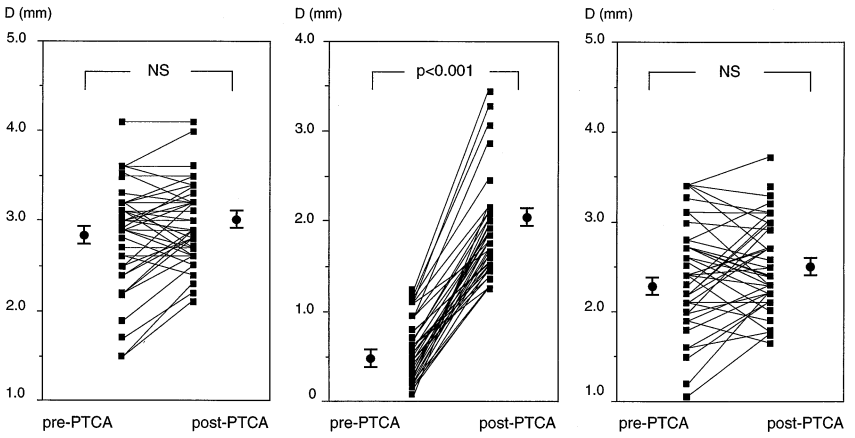


Figure 1. Individual coronary artery diameters (D) of the proximal **(left panel)** and distal **(right panel)** vessel segments as well as minimal lumen diameters **(middle panel)** before and after PTCA in all 39 patients. The minimal lumen diameters show a significant increase after PTCA, but the proximal and distal vessel diameters do not change. However, paired data for the proximal and distal vessel diameters show that there are two subgroups of patients with regard to dimensional changes after PTCA, some with vasoconstriction and some with vasodilation. **Circles and error bars** = mean value ± SEM.

Increase in coronary distending pressure after PTCA and diameter changes distal to dilation site. Coronary distending pressure increased after PTCA, from 43 ± 2 to 58 ± 5 mm Hg ($p < 0.001$) in group 1 and from 11 ± 4 to 66 ± 4 mm Hg ($p < 0.0001$) in group 2 (Fig. 6). The increase in distending pressure was significantly higher in group 2 than group 1 (55 ± 4 vs. 14 ± 3 mm Hg, $p < 0.0001$). There was a significant correlation between the change in distal vessel diameter and the increase in distending pressure after PTCA ($r = 0.57$, $p < 0.001$).

Discussion

PTCA is being increasingly utilized as a therapeutic tool for patients with ischemic heart disease. However, local vasoconstriction and severe vasospasm of the stenotic and post-stenotic vessel segments are frequently observed after PTCA (1,3–6,24). The *major finding of the present study* is that the early vasomotor response to PTCA distal to the dilation site differs with regard to stenosis severity; that is, coronary vasoconstriction occurs in moderate lesions, but vasodilation in narrow lesions. The cutoff point for coronary vasoconstriction or vasodilation seems to be close to a diameter stenosis of 85%. The exact pathophysiologic mechanism of this phenomenon is not clear but is thought to be due to an imbalance between flow-mediated vasodilating and local vasoconstricting factors that are essential for the regulation of coronary vascular tone (24). This imbalance favored vasoconstrictors in moderate lesions where blood flow did not change after PTCA and favored vasodilators in tight lesions where marked flow increase was observed. However, passive changes might influence the behavior of the distal vessel as well because an increase in distending pressure early after PTCA might overrule vasoconstrictor influences when flow-mediated vasodilation is lacking in the presence of endothelial dysfunction, which is common in patients with coronary artery disease.

Vasoconstrictor effects. Although coronary vasoconstriction has been previously reported after successful angioplasty, the individual factors are not yet fully understood. Fischell et al. (1,4) observed coronary vasoconstriction of the distal vessel segment after PTCA that started shortly after intervention and reached its maximum 30 min thereafter. Distal vessel vasoconstriction was directly related to stenosis severity before PTCA (4) and was explained by pressure/stretch-induced myogenic activation (2,4). Increased vasoconstrictor effects 30 min after successful revascularization of chronic occlusions were reported by Leung (5) who found a significant correlation between the degree of distal vasoconstriction after PTCA and the increase in distal coronary perfusion pressure immediately

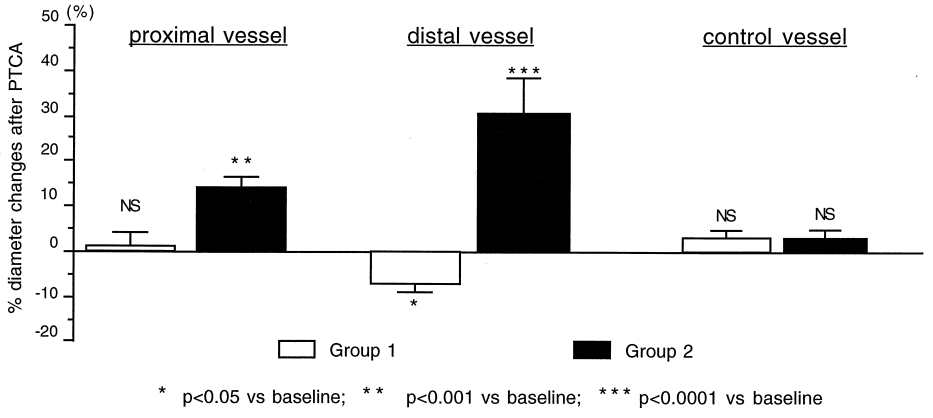


Figure 2. Bar graphs representing changes in coronary diameters of the proximal, distal and control vessel segments from baseline to after PTCA. In group 1, there is significant vasoconstriction of the distal vessel segment, whereas in group 2, both proximal and distal segments show significant vasodilation. The control vessel remains unchanged in the two groups.

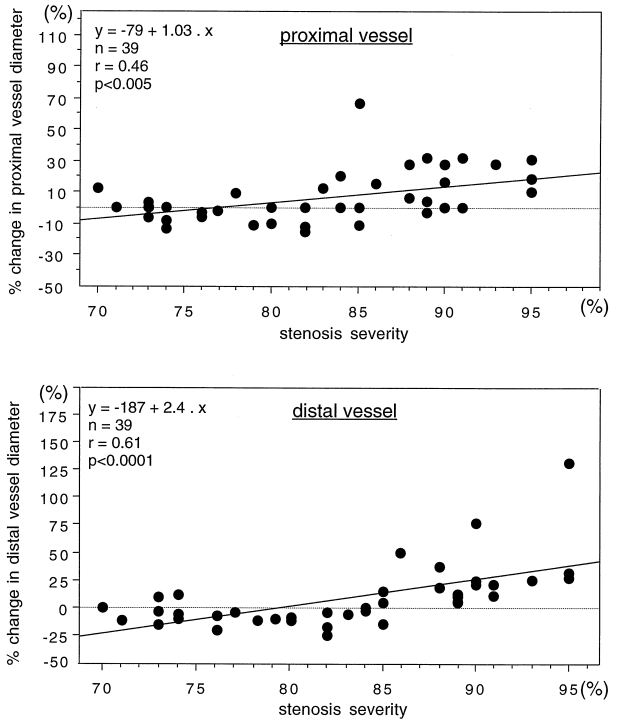


Figure 3. Correlation between stenosis severity and percent diameter change of the proximal and distal segments. Patients with narrow lesions (>85% diameter stenosis) demonstrate vasodilation, whereas those with moderate lesions show vasoconstriction.

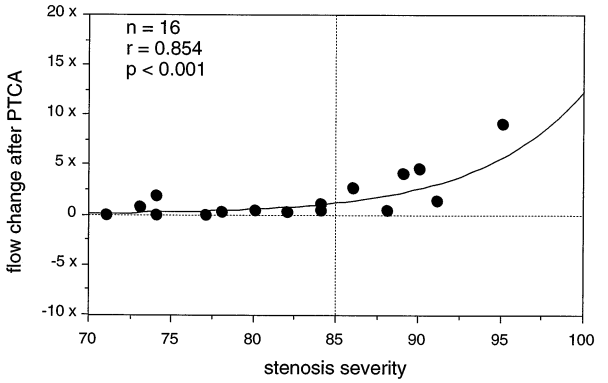


Figure 5. Correlation between diameter stenosis of the culprit lesion and percent change in basal coronary flow after PTCA. A flow increase after PTCA can be seen in narrow lesions (>85% diameter stenosis), whereas no change in basal coronary flow occurs in moderate lesions.

after the last balloon inflation. Hypersensitivity of smooth muscle cells to vasoconstrictor agents in atherosclerotic arteries was also suggested as a possible cause of coronary vasoconstriction (25). Experimental studies (26–29) have provided further support for increased vasoconstrictor activity of coronary arteries with endothelial damage, but there is evidence that other factors might also be involved in this process, for instance, reduction of EDRF release (30), alteration in vessel wall arachidonate metabolism (31), dysfunction of the adrenergic system (32) and increased levels of endothelin (7), serotonin (8) and thromboxane (9,10). In this regard, administration of an alpha-blocker (6) or serotonin- (8,11) or endothelin-receptor antagonist (12,33) prevented vasoconstriction after PTCA, suggesting that humoral factors might play an important role in the pathophysiology of coronary vasoconstriction after PTCA.

Vasodilator effects. Large coronary arteries are responsive to changes in flow and pressure. A flow-dependent increase in vessel diameter of smooth coronary arteries has been reported in humans; however, this vasodilation was attenuated or even lost in atherosclerotic segments (34,35). Flow-dependent coronary vasodilation was preserved in angiographically smooth arteries in patients with hypercholesteremia as well as in those with CAD but was abolished in vessels with lumen irregularities (36). A low basal and stimulated release of nitric oxide can explain the diminished endothelium-dependent (flow-dependent) vasodilation in atherosclerotic coronary arteries (30). Therefore, vasoconstrictor influences after PTCA may not be compensated by a flow increase with an enhanced release of EDRF.

Two types of reaction of the coronary arteries can be expected after PTCA, depending on the functional status of the endocardium. In the presence of an intact endothelium,

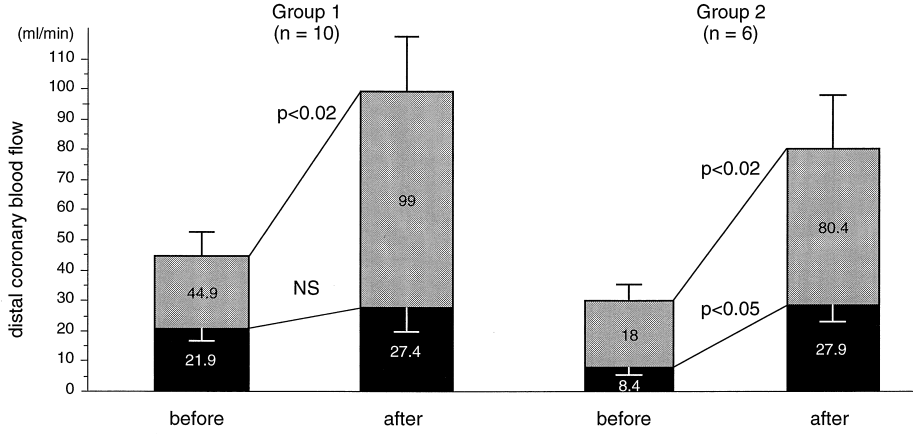


Figure 4. Absolute changes in coronary blood flow in patients with moderate (group 1) and severe lesions (group 2). Basal (**solid bars**) and maximal flow (**gray bars**) are shown before and after PTCA. In patients with moderate lesions, basal flow remains unchanged, and maximal flow as well as coronary flow reserve increase significantly after PTCA, whereas in patients with tight lesions, both basal and maximal flow increase significantly after the intervention. **Column** = mean value ± SEM (**vertical bars**).

Table 3. Correlations

	No. of Pts	r Coeff	p Value
Stenosis severity (% DS) versus			
Change in vessel diam			
Distal vessel seg	39	0.61	<0.0001
Proximal vessel seg	39	0.46	<0.01
Control vessel seg	21	0.15	0.811
Change in basal CBF	16	0.85	<0.001
Change in MLD	39	0.89	<0.001
Changes in MLD versus			
Change in vessel diam			
Distal vessel seg	39	0.75	<0.0001
Proximal vessel seg	39	0.50	<0.002
Control vessel seg	21	0.02	0.998
Change in basal CBF	16	0.88	<0.005
Changes in distal CBF versus change in distal vessel diam	16	0.90	<0.0001
Changes in distending pressure versus change in distal vessel diam	39	0.56	<0.001

CBF = coronary blood flow; Coeff = coefficient; diam = diameter; DS = diameter stenosis; MLD = minimal lumen diameter; Pts = patients; seg = segment.

coronary vasodilation can occur in response to the flow change. In the presence of endothelial dysfunction, an abrupt increase in flow after dilation of severe lesions can evoke passive pressure/distension effects on the distal lumen. These different types of reaction might determine the behavior of the vessel after PTCA, that is, coronary vasodilation when the endothelium is intact or vasoconstriction when the endothelium is damaged.

Clinical implications. Coronary vasomotion plays an important role in the outcome of PTCA. Vasoconstriction may precipitate early occlusion because the occurrence of coronary artery spasm of the dilated vessel segment has been reported to be frequently accompanied by restenosis (37). Thus, vasodilation after PTCA may have a beneficial effect on long-term outcome because coronary blood flow may enhance coronary healing. In contrast, vasoconstriction carries the risk of acute vessel occlusion or the development of restenosis. Thus, the main determinant of coronary vasomotion after PTCA is flow, but passive pressure/distension effects might become significant when endothelial control of coronary vasomotion is reduced. As a consequence, in the presence of endothelial dysfunction, coronary distending pressure may be important and may nullify the vasoconstrictor effects immediately after PTCA. However, which mechanism plays a more important role in the observed vasodilation immediately after PTCA cannot be determined because changes in flow and passive pressure/distension are in the same direction.

Study limitations. There are several limitations to the present study that need to be addressed:

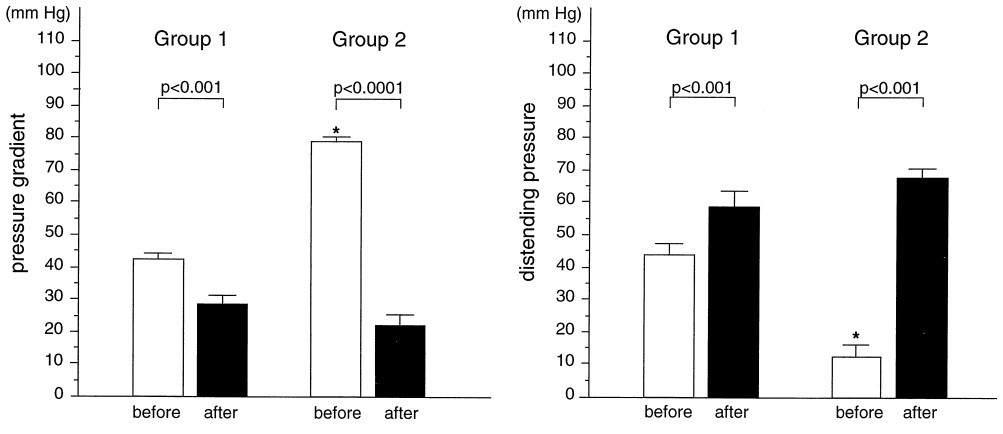
1. Previous investigators (1,2,4,5) reported coronary vasoconstriction 15 to 30 min after PTCA. However, our measurements were carried out ~10 min after PTCA; thus, vasoconstrictor effects may not have been fully active. There are a few reports (11) of coronary vasoconstriction as early as 5 min after PTCA, such as in our group with moderate lesions. The delayed response to PTCA has been attributed to several factors but is most likely due to increased endothelin levels, which have been reported (38) to become fully active after an interval of >30 min.

2. Intravascular ultrasound was not performed in the present study. However, all patients with angiographically visible dissections after PTCA were excluded from the analysis. Determination of vasoactive substances may have provided additional information on the pathophysiologic mechanisms involved in the behavior of the distal vessel segment after PTCA; however, these substances were not investigated.

3. Intracoronary blood flow velocity was measured with the FloWire technique, which requires optimal placement of the transducer parallel to the blood flow. In tortuous vessels, some manipulation may be required to achieve the proper positioning of the flow probe, but only patients with good flow signals were included in the present analysis.

4. Intracoronary pressure measurements were not per-

Figure 6. Calculated pressure gradient **(left panel)** and distending pressure **(right panel)** before and after PTCA in both study groups. The pressure gradient decreases significantly, and the distending pressure increases in the two groups; however, these changes are more pronounced in group 2 with narrow lesions. **Columns** = mean value ± SEM **(vertical bars)**. *p < 0.0001, group 1 versus group 2.



formed; thus, no definite statement on distal coronary distending pressure can be made.

Conclusions. The coronary vasomotor response of the distal vessel segment after PTCA differs with regard to stenosis severity. After PTCA, vasoconstriction of the distal segment can be observed in patients with moderate lesions and vasodilation in those with severe lesions. The mechanism of this opposite reaction in patients with moderate and severe lesions is not clear but can be explained either by flow changes in the presence of an intact endothelium or by changes in passive distending pressure in the presence of endothelial dysfunction, or both. Thus, vasomotion of the post-stenotic vessel segment depends on the severity of the culprit lesion and appears to be influenced by changes in flow or passive pressure/distension effects, or both.

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