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Transstenotic Coronary Pressure Gradient Measurement in Humans: In Vitro and In Vivo Evaluation of a New Pressure Monitoring Angioplasty Guide Wire

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Objectives. The present study was designed to investigate 1) the feasibility and accuracy of coronary pressure measurements with a novel 0.015-in. (0.038 cm) fluid-filled guide wire, and 2) the effect of the guide wire itself on stenosis hemodynamics.

Background. To assess the functional results of coronary angioplasty, measurements of the transstenotic pressure gradient have been advocated. However, this gradient is no longer routinely measured because it is not reliable when determined with the angioplasty catheter.

Methods. A fluid-filled 0.015-in. guide wire to be connected to a conventional pressure transducer was developed. Five wires were tested for their frequency response characteristics and for their accuracy in measuring hydrostatic pressure. In an in vitro model of stenosis (reference diameter 4 mm), the pressure gradient was determined at incremental flow levels for varying stenosis severity with and without a 0.015-in. guide wire through the narrowing. In 37 patients, the transstenotic pressure gradient was measured before and after angioplasty and compared with obstruction area and percent area stenosis as determined by quantitative coronary angiography.

Results. The correlation between the actual pressure and the pressure recorded by the guide wire was excellent (r = 0.98)

Measurement of the distal coronary pressure in the setting of percutaneous transluminal coronary angioplasty has proved to yield useful clinical information. 1) The translesional pressure gradient is a functional index of coronary stenosis severity taking into account all morphologic determinants of the lesion for a given coronary flow (1,2). 2) During balloon inflation, coronary wedge pressure reflects the functional importance of collateral vessels during coronary occlusion (3,4). 3) The reduction of the pressure gradient after dilation has been used to confirm the primary success of the procedespite a slight underestimation ($-3 \pm 5\%$). Phasic pressure recordings were precluded by a long time constant of 16 \pm 4 s. The presence of the guide wire produced a significant overestimation (>20%) of the pressure decrease only in cases of tight stenosis (>90% area reduction). Furthermore, a theoretic model based on the fluid dynamic equation predicted that this overestimation was inversely proportional to the reference diameter of the vessel, yet was only slightly influenced by the flow. The lesion was crossed in all but one patient (97%) and pressure gradient was recorded throughout the study in 34 (94%) of 36 patients. The mean pressure gradient decreased from 30 ± 19 before to 3 ± 5 mm Hg after angioplasty (p < 0.01). A curvilinear relation was found between the pressure gradient and both percent area stenosis $(r^2 = 0.67)$ and obstruction area $(r^2 = 0.72)$. A sharp increase in pressure gradient was noted once the stenosis exceeded 75% area reduction.

Conclusions. Mean transstenotic pressure gradients can be easily and reliably recorded with a 0.015-in. fluid-filled guide wire. This ability should facilitate the functional assessment of coronary stenoses of intermediate severity and of immediate postangioplasty results.

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dure (5), and 4) the final translessional gradient as well as the coronary wedge pressure has been found to be predictive of early (6,7) or late (8,9) recurrent ischemia. Nevertheless, measurements of the distal coronary pressure are no longer performed routinely during angioplasty not only because of the increasing use of monorail catheter systems, but more importantly, because of the known overestimation of the true pressure gradient, caused by the presence of the balloon catheter across the stenosis. To measure the distal pressure, the use of a thin angioplasty guide wire should theoretically overcome both limitations. Accordingly, a fluid-filled angioplasty pressure monitoring guide wire was developed. The purpose of this study was twofold: 1) to investigate in vitro and in vivo the accuracy and feasibility of the pressure measurements with this pressure monitoring guide wire, and 2) to investigate the influence of the presence of the guide

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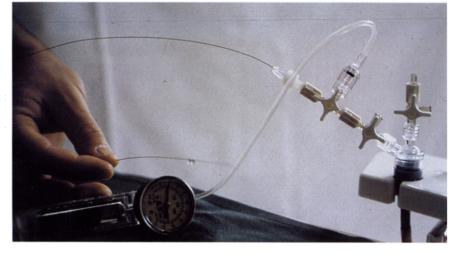


Figure 1. Photograph of a 0.015-in. fluid-filled guide wire connected to a pressure transducer. The angioplasty inflation device is filled with heparinized saline solution and allows for frequent flushing of the lumen of the wire.

wire itself through a coronary stenosis on coronary hemodynamics.

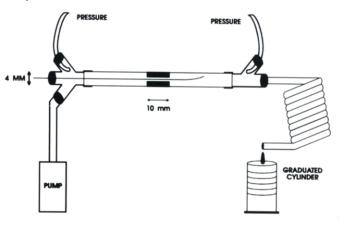
Methods

The guide wire. The pressure monitoring guide wire is a steerable angioplasty guide wire developed by Advanced Cardiovascular Systems. Pressure is transmitted through a fluid column. The proximal 129-cm section consists of a Teflon-coated hypotube with an external diameter of 0.015 in. (0.038 cm). The next 45 cm is made of a hypotube coaxial to a core wire. Ten 0.002-in. (0.005 cm) diameter pressure monitoring ports are located 3 cm proximal to the tip at the junction of the radiopaque and the nonradiopaque portion of the guide wire. The 3-cm tip is radiopaque, flexible (floppy) and capable of being shaped. The guide wire can be connected to a conventional pressure transducer for pressure monitoring (Fig. 1).

In vitro testing of the guide wire characteristics and the accuracy of the pressure measurements. Five pressure monitoring guide wires were tested in a hydrostatic model in a water-filled barrel connected to a Silastic tube with an inner diameter of 1.2 cm. The distal end of this tube was Y shaped. A Millar microtipped-catheter transducer (SP-780 C) was introduced in one leg of the Y and the pressure monitoring guide wire to be tested and a 5F right Judkins coronary catheter were introduced in the other leg. Measurements at different pressures of 0, 50, 100 and 150 cm H₂O were performed by adjusting the height of the barrel. At the start of the experiment and again at the end of the experiment, both the Millar catheter and the Judkins catheter were tested at the four pressure levels and this was repeated as a control measurement of the pressure transducers. The pressure monitoring wires were tested consecutively in the eight following steps: from 0 to 50 cm H₂O, 50 to 100 cm H₂O, 100 to 150 cm H_2O , 150 to 0 cm H_2O , 0 to 150 cm H_2O , 150 to 100 cm H_2O , 100 to 50 cm H_2O and 50 to 0 cm H_2O . At every level, pressure measured with the Millar manometer and the pressure monitoring guide wire were simultaneously recorded until the guide wire pressure reached a steady state. The absolute pressures were compared and the percent differences calculated. Moreover, the time constant for the pressure monitoring guide wire (defined as the time required for the signal to reach 63.2% of its final value) was calculated for every step.

In vitro testing of the influence of a guide wire on stenosis dynamics. The in vitro model of stenosis is illustrated in Figure 2. Seven 10-mm long narrowings were created in rigid plastic tubes with an inner diameter of 4 mm and a length of 12 cm. The percent area reduction of the stenosis ranged from 50% to 95% (50%, 60%, 70%, 80%, 85%, 90%, 95%). One end of the tube was attached to a connector with three side arms. A high fidelity pressure monitoring catheter (model 110-4, Camino Laboratories) was advanced through one valve of this connector. The 0.015-in. (0.038 cm) pressure monitoring wire was placed through the valve of the second arm and the third arm was connected to a power injector (Mark IV, Medrad Inc.). The other end of the tube was attached to a conventional angioplasty Y connector. A second high fidelity pressure monitoring catheter was introduced through the valve of this connector, and the other arm was left open. The power injector was filled with saline

Figure 2. In vitro model of coronary stenosis (see text).



solution and constant flow rates of 0.5, 1, 1.5, 2, 3, 4 and 5 ml/s were infused in the stenosis model. The accuracy and constancy of the flow rates were controlled with a graduated cylinder placed under the distal opening of the system. For each level of stenosis severity, pressures were recorded proximally and distally to the stenosis for the different flow rates. Each measurement was performed successively without and with the guide wire through the narrowing.

Theoretic calculation of the influence of a guide wire on stenosis dynamics. In a theoretic model, the fluid dynamic equation was used to calculate the theoretic overestimation of the pressure gradient produced by the presence of the guide wire itself through the stenosis. The overestimation of the gradient determined with the guide wire advanced through the lesion as compared with the true transstenotic gradient (without the guide wire in the obstruction) was studied in a 10-mm long lesion of increasing severity in vessels of different reference diameters at constant flow rates of 1 and 5 ml/s. The overestimation was calculated for two different guide wire cross-sectional diameters: 0.015 in. and 0.018 in (0.045 cm). The fluid dynamic equation for calculation of the pressure decrease across stenoses follows:

$$\Delta \mathbf{P} = \frac{8\pi\mu L}{\mathbf{A}_{\mathrm{s}}^2} \cdot \mathbf{Q} + \frac{\rho}{2} \left(\frac{1}{\mathbf{A}_{\mathrm{s}}} - \frac{1}{\mathbf{A}_{\mathrm{n}}}\right)^2 \cdot \mathbf{Q}^2,$$

where ΔP = mean pressure gradient (mm Hg), μ = blood viscosity (0.03 g/cm per s), L = stenosis length (10 mm), A_s = stenotic area (mm²), A_n = reference area (mm²), ρ = blood density (1 g/ml) and Q = mean flow (ml/s).

In vivo studies. The study group consisted of 37 patients (29 men and 8 women with a mean age of 51 ± 14 years) who underwent coronary angioplasty of the left anterior descending (n = 14), right (n = 15) or left circumflex coronary artery (n = 8). Nineteen patients had a type A lesion, 12 a type B lesion and 6 a type C lesion. Total coronary occlusions were excluded from the study. The patients were selected for the study only if the stenotic segment to be dilated could reliably be analyzed by quantitative coronary angiography in at least two projections. A 9F introduction sheath was inserted in the temoral artery and an 8F Judkins guiding catheter was used to cannulate the coronary ostium. The side arm of the sheath and the guiding catheter were each connected to a Spectranetics P23 Statham pressure transducer. The pressure monitoring guide wire was flushed with heparinized saline solution and attached by two three-way high pressure stopcocks to the pressure transducer. The side arm of the distal stopcock was connected to an inflation device filled with heparinized saline solution for frequent and powerful flushing of the guide wire. Zero reference for the three pressure transducers was at mid-chest level. The pressure monitoring guide wire was placed at the tip of the guiding catheter, where mean and phasic guiding catheter pressure and guide wire pressure were simultaneously recorded. After administration of 2 mg of intracoronary isosorbide dinitrate, the guide wire was advanced through the stenotic segment while the phasic pressure of the femoral sheath and the mean pressure of both the guiding catheter and the guide wire were continuously recorded. Care was taken to perform the registration ≥ 3 min after the last injection of contrast medium. In most cases, the guide wire could be advanced through the lesion while remaining connected to the pressure transducer. When this was not possible, the guide wire was disconnected from the transducer for better torque control. Only the radiopaque tip was placed distal to the narrowing so that the side holes remained proximal to the stenosis. The proximal part of the guide wire was again connected to the pressure transducer. Under continuous pressure monitoring, the guide wire was advanced so that the side holes were positioned across the stenosis. This maneuver made it possible to record the gradient in all cases by advancing the distal holes of the guide wire through the lesion. The dilations were performed with "monorail" angioplasty catheter systems. After angioplasty was completed, the balloon catheter was retrieved from the dilated segment, the pressure transducers were referenced to zero again and 2 mg of intracoronary isosorbide dinitrate was injected. The final translesional gradient was measured again ≥ 3 min after the last balloon inflation and the last contrast medium injection. While the mean pressure was recorded, the guide wire was pulled back into the guiding catheter where the mean preslare of the guiding catheter and the pressure monitoring guide wire were again compared.

Quantitative coronary angiography. Quantitative analysis of the selected coronary segment was carried out by the computer-based Cardiovascular Angiography Analysis System (CAAS), which has been validated and described in detail by Reiber et al. (10). Briefly, edges of the relevant coronary artery segment are detected automatically from an end-diastolic digitized frame of the coronary angiogram. The absolute diameter of the stenosis is determined using the guiding catheter as a calibration device. A computer estimation of original dimension at the site of the obstruction is used to define the interpolated reference area or diameter. The interpolated percent area stenosis and the minimal lumen cross-sectional area are calculated and averaged from at least two perpendicular projections.

Statistics. All values are expressed as mean value ± 1 SD. The Student *t* test was used to compare paired data. Linear regression coefficients were obtained by the least squares method. The goodness of fit of the curvilinear relation between the pressure gradient and either percent area stenosis or obstruction area was assessed using chi-square statistics. A p value < 0.05 was used to define statistical significance.

Results

In vitro studies. As expected, true hydrostatic pressure and the pressure recorded by the Millar catheter were identical, and therefore the latter was used as an equivalent of the true pressure. Pressure recordings by the 5F right

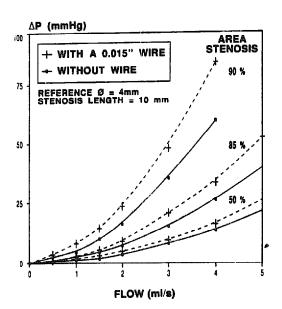


Figure 3. Plot of the in vitro-measured pressure gradient (ΔP) for varying stenosis severities (50%, 85% and 90% area stenosis) at incremental flow rates. Each measurement was obtained with (continuous line) and without (broken line) the 0.015-in. guide wire through the stenosis.

Judkins catheter approximated the true pressure very well within negligible time, ensuring the accuracy of the pressure transducers used. There was an overall excellent correlation (r = 0.98) between both pressure measurements. The pressure recorded by the pressure monitoring guide wires slightly underestimated the true pressure in all cases. The percent difference between both measurements was $-3 \pm 5\%$ (n = 15). The time constant for all steps in a single pressure monitoring guide wire was almost identical. The mean time constant varied from 9 to 27 s for the five guide wires, with a mean value of 16 ± 5 s.

Figure 3 shows the pressure gradient measured in vitro through stenotic models. In mild lesions (50% area stenosis), the difference in the pressure decrease measured without and with the 0.015-in. guide wire through the lesion remained small (<5 mm Hg) even at high flow rates (5 ml/s). In contrast, in severe stenoses (≥90% area stenosis), a significant overestimation of the true gradient was induced by the presence of the guide wire itself through the stenotic segment even at relatively low flow rates. For example, in a stenosis of 90% area reduction at a flow rate of 2 ml/s, the pressure gradient measured across the lesion was 23 mm Hg with the guide wire compared with only 16 mm Hg without the guide wire. In intermediate lesions, the overestimation became significant only at high flow rates. For example, in an 80% area stenosis (obstruction area 2.5 mm² in our in vitro model), the measured gradient was 23% greater with than without the guide wire (5.9 versus 4.8 mm Hg) for a 3 ml/s flow. At the flow rate of 5 ml/s, the gradient obtained was 25% greater with than without the guide wire (36 vs. 27 mm Hg).

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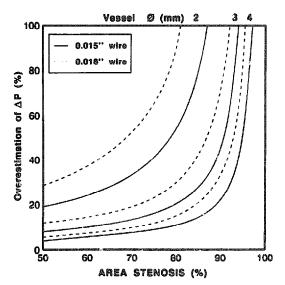


Figure 4. Computer-derived calculation of the overestimation of the pressure gradient (P) due to the presence in a 10-mm long stenosis of a 0.015-in. guide wire (continuous line) and of a 0.018-in. guide wire (broken line) for varying area stenoses and reference diameters. Flow rate is 1 ml/s.

Theoretic model. Results from the in vitro studies were confirmed by the theoretic model using the Newtonian fluid dynamic equation (Fig. 4). The percent overestimation in pressure gradient due to the presence of the guide wire through the stenosis increased as an exponential function of the percent stenotic area as well as an inverse function of the reference diameter of the diseased vessel. In a 2-mm vessel, an overestimation >20% occurred from a 60% area stenosis. In a 3- and 4-mm vessel, the overestimation in pressure gradient due to the presence of the 0.015-in. guide wire through the lesion was >20% when the area stenosis exceeded 80% and 90%, respectively. When a 0.018-in, guide wire was advanced through the obstruction, a 20% overestimation was reached once the area stenosis exceeded 84% in a 4-mm vessel and 72% in a 3-mm vessel. In contrast, the flow influenced the percent overestimation of the pressure gradient very little (Fig. 5).

In vivo studies. The target vessel could be reached in every case. With the exception of one lesion, all (97%) were crossed with the pressure monitoring guide wire without any particular difficulty and balloon angioplasty was successfully completed without the need for another guide wire and without any complication. The only lesion that could not be crossed with the 0.015-in. pressure monitoring guide wire was an eccentric 88% area stenosis followed by a bend of approximately 80° in the mid-left anterior descending artery. The plot of the pressures measured by the guiding catheter and by the guide wire are shown in Figure 6. A good correlation with a regression line close to the line of identity was present both before and after angioplasty.

The phasic pressure tracings were very damped compared with those simultaneously obtained by the guiding

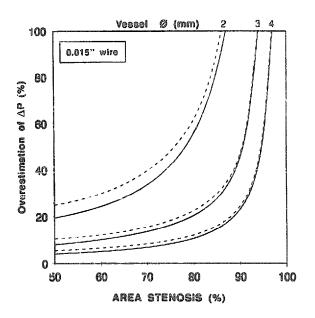


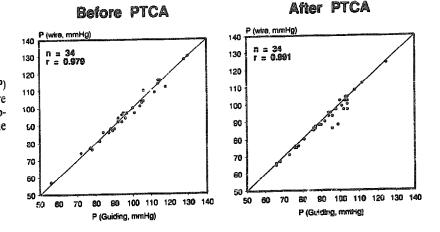
Figure 5. Plot of a computer-derived calculation of the overestimation of the pressure gradient (ΔP) measured with a 0.015-in. wire as a result of the presence of the wire itself in the stenosis. The pressure gradients were calculated by means of a fluid dynamic equation (see text). The overestimation was determined for incremental levels of percent area stenosis (from 50% to 100%), for incremental reference diameters (from 2 to 4 mm) and for two different flow rate values (1 ml/s [continuous line] and 5 ml/s [broken line]).

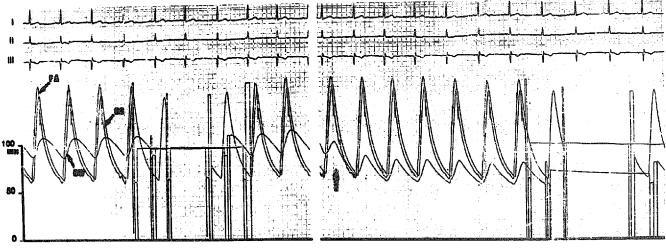
catheter (Fig. 7). The mean transstenotic pressure gradient decreased from 30 ± 19 mm Hg before to 3 ± 5 mm Hg after angioplasty (p < 0.01). The relation between the measured pressure gradient and both the minimal obstruction area (before and after angioplasty) and the percent area stenosis was best fit by a quadratic equation as an inverse function of the obstruction area and the percent area stenosis, respectively. This relation showed a steep decrease in gradient once the obstruction area was >1.6 mm² and a steep increase in gradient once the percent area reduction was >80%. Although significant, the relation was rather weak, with some marked differences in measured gradient for stenoses of similar angiographic severity (Fig. 8).

Discussion

Need for transstenotic pressure gradient measurements. The significance of a coronary stenosis depends on its effect on coronary blood flow. Because regional coronary flow is difficult to measure during catheterization, the functional significance of a coronary stenosis is still mostly derived from its morphology at angiography. However, the accuracy of coronary angiography compared with that of postmortem examination (11,12) and inter- and intraobserver variability remain a major concern, especially after angioplasty (13,14). The need for a simple tool to provide information about the physiologic repercussion of coronary stenoses is evident when dealing with lesions of angiographic intermediate severity or with postangioplasty segments. Several methods were developed using either Doppler velocitometry, myocardial densitometry or quantitative coronary angiography. All are based on the concept of absolute coronary flow reserve (15-18) or on measurement of maximal achievable flow (19,20). However, in addition to some theoretic shortcomings, these methods require sophisticated equipment and, often, off-line, time-consuming data processing that preclude their use in unselected patients (21,22). In contrast, the pressure gradient across a coronary stenosis is basically simple to obtain and is a functional index of stenosis severity, taking into account all morphologic determinants of the lesion for a given coronary flow. Moreover, we (23,24) have recently shown that fractional flow reserve of epicardial coronary arteries and of the myocardium and the contribution of collateral blood flow to total flow can be determined solely from pressure measurements. To date, distal coronary pressure has been measured through the angioplasty balloon catheter. Yet, the presence of the catheter itself across the stenosis leads to an artifactual overestimation of the true pressure gradient because the functional stenotic lumen is further reduced by the balloon catheter. For comparison between the obstruction area and the transstenotic pressure gradient, this overestimation was accounted for in the study of Wijns et al. (25) by subtracting the cross-sectional area of the balloon catheter from the stenotic area measured from the coronary angiogram. However, in 33% of patients,

Figure 6. Correlation between mean pressure (P) measured by the guiding catheter and mean pressure obtained with the fluid-filled guide wire before angioplasty (PTCA) (left panel) and at the end of the procedure (right panel).





WIRE PROXIMAL TO THE STENOSIS

WINE DISTAL TO THE STENOSIS

Figure 7. Example of pressure tracing recorded with a 0.015-in. fluid-filled angioplasty guide wire. Compared with the pressure tracings recorded in the femoral artery (FA) and at the tip of the guiding catheter (GC), the pressure tracing recorded with the pressure monitoring guide wire (GW) is damped. However, the mean pressure measured by all three catheters is identical. After the distal side holes of the pressure monitoring guide wire are advanced distally to the lesion (arrow), the transstenotic pressure gradient can be measured.

the cross-sectional area of the catheter was larger than the obstruction area, leading to a complete occlusion of the segment by the deflated balloon. When anterograde flow is interrupted, the distal pressure is mainly determined by the collateral flow (3) and no longer provides reliable information about the stenosis itself. Even the 2F catheters used by Ganz et al. (26,27) would have been occlusive in approximately 40% of the preangioplasty stenotic segments that we investigated.

In vivo and in vitro results with the 0.015-in. pressure monitoring guide wire. In this study, we evaluated the accuracy of pressure measurements obtained with a newly developed 0.015-in. fluid-filled guide wire and tested the feasibility of using the device as a conventional angioplasty guide wire. As shown in the in vitro tests, the pressure monitoring wire approximated real hydrostatic pressure reasonably well in most cases once steady state pressure was reached. The mean underestimation of the real pressure reached $3 \pm 5\%$, depending on the wire tested. In vivo, the mean pressure measured with the open end wire advanced at the tip of the guiding catheter correlated very well with the mean pressure obtained by the guiding catheter introduced in the ostium of the coronary artery. This good concordance between both mean pressures also held true at the end of the procedure. Only two patients exhibited a difference >5% (Fig. 5). As expected from the small inner diameter of the guide wire, the time constant of the guide wire was long $(16 \pm 14 \text{ s})$, thus precluding the recording of phasic tracings. All but one procedure (97%) could be accomplished with the pressure monitoring guide wire without the need to use another guide wire to cross the lesion. No technical problems were encountered when crossing the lesion with the guide wire. However, the majority of the attempted lesions were of low or moderate difficulty because long, tortuous and eccentric lesions were often excluded from the study because they were not well suited for quantitative coronary

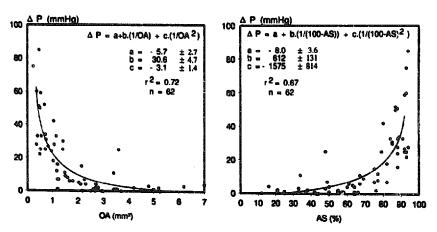


Figure 8. Relation between the transstenotic pressure gradient (ΔP) measured with the fluid-filled pressure monitoring guide wire and either the obstruction area (OA) (left panel) or the percent area stenosis (AS) (right panel) determined by quantitative coronary angiography.

angiography. The use of monorail angioplasty catheter systems greatly facilitated the manipulation of the guide wire and the pressure recordings. In two patients, the guide wire became obstructed, thereby excluding further pressure measurements. This finding emphasizes the need for frequent flushing of the guide wire.

Correlation with quantitative angiography. The exponential relation between percent area stenosis and obstruction area on the one hand and measured pressure gradient on the other confirms previous animal experiments in which a sharp decrease in coronary flow reserve was noted once the stenosis exceeded 75% area reduction (1). These similar relations strongly suggest the role of the mean pressure gradient as a functional index of severity of coronary stenosis. The scatter of the relation between area stenosis and pressure gradient underscores the important effect of factors other than area stenosis on the resistance to flow through a stenosis. Alternatively, even though the lesions could easily be analyzed by the CAAS system, the marked differences in measured pressure gradient for stenoses of similar angiographic severity can also be due in part to the inaccuracy of quantitative measurements, especially after angioplasty. Finally, the overestimation of the pressure gradient when measured with the guide wire through the lesion can also contribute to the difficulty in comparing anatomic and functional characterization of a coronary stenosis.

Influence of the presence of the guide wire across the lesion on stenosis hemodynamics. Because the cross-sectional area of the guide wire is small (0.114 mm²), one could expect no further increase in the pressure gradient. In our series of patients, the smallest obstruction area reached 0.24 mm². In this particular case, the presence of the guide wire further decreased the functional stenotic area by 50%, inducing an artifactual increase in the percent area stenosis from 93% to 96.5%. The in vitro results showed that in mild to moderate stenoses, the overestimation produced by the presence of the guide wire itself can be neglected because of the large ratio between obstruction area and cross-sectional area of the guide wire. According to the fluid dynamic equation, an inverse relation exists between the relative overestimation in pressure gradient due to the guide wire and the reference diameter of the vessel. This implies that in small coronary arteries, the overestimation of the measured pressure gradient produced by the presence of the guide wire itself will be larger than in large coronary vessels for a given percent area stenosis. At low flow rates (equivalent to basal coronary flow), the absolute overestimation of pressure gradient due to the presence of the guide wire in the obstruction remained small (Fig. 3 and 4). At high flow rates, these absolute values increased. Even if the relative value of overestimation was little influenced by the flow for the same degree of stenosis (Fig. 5), the level of pressure gradient when this overestimation was observed at high flow rate could induce a significant error (Fig. 3). This influence of a guide wire on coronary stenosis dynamics should be kept in mind when using recently developed Doppler (28) and micromanometer-

tipped (29) guide wares. record theless, these findings are not clinically relevant: the mean pressure necessary to increase the flow above 3 ml/s through a 90% area stenosis exceeds the physiologic range (>190 mm Hg). Maximal coronary flow is limited in tight stenoses and no major increase in flow is to be expected once the stenosis exceeds 80% diameter reduction (1). Furthermore, in clinical practice, these lesions do not require sophisticated measurements to be considered critical or to trigger adequate clinical decision. In lesions of intermediate severity and in postangioplasty segments where angiography often fails to be conclusive, the guide wire should provide a true pressure gradient value that can be helpful in clinical decision making. Finally, because the relative overestimation of the pressure decrease produced by the guide wire across the lesion is little influenced by the flow, the ratio of hyperemic to rest transstenotic gradient will not change significantly. However, neither the theoretic model of the fluid dynamic equation nor the in vitro model of coronary stenosis closely reflects the flow dynamics of a narrowed coronary artery in vivo. The entrance and exit angles almost never reach 90° in vivo; moreover, both models assume a nonpulsatile flow without taking into account the effects of the curvatures and the compliance of the coronary segments (30).

Advantages and disadvantages of the fluid-filled wires as compared with micromanometer-tipped guide wires. In addition to its very low cost, the main advantage of the system presented in this study is its extreme ease of use. Because this pressure monitoring guide wire has essentially the same technical characteristics of most of the currently available angioplasty guide wires, it can be used in a large majority of the attempted lesions and could even be proposed to measure gradients across dubious lesions during diagnostic catheterization. Because of the small cross-sectional area of the guide wire, only very limited hindrance to coronary hemodynamics is to be expected. The main disadvantage of this fluid-filled system is its inability to measure phasic pressure tracings, although only a mean pressure is required to assess the mean pressure gradient that reflects overall hydraulic properties of a stenosis. Because coronary blood flow occurs predominantly during diastole, a gradient may exclusively occur during diastole in mild stenoses as in postangioplasty segments. With this fluid-filled guide wire, systolic and diastolic gradients cannot be distinguished.

Conclusions. This study validates the feasibility and accuracy of distal pressure measurements made by a fluid-filled 0.015-in. guide wire during coronary angioplasty. Except when dealing with tight stenoses in small coronary arteries, this new device provides the opportunity to assess reliably the transstenotic pressure gradient in humans. The ease of use and the high success rate of this new pressure monitoring guide wire should facilitate the evaluation of the functional repercussion of coronary narrowings and improve the assessment of immediate postangioplasty results. We greatly appreciate the skillful technical assistance of Jan Stockbroeckx, Danny De Moor and Olivier Neils, as well as the dedicated secretarial help of Josefa Cano.

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