The transstenotic pressure gradient trend as a predictor of acute complications after percutaneous transluminal coronary angioplasty

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ABSTRACT The transstenotic pressure gradient recorded during coronary angioplasty (PTCA) reflects the dynamic relationship that exists between coronary blood flow and the effective cross-sectional area of the arterial lumen. An apparent relationship between the dynamic behavior of the pressure gradient and subsequent acute vessel closure was observed in our catheterization laboratory. We therefore examined the usefulness of the pressure gradient trend in predicting acute complications after 463 attempted PTCA procedures. Two pressure gradient trend patterns were identified: (1) a rising trend pattern identified by an increasing pressure gradient in the interval after deflation of the angioplasty, and (2) a stable trend pattern identified by a constant or decreasing pressure gradient. The incidence of acute vessel closure (17% vs 4%, $p = .0001$), emergency CABG (5.6% versus 1%, $p < .05$), and myocardial infarction (13% versus 2%, $p < .0001$) after the PTCA procedure was significantly higher among patients with rising trend patterns when compared with patients with stable trend patterns. Multivariate analysis identified independent predictors for an acute closure event as a rising trend pattern ($p < .001$), post-PTCA gradient ($p < .05$), and post-PTCA percent diameter stenosis ($p < .02$). Independent predictors for emergency coronary artery bypass grafting and myocardial infarction were post-PTCA gradient ($p < .001$) and a rising trend pattern (odds ratio = 2.91, $p < .001$), respectively. The dynamic behavior of the gradient trend provides additional useful information about the results of dilatation. This information is available before completion of dilatation and may assist in clinical decision making by identifying those patients who may be at risk for developing acute complications in the post-PTCA period.
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PERCUTANEOUS transluminal coronary angioplasty (PTCA)[1–3] has evolved into a widely accepted treatment modality for certain patients with coronary artery disease. Acute vessel closure remains a significant problem and is the principle factor underlying the major complications of emergency bypass graft surgery (CABG), myocardial infarction, and death.[4] Immediately after deflation of the angioplasty balloon and for the next 48 hr, intimal dissection, thrombosis, arterial spasm, or a combination of these events may lead to acute closure of the dilated artery. The availability of devices with potential for preventing and treating acute closure has increased interest in predicting this outcome.[5, 6]

The transstenotic pressure gradient recorded after deflation of the angioplasty balloon reflects the dynamic relationship between coronary arterial blood flow and effective cross-sectional luminal area. The absolute gradient measurement is influenced by other factors, including the cross-sectional area of the balloon catheter, presence of collaterals, and status of the distal vessel.[7] However, in the presence of constant coronary blood flow, an alteration in the effective lumen is manifested by a change in the measured transstenotic pressure gradient.[8] During PTCA, the lumen of the artery undergoing dilatation increases and this is usually associated with a rapid decline in the transstenotic pressure gradient.[7] If dilatation causes intimal disruption with a subsequent decrease in the effective lumen, the resulting transstenotic pressure gradient will increase over time.

We observed an apparent relationship between a rising pressure gradient trend after balloon deflation and subsequent acute vessel closure occurring either in the laboratory or shortly thereafter. This study was

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designed to determine if the pressure gradient trend would be useful in identifying those patients at risk of developing acute complications after PTCA.

Methods

Patient selection. Data were collected in two phases. In the initial phase between June and October 1984, 113 patients undergoing PTCA procedures were randomly selected for prospective study. Because acute complications in this group were too few for analysis, an additional 350 consecutive patients were studied between April and June 1985. Of the total 463 procedures, inadequate pressure gradient data were obtained in 83 (18%). In 21 of these 83 patients, a transstenotic pressure gradient was not recorded because the coronary artery segment could not be crossed with the angioplasty balloon catheter. In the remaining 62, pressure gradient recordings were unavailable because the data were either unclassifiable by the authors (n = 10) or too brief for analysis (n = 5); the remainder went unrecorded (n = 47) because the clinical situation or other technical factors did not permit. Thus, 380 patients undergoing PTCA had pressure gradient trends suitable for analysis.

Study design. The transstenotic pressure gradient information collected during phase I (June to November, 1984) was obtained prospectively by one of the investigators (D.R.) when available to continuously observe a procedure. A comprehensive record of each procedure was made that included balloon inflations, intracoronary contrast injections, intracoronary nitroglycerin administration, and balloon "crossings" and "pullbacks." The transstenotic pressure gradient trend was then analyzed with the use of this intervention log, thus allowing separation of artifact from pressure gradient trend information during data analysis. The data collected during phase II (April to June 1985) were obtained retrospectively from a review of all pressure gradient recordings during this period. Analysis of this transstenotic pressure gradient information was aided by a similar, but less comprehensive, intervention log recorded by a catheterization laboratory technician.

Recording and classification of gradient trends. Classification of the transstenotic pressure gradient data was done by consensus among three experienced angioplasty operators who were blinded to the angiographic and clinical outcome of each case. A computerized physiologic monitoring system (Meddars Series 300, Honeywell, Inc.) was used to record the arterial pressures measure through both the guiding and balloon catheters.

A schematic diagram of the system used to compute the transstenotic pressure gradient is shown in figure 1. With the guidewire and balloon catheter positioned across the coronary artery stenosis, a pressure port distal to the balloon segment monitors the arterial pressure distal to the stenosis. The proximal arterial pressure is recorded through the guiding catheter tip, which is positioned at the coronary ostium. These phasic pressures are electronically integrated over a 1 sec sample interval. The mean distal arterial pressure is then subtracted from the mean proximal pressure, which yields the transstenotic pressure gradient. This record is updated each second throughout the PTCA procedure. A computer program that generates the transstenotic pressure gradient is activated after the balloon catheter is advanced across the coronary artery stenosis and the proximal and distal pressure recordings have stabilized. The program records a maximum of 500 sec of information before it is transferred to hard copy and restarted. This plot of the transstenotic pressure gradient versus time is available with the pressure gradient (mm Hg) displayed along the ordinate and time (seconds) along the abscissa. The information is displayed in real-time on a video monitor for the angioplasty operator and also

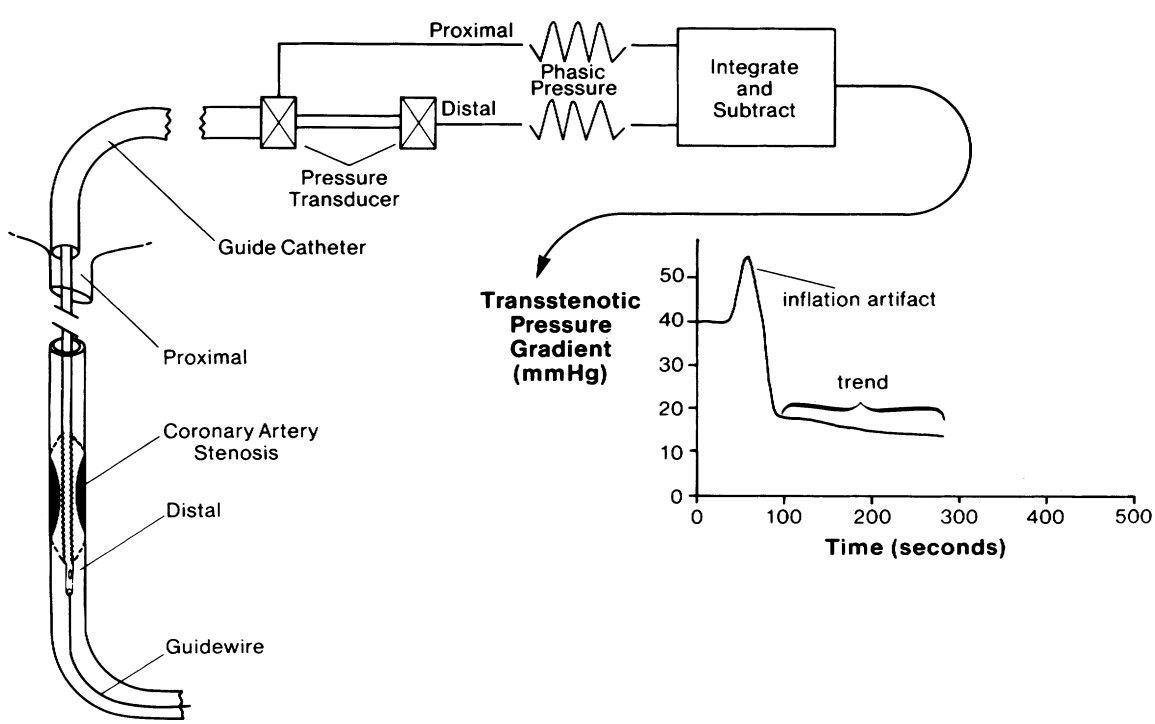


FIGURE 1. The guidewire and balloon catheter are positioned across the coronary artery stenosis. Distal arterial pressure is monitored through the balloon catheter, while proximal pressure is monitored through the guide catheter. Phasic pressures are electronically integrated and then distal pressure is subtracted from the proximal value to obtain the transstenotic pressure gradient. Plots of pressure gradient (mm Hg) are presented with time in seconds on the ordinate since pressure gradient (the abscissa) is available throughout the angioplasty procedure.

recorded on hard copy for subsequent analysis. During the time period of this study, gradient trends were recorded for variable periods of time depending on clinical circumstances. Absolute gradient values were used as a guide to completeness of dilatation in association with angiographic data. Trend information was not used at that time to determine the need for further inflations. During the period of the study, no decisions concerning the need for CABG were based on trend data.

In figure 1, a simplified example of this transstenotic pressure gradient record is shown. The transstenotic pressure gradient was 40 mm Hg before inflation. During inflation, the pressure gradient increased reflecting the difference between the mean proximal pressure and the distal coronary artery pressure. After balloon catheter deflation, the pressure gradient rapidly fell, completing the "inflation artifact." After the inflation artifact, the pressure gradient may gradually stabilize and decline, as illustrated in this figure, or may slowly increase. We defined the dynamic behavior of the pressure gradient in the time interval after balloon deflation as the transstenotic pressure gradient "trend."

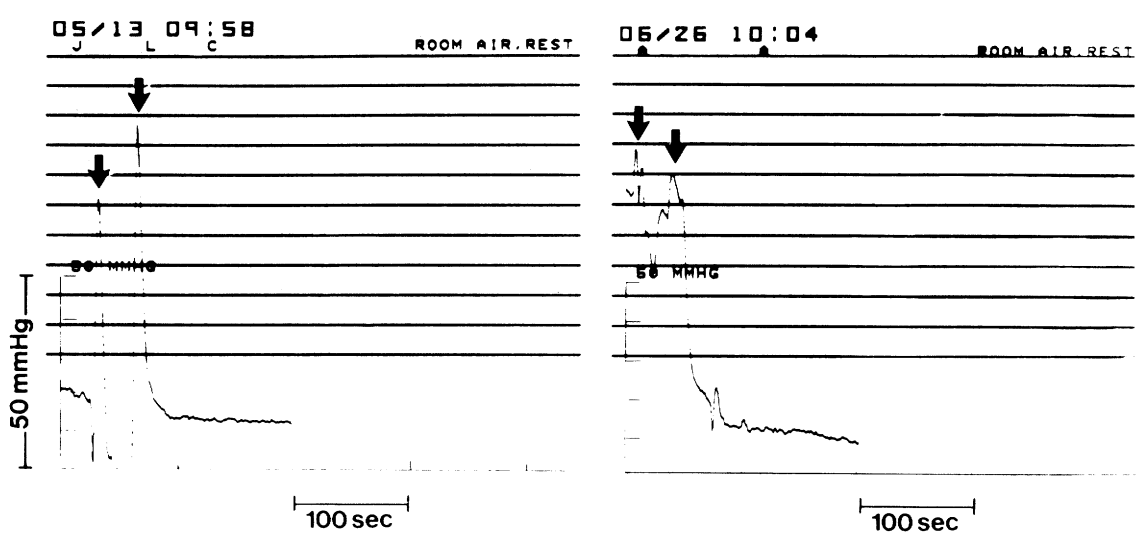
Classification of trends. Classification of transstenotic pressure gradient trends required a minimum of 25 sec of undisturbed recording. Two pressure gradient trend patterns were defined. A stable trend was defined as a horizontal or slowly declining trend after the balloon inflation artifact (figure 2A). We observed no difference in outcome between these two trend patterns and thus combined them as defining a stable pattern for the purpose of the study. A rising trend was defined as one in which the pressure gradient increased after the inflation artifact (figures 2B and 2C). Because multiple balloon inflations were often performed during a procedure, more than one trend pattern was observed. The following hierarchy was adopted to allow each procedure to be given a single overall classification. If any rising trend pattern was observed, the procedure was classified as overall rising. If only stable trend patterns were observed, the procedure was classified as overall stable. Analysis was also performed with the use of the trend classification after the final balloon inflation.

Coronary angiography. Cineangiography was performed by the percutaneous transfemoral technique and recorded on 35 mm film in at least two projections both before and after all PTCA procedures. The films were viewed prospectively, with data elements recorded on standard forms for entry into a cardiac data base.

Intimal tear (intimal disruption limited to the lesion) was defined as the angiographic appearance of an intraluminal filling defect, extravasation of contrast material, or a linear luminal density or staining within the confines of the original lesion undergoing dilatation.[9, 10] Arterial dissection was defined as the presence of any of these angiographic findings extending proximally or distally beyond the length of the original lesion. Percent diameter stenosis before and after PTCA was assessed by taking the mean of at least two projections[11] with a digital caliper technique.[12] Angiographic success was defined by a 20 or greater percentage point reduction in the luminal percent diameter stenosis.[13] Lesions were also defined as either concentric or eccentric,[14] and either discrete (single lesions <10 mm in length) or multiple.

Clinical outcome. All patients were monitored closely with electrocardiographic telemetry in a specialized nursing unit or in an intensive care unit for at least 48 hr after PTCA. Twelve-lead electrocardiograms were performed within 1 hr after the procedure and at least once during each following 24 hr period. Creatine kinase levels, with an assessment of MB isoenzymes, were measured in all patients at 8 and 16 hr after they were returned to the floor. Episodes of chest discomfort were recorded, and depending on the circumstances, patients underwent another ECG examination and consultation by the angioplasty operator.

Acute vessel closure was defined as a syndrome characterized by the acute onset of ischemic chest pain, with or without ECG evidence of myocardial ischemia that was not relieved by med-



STABLE

FIGURE 2A. Recordings of the transstenotic pressure gradient during PTCA illustrates stable pressure gradient trends. On the *left*, the pressure gradient remains constant, while on the *right* it slowly declines. Note the characteristic inflation artifact (arrows) in each case.

ical therapy, required emergency coronary angiography or emergency CABG and/or resulted in myocardial infarction or death. Emergency surgery was defined as CABG performed after PTCA because of acute ischemia unrelieved by either medical therapy or repeat PTCA, or because the initial angiographic appearance of the dilated artery suggested the patient was at increased risk for acute closure and myocardial infarction. Myocardial infarction was defined as the presence of new pathologic Q waves (≥40 msec), and/or an increase in creatine kinase (CK) to 520 or more units (three times the upper limit of normal in our laboratory); measures of CK must have included CK-MB isoenzyme subfractions.

Statistical analysis. Clinical and angiographic data and pressure gradient trend classifications representing both categorical and continuous data elements were recorded on a VAX-750 computer system. Univariate statistical analysis of these data elements was achieved with the SPSS-X program. Chi-square analysis was performed on categorical data elements. Student's t test was performed on continuous data elements. Multivariate analysis of these data with categorical variables serving as end points was achieved through stepwise logistic regression and BMDP statistical software. Statistical significance was indicated when p values of ≤.05 were achieved.

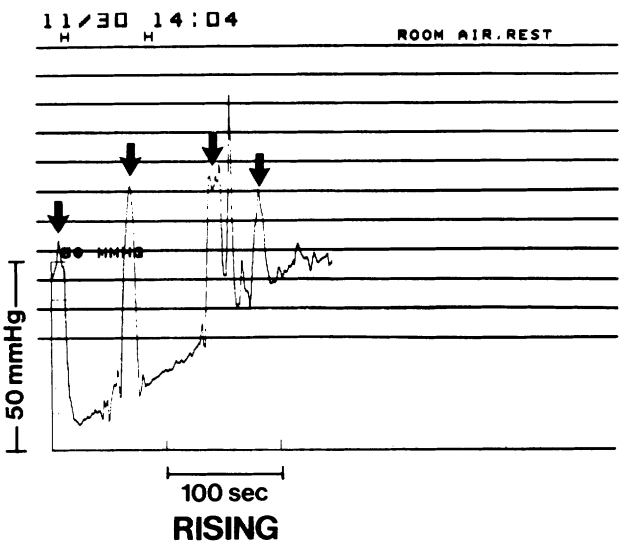


FIGURE 2B. Recordings of the transstenotic pressure gradient during PTCA illustrate a rising pressure gradient trend. The pressure gradient rises consistently after each inflation (artifact is indicated by arrows). The pressure gradient increases at an angle approximately 37 degrees above the horizontal (rise angle).

Results

Clinical and angiographic characteristics for patients with stable and rising trend patterns are detailed in tables 1 to 3. A total of 464 stenoses were dilated (maximum of four) with an average of 1.2 stenoses per patient. Of the total 380 procedures, intimal tear was observed in 110 patients (29%), arterial dissection occurred in 38 patients (10%), acute vessel closure occurred in 23 patients (6%), emergency CABG was necessary in seven patients (2%), and myocardial infarction occurred in 14 patients (4%). There were no deaths. Stable pressure gradient trends were observed during 308 procedures (81%) and a rising trend pattern was seen during 72 procedures (19%).

Patients in the two groups were similar in age, sex, history of prior myocardial infarction, history of diabetes mellitus, duration of angina pectoris, and levels of blood cholesterol. The rising trend group included

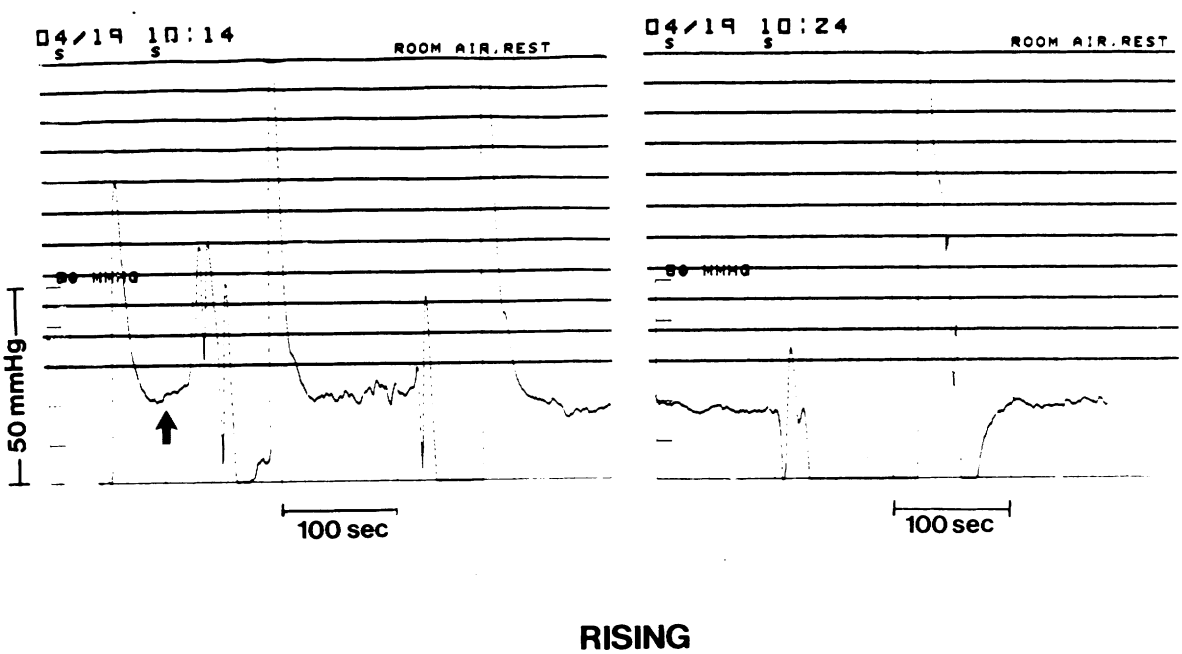


FIGURE 2C. The pressure gradient rises at 29 degrees after the first inflation (arrow) in this otherwise characteristic pressure gradient trend recording that spans two separate recordings. The gradient stabilizes before the final deflation. Intimal tear was the only event occurring in this case.

TABLE 1
Characteristics of the study patients

	Stable pattern (n = 308)	Rising trend pattern (n = 72)	p value
Age (yr)	56 ± 10	58 ± 10	NS
Sex			
Male	253 (82%)	53 (74%)	NS
Female	55 (18%)	19 (26%)	
History			
Prior MI	109 (35%)	30 (42%)	NS
Diabetes	24 (8%)	8 (11%)	NS
Unstable angina	152 (49%)	50 (69%)	<.005
Duration angina (mo)	17 ± 32	14 ± 33	NS
Cholesterol (mg%)	229 ± 46	234 ± 54	NS
PTCA vessel	(n = 376)	(n = 88)	
LAD	175 (47%)	52 (59%)	<.05
Proximal	141 (81%)	45 (87%)	NS
LCx	72 (19%)	18 (20%)	NS
Proximal	22 (31%)	10 (56%)	<.05
RCA	129 (34%)	18 (21%)	<.025
Proximal	93 (72%)	15 (83%)	NS

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

more patients presenting with unstable angina (69% vs 49%, p<.005), higher percentages of left anterior descending artery stenoses (59% vs 47%, p<.05) and proximal circumflex stenoses (56% vs 31%, p<.05), and fewer right coronary artery lesions (21% vs 34%, p<.025).

Patients with rising trends also had fewer lesions with discrete morphology (72% compared with 83%, p<.025). There was no significant difference in eccentricity, presence of calcium within the lesion, mean percent diameter stenosis before PTCA or after PTCA, or mean pressure gradient before PTCA. The rising trend group had a higher pressure gradient after PTCA (final absolute gradient 17 ± 9 vs 12 ± 8 mm Hg, p<.0001).

TABLE 2
Angiographic characteristics of the study patients

	Stable pattern (n = 308)	Rising trend pattern (n = 72)	p value
Lesion morphology	(n = 376)	(n = 88)	
Discrete	313 (83%)	63 (72%)	<.025
Eccentric	190 (51%)	49 (56%)	NS
Calcified	12 (3%)	3 (3%)	NS
Extent of disease			
Single-vessel disease	216 (70%)	51 (71%)	NS
Double-vessel disease	77 (25%)	18 (25%)	NS
Triple-vessel disease	15 (5%)	3 (4%)	NS
Arteriography			
Lesion length	8 ± 4 mm	8 ± 5 mm	NS
Ejection fraction	57 ± 8	58 ± 9	NS
Mean pre-PTCA % diameter	75 ± 13	77 ± 14	NS
Mean post-PTCA % diameter	26 ± 12	27 ± 16	NS
Intimal tear	87 (28%)	23 (32%)	NS
Arterial dissection	20 (7%)	18 (25%)	<.0001
Tear or dissection	107 (35%)	41 (57%)	<.001

TABLE 3
Hemodynamic and clinical outcome in the study group

	Stable pattern (n = 308)	Rising trend pattern (n = 72)	p value
Hemodynamics			
Mean pre-PTCA gradient (mm Hg)	52 ± 16	54 ± 14	NS
Mean post-PTCA gradient (mm Hg)	12 ± 8	17 ± 9	<.0001
Clinical outcome			
Acute vessel closure	11 (4%)	12 (17%)	.0001
Emergency CABG	3 (1%)	4 (4.6%)	<.05
Myocardial infarction	5 (2%)	9 (13%)	<.0001

Patients with a rising trend pattern had a higher incidence of intimal tear or arterial dissection (57% vs 35%, p<.001, with an odds ratio of 2.5) and arterial dissection alone (25% vs 7%, p<.0001, odds ratio 4.8), but not a higher incidence of isolated intimal tear alone (figure 3). A rising trend was associated with an increased incidence of acute closure (17% vs 4%, p = .0001, odds ratio 5.4), emergency CABG (5.6% vs 1%, p<.05, odds ratio 6.0), and myocardial infarction (13% vs 2%, p<.0001, odds ratio 8.7; figure 4).

In the rising trend group, the average duration of observation after inflation artifact was 72 ± 50 sec (table 4). Patients requiring emergency CABG had a

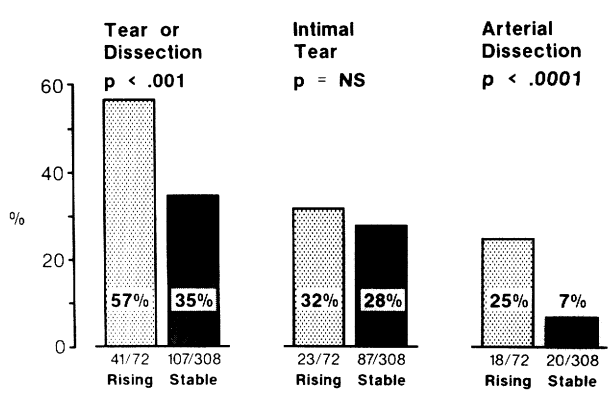


FIGURE 3. Incidence of intimal tear or arterial dissection for the two trend classifications. Odds ratio (OR) for intimal disruption was 2.5; OR for arterial dissection was 4.8.

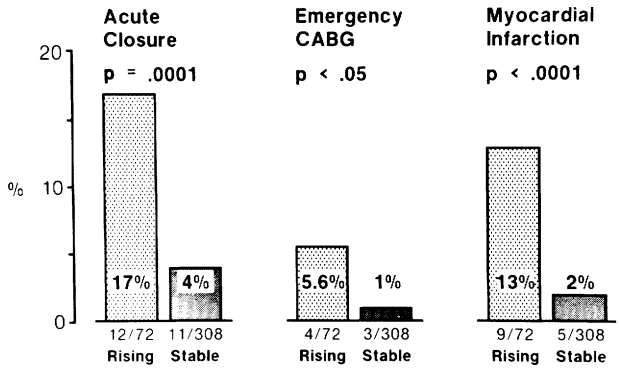


FIGURE 4. Incidence of acute vessel closure, emergency CABG, and myocardial infarction for the two trend classifications. OR (odds ratio) = 5.4 for acute vessel closure; OR = 6.0 for emergency CABG; OR = 8.7 for myocardial infarction.

significantly greater rise angle (27.9 ± 9.1 degrees) than the group of patients with no angiographic or clinical evidence of complications (20.6 ± 8.4 degrees, $p<.05$).

When the trend following the final balloon deflation (instead of "any" rising trend) was used to define those in the rising group (n = 31), nine of 12 patients with acute vessel closures, three of four undergoing emergency CABG procedures, and six of nine suffering myocardial infarctions were found to have a final rising trend pattern. The positive predictive value (PPV) and specificity of a rising trend pattern was increased when this modified definition was used; the PPV for acute vessel closure was 29% vs 17%, that for emergency CABG was 10% vs 6%, and that for myocardial infarction was 19% vs 13%.

The results of univariate analysis of the relationship between pressure gradient trend, intimal tear, arterial dissection, final absolute gradient, post-PTCA percent diameter stenosis, and the development of ischemic complications is shown in table 5. The post-PTCA percent diameter stenosis, presence of arterial dissection, and a rising trend pattern all significantly predicted the risk of acute vessel closure and emergency CABG. A final gradient of 15 mm Hg or more was only predictive of acute vessel closure. In the presence of a rising trend, the observation of intimal tear or arterial dissection added no additional information concerning the risk of acute vessel closure, emergency CABG, or myocardial infarction. In the absence of angiographic evidence of intimal disruption, the presence of a rising trend significantly increased at risk of acute vessel closure and myocardial infarction ($p<.0005$). The risk of emergency CABG was also increased, but due to the small numbers involved, significance was not achieved.

Independent pre-PTCA and post-PTCA predictors of an acute complication are listed in table 6. Lesion length and pre-PTCA percent diameter stenosis were both independent but weak predictors of an intimal tear. A rising gradient trend was the most important predictor of arterial dissection (odds ratio 1.99, $p = .002$), followed by lesion length, post-PTCA gradient, and post-PTCA percent diameter stenosis. A rising gradient trend was also the most important independent predictor of an acute closure event and myocardial infarction ($p<.001$ for both). The post-PTCA gradient was the only independent predictor of emergency CABG after PTCA.

Discussion

Broader application of PTCA depends largely on increasing its safety, particularly in patients with high-risk coronary anatomy. Acute closure of the dilated vessel during or after the PTCA procedure is the principal concern. This complication is responsible for myocardial infarction, death, and the morbidity associated with emergency CABG. Anticipation of acute closure by the angioplasty operator can reduce morbidity and mortality through close monitoring, timely medical intervention, and coordination of surgical support. The current ability to insert a perfusion catheter to restore distal coronary flow[15] makes identification of impending closure before removal of the guidewire even more important. The future availability of intracoronary stenting devices also emphasizes the importance of being able to predict acute closure events after PTCA.

In a previous study we demonstrated that evidence of intimal tear or dissection was associated with a sixfold increase in the risk of myocardial infarction, emergency CABG, and/or death.[4] In the present study,

TABLE 4
Characteristics of rising trend patterns (average time of observation 72 ± 50 sec)

Event	Average rise angle (degrees)	p value
Mean for entire group	22.7 ± 9.7	
No arteriographic evidence of intimal disruption or ischemic complications[A]	20.6 ± 8.4	
Intimal tear	23.8 ± 10.8	NS
Arterial dissection	24.2 ± 9.2	NS
Acute vessel closure	23.8 ± 9.7	NS
Emergency CABG	27.9 ± 9.1	<.05
Myocardial infarction	24.6 ± 10.6	NS

Values are mean ± SD.
[A]Acute vessel closure, emergency CABG, or myocardial infarction.

TABLE 5
Incidence and odds ratios (ORs) of ischemic complications for indicated events

Event	CABG	AC	MI
Tear only (n = 110)	3 (3%)	7 (6%)	4 (4%)
No tear or dissection (n = 232)	1 (0.4%)	9 (4%)	5 (2%)
	OR = 6.5, p = NS	OR = 1.7, p = NS	OR = 1.7, p = NS
Dissection only (n = 38)	3 (8%)	7 (18%)	5 (13%)
No dissection (n = 342)	4 (1%)	16 (5%)	9 (3%)
	OR = 7.2, p < .025	OR = 4.6, p < .005	OR = 5.6, p < .005
Gradient >15 mm Hg (n = 93)	4 (4%)	11 (12%)	6 (6%)
Gradient ≤15 mm Hg (n = 263)	2 (1%)	11 (4%)	7 (3%)
	OR = 5.9, p = NS	OR = 3.1, p < .02	OR = 2.5, p = NS
>50% diameter (n = 11)	3 (27%)	4 (36%)	1 (9%)
≤50% diameter (n = 369)	4 (1%)	19 (5%)	13 (4%)
	OR = 34, p < .0001	OR = 10.5 p < .0005	OR = 2.7, p = NS
Rising trend (n = 72)	4 (6%)	12 (17%)	9 (13%)
Stable trend (n = 308)	3 (1%)	11 (4%)	5 (2%)
	OR = 6.0, p < .05	OR = 5.4, p = .0001	OR = 8.7, p < .0001
Rising trend and tear only (n = 23)	0 (0%)	1 (4%)	1 (4%)
Stable trend and tear only (n = 87)	3 (3%)	6 (7%)	3 (3%)
	OR = 0, p = NS	OR = 0.6, p = NS	OR = 1.3, p = NS
Dissection only (n = 18)	3 (17%)	5 (28%)	4 (22%)
Stable trend and dissection only (n = 20)	0 (0%)	2 (10%)	1 (5%)
	[A], p = NS	OR = 3.5, p = NS	OR = 5.4, p = NS
Rising trend and no tear or dissection (n = 31)	1 (3%)	6 (19%)	4 (13%)
Stable trend and no tear or dissection (n = 201)	0 (0%)	3 (1.5%)	1 (0.5%)
	[A], p = NS	OR = 16, p < .0001	OR = 30, p < .0005

Final gradient was unrecorded in 24 patients.
[A]The odds ratio was undefined in those cases in which the incidence in the stable group was 0%.

39% of patients had angiographic evidence of some intimal disruption, yet only 6% had acute vessel closure. Angiographic evidence of intimal disruption is common and is, in itself, a suboptimal predictor of those patients who have ischemic complications. Also, with current imaging equipment, significant intimal disruption is not always evident before completion of dilatation. While severe intimal disruption may result in acute closure of the artery and the need for emergency CABG, its presence does not always imply a poor immediate or long-term result, e.g., the presence of an intimal tear without a significant residual pressure gradient has been shown to have a small but favorable influence on restenosis.[16] This study again emphasizes the relationship between the presence of disruption and subsequent acute ischemic complications. In addition, grading the degree of disruption into "tears" or "dissections" appeared useful in assessing their clinical significance. Arterial dissections, i.e., intimal disruption propagating beyond the confines of the original lesion, were predictive of all major complications, but intimal tear (filling defects and contrast extravasation limited to the original lesion) was not.

The definition of acute closure used in this study was not limited to angiographically documented arterial obstruction. This latter approach fails to account for the large proportion of patients with ischemic events after PTCA who proceed directly to CABG with either "clinical closure" or "threatened closure," nor does it account for the significant numbers of patients who suffer myocardial infarction after PTCA without undergoing repeat angiography. Therefore, our definition of acute closure includes all ischemic complications, including urgent or emergency CABG related to actual or threatened closure of the dilated artery. It also includes a small number of patients who undergo emer-

TABLE 6
Multivariate predictors of arteriographic outcome and ischemic complications

Event	Predictor	Odds ratio	p value
Intimal tear	Lesion length,	1.06	.038
	pre-PTCA % diameter	1.03	.006
Arterial dissection	Rising trend,	1.99	.002
	lesion length,	1.11	.007
	post-PTCA gradient,	1.06	<.001
	post-PTCA % diameter	1.04	.021
AC	Rising trend,	2.04	<.001
	post-PTCA gradient,	1.05	.046
	post-PTCA % diameter	1.05	.019
CABG	Post-PTCA gradient	1.13	<.001
MI	Rising trend	2.91	<.001

AC = acute vessel closure; MI = myocardial infarction.

gency repeat angiography because of acute ischemic chest pain with ECG changes who are found to have a patent vessel. In these cases, immediate administration of coronary vasodilators and anticoagulants probably promotes reopening of the vessel in the intervening period.

Andreas Gruentzig recognized the significance of the transstenotic pressure gradient when he first introduced the PTCA technique.[1] During normal resting conditions, arterial blood flow remains essentially constant as the lumen is reduced in diameter until a critical stenosis is reached,[17] at which point the flow rate begins to drop precipitously. Estimates of this critical stenosis diameter range from 60% to 80% (80% to 85% area reduction).[18] Thus, even relatively small increases in the luminal diameter brought about by balloon dilatation can produce significant reductions in the measured pressure gradient. Although several artifacts of pressure gradient recordings have been identified,[7] hemodynamic success has been measured through a reduction in this pressure gradient, with the absolute magnitude of the gradient serving as an end point for dilatation. The desirability of a low final absolute gradient has been emphasized by studies that have shown that the final gradient correlates with improvements in exercise-induced thallium perfusion abnormalities,[19] left ventricular dysfunction,[20] and coronary vasodilatory reserve flow after PTCA.[21] A higher residual gradient is related to an increased restenosis rate.[22–24] However, because the dilatation catheter is known to overestimate the true transstenotic pressure gradient,[25] its use as an absolute measure is somewhat limited.

Accurate prediction of acute vessel closure in the post-PTCA period has been suboptimal by use of angiographic or absolute hemodynamic measures. Examination of the dynamic behavior of the pressure gradient after each balloon deflation, regardless of absolute magnitude, provides the angioplasty operator with additional information. In the present study, the gradient trends were classified by experienced angioplasty operators who had been blinded to all angiographic data and the subsequent clinical outcome. Although the angiographic appearance of arterial dissection after PTCA was significantly associated with a subsequent ischemic complication, multivariate analysis demonstrated that information concerning the pressure gradient trend, absolute final gradient, and post-PTCA diameter stenosis were more useful in predicting these complications. Accurate measurements of the post-PTCA diameter stenosis, however, are not available during the procedure.

A number of pathophysiologic mechanisms may explain the existence of a rising trend pattern. After intimal disruption, the effective arterial lumen may be reduced by an expanding intramural hematoma. The hematoma may be compressed during balloon inflation and then reexpand after deflation; the pressure gradient would then rise, reflecting this progressive narrowing of the lumen. A flap of intima or plaque torn loose could also back-fill with blood, thereby reducing the effective vessel lumen and producing a rising pressure gradient trend. These dynamic changes in the pressure gradient trend may therefore reflect the underlying hemodynamic status of the arterial segment undergoing dilatation. Future angioscopic studies may clarify the pathologic basis for these observations.

Other mechanisms may also explain the generation of rising trends in patients without angiographic evidence of intimal disruption. Vasospasm is responsible in some episodes. In the present study, a small number of patients had rising trend patterns reversed after the administration of intracoronary nitroglycerin. An additional subset of patients had rising trends that stabilized after repeat balloon inflations, possibly indicating the stabilization of an intramural hematoma or intimal flap. An increase in blood flow in the presence of a morphologically stable stenosis could also produce an increase in the transstenotic pressure gradient.[26, 27] Such mechanisms may explain the reduced specificity (82%) of a rising trend as a predictor of ischemic complications.

In this study, the dichotomy of trend classification was based on the observation of at least one rising trend during dilatation. This definition was adopted because it was believed that once this pattern was observed, the substrate for the development of potential complications existed. Subsequent trends might be stabilized by

repeat dilatations, but the potential for further problems remains. This view was supported by the finding that the presence of any rising trend was a more sensitive predictor of ischemic complications that was use of the trend pattern after the final balloon deflation. As expected, use of the latter definition increased the specificity of the finding.

A rising pressure gradient trend should alert the angioplasty operator to the presence of an incomplete dilatation and to the potential for acute vessel closure. The patient should be observed for an extended period of time before removal of the guidewire from the coronary artery. Should acute closure occur with the guidewire in place, the stenosis can be recrossed safely for repeat dilatation, placement of an intracoronary stent, or placement of an intracoronary perfusion catheter before proceeding to bypass surgery.[15] Surgery performed in the absence of acute ischemia or hemodynamic compromise reduces morbidity and mortality and allows the surgeon extra time to use internal mammary artery conduits if desired. If a rising trend pattern persists in spite of multiple balloon inflations, dilatation efforts can be terminated if the patient is comfortable and there is angiographic evidence of an adequate arterial lumen and good distal flow after contrast injection. The patient should then be monitored in an intensive care unit with adjunctive anticoagulant therapy and close attention to maintaining adequate coronary perfusion pressure by avoiding systemic hypotension. If acute vessel closure occurs, rapid intervention can be initiated. Emergency CABG is useful in certain patients, e.g., those in whom the coronary anatomy or the initial angiographic result makes redilatation undesirable. Emergency repeat PTCA is effective in managing other patients.[28, 29] Additionally, recatheterization permits the opportunity to quickly stabilize the patient with a perfusion catheter while surgical facilities are being prepared.

In conclusion, the transstenotic pressure gradient trend provides valuable information to the angioplasty operator concerning the hemodynamic status of the coronary artery segment undergoing dilatation. A rising trend is often related to the presence of intimal tear and arterial dissection and may serve to identify those patients at risk for acute vessel closure, myocardial infarction, emergency surgical revascularization, or possibly in the future placement of intracoronary stenting devices.

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