Functional Significance of Collaterals During Ameroid-induced Coronary Stenosis in Conscious Dogs

Interrelationships Among Regional Shortening, Regional Flow and Grade of Coronary Stenosis

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SUMMARY We studied the relationships among collateral flow, regional myocardial shortening and the grade of coronary stenosis during ameroid-induced chronic coronary constriction in 22 conscious dogs. A radiolucent ameroid, a Doppler flow probe and a cuff occluder were placed on the left circumflex coronary artery (LCx). Regional myocardial shortening and regional myocardial blood flow were assessed simultaneously using ultrasonic dimension gauges and the tracer microsphere technique, respectively, during temporary occlusion of the LCX. Regional hypokinesia and ischemia were attenuated as a function of time during progressive coronary stenosis. Fifty percent recovery and full recovery of regional shortening during occlusion were observed 19 ± 3 and 25 ± 4 days after instrumentation, respectively, when the endocardial blood flow recovered from 0.42 ± 0.07 ml/min/g at 7 days to 0.56 ± 0.07 and 0.80 ± 0.05 ml/min/g, respectively. Greater than 75% coronary stenosis coincided with collateral development, as estimated from regional shortening rate and the appearance of angiographically opacified collaterals. Our study confirms that the development of collateral vessels reduces regional ischemia and hypokinesia induced during abrupt coronary occlusion in a canine model.

GRADUAL coronary occlusion over a period of days, especially in a canine model, leads to the development of collateral channels, which can be evaluated by measuring peripheral coronary pressure,1 retrograde flow,2 reactive hyperemia3 and regional contraction abnormalities that appear during temporary coronary occlusion.4-6 However, the relationship between the grade of coronary stenosis and the functional reserve of collateral vessels and the relationship between collateral blood flow and regional shortening during progression of collateral development are unclear.

The functional state of collateral channels has been estimated according to the degree of reactive hyperemia4 and by regional myocardial contraction.5-6 Recently, an inverse linear relation between the reduction of reactive hyperemia and recovery of contraction abnormalities during temporary coronary occlusion was noted after the implantation of an ameroid constrito." However, the grade of coronary stenosis is a major determinant of reactive hyperemia.7,8 Therefore, to define the extent to which reactive hyperemia reflects collateral function per se, it may be important to assess both the grade of coronary stenosis and collateral function. In a previous study, we devised a radiolucent ameroid constrictor to determine the grade of coronary stenosis angiographically.13 This device makes it possible to study the relationship between the grade of coronary stenosis as assessed by angiography and the functional state of the collateral vessels.

Several studies have shown that myocardial shortening is related to coronary blood flow.14-17 However, it has not been confirmed whether the relation between collateral flow and regional shortening during collateral development is similar to that in acute reduction of coronary flow.

Accordingly, to better define the relationship between the progression of coronary stenosis and augmentation of collateral functional reserve, as well as the relationship between regional flow and shortening during collateral development, we simultaneously measured myocardial blood flow with tracer microspheres and myocardial shortening with miniature ultrasonic crystals. This was done during a temporary coronary occlusion. Coronary angiography was performed before and after coronary occlusion and at a definite time after ameroid implantation.

Methods

Instrumentation

Twenty-two adult mongrel dogs that weighed 15–23 kg (mean 19 ± 1 kg) were anesthetized with i.v. sodium pentobarbital, 25 mg/kg, and ventilated with a Harvard respirator. An aseptic thoracotomy was performed through the fourth intercostal space. Polyvinyl catheters (2.3 mm o.d.) were inserted into the left atrial cavity through the atrial appendage and into the left ventricular (LV) cavity through the LV apex and were secured with a pursestring suture. A #8F polyvinyl catheter (3.0 mm o.d.) was inserted into the root of the aorta through the left internal thoracic artery.

Two pairs of ultrasonic crystals made of 5-MHz lead zirconate titanate (TDK) (diameter 2 mm) were im-
planted subendocardially into the LV wall; one pair was placed in the area of the left anterior descending coronary artery (LAD) as a control segment length and the other was placed in the center of the left circumflex coronary artery (LCx) area to provide a length of ischemic segment. The presence of ischemia was confirmed by cyanosis and bulging of the segment during acute coronary occlusion. At autopsy, all crystals were firmly embedded in the inner third of the ventricular wall.

The LCx was dissected free near its origin and a 10-MHz Doppler flow probe, a radiolucent ameoid constrictor and a hydraulic cuff occluder were placed around it. This constrictor was constructed in our laboratory and the gradual coronary constriction produced was documented by coronary cineangiography. The pericardium was left open and all wires and tubings were tunneled subcutaneously to the base of the neck. The chest cavity was closed and pentillin-G, 1.0 million units, and streptomycin, 0.3 g, were administered intramuscularly for 4 days after the instrumentation.

**Experimental Measurements**

Aortic, left atrial and LV pressures were measured through the previously implanted catheters attached to calibrated strain-gauge manometers (Statham P-23Db). Coronary blood flow velocity of the LCx was measured with the implanted 10-MHz flow probe. A continuous Doppler flowmeter was used to sample red blood cell velocity. Mean aortic and left atrial pressures and mean coronary blood flow were obtained using a 2-second time constant filter.

Regional segment length was measured by an ultrasonic dimension gauge technique. We could not technically record both regional segment length and a Doppler shift from the LCx simultaneously; therefore, alternate recordings of these variables were performed according to the experimental protocol.

Coronary cineangiograms were obtained by selective injections of 3–5 ml of meglumine diatrizate (Urografin 76) before and during LCx occlusion. Stenosis was expressed as percent stenosis from the width of the LCx in its normal proximal portion compared with that of the ameoid portion. Collaterals were defined as the distinct angiographic visualization of the distal LCx during occlusion. Coronary cineangiography and data processing were performed as reported previously.

Regional myocardial blood flows were measured with isotopically labeled tracer microspheres (3M Co.) 9–10 μ in diameter with γ-emitting radionuclides, 46Sc, 85Sr and 111In. The microspheres were diluted in 10% low-molecular-weight dextran with Tween 80 added. Before each injection, the microspheres were mixed thoroughly by direct application of an ultrasonic probe (UR-150 P, Tomy Seiko) for 5 minutes. During each injection, 4–6 × 10^6 microspheres were delivered into the left atrium and flushed with 8 ml of isotonic warm saline. Ten seconds before microsphere injections, a reference sample of arterial blood was withdrawn from the previously implanted aortic catheter at a constant rate of 6.2 ml/min for 90 seconds.

The radioactive label of the microspheres (85Sr, 111In or 46Sc) was chosen at random. To assure adequate mixing of tracer microspheres in the blood after left atrial injection, bilateral renal cortical flows were measured. Renal cortical flow of the right kidney was 1.81 ± 0.22 ml/g/min and the difference of flow between the right and left kidneys was 0.01 ± 0.08 ml/g/min (NS).

After the study, each dog was anesthetized with i.v. sodium pentobarbital, 25 mg/kg, and given a lethal dose of saturated potassium chloride. The heart was removed and separated into the LV free wall (LVFW), ventricular septum and right ventricle. The LVFW was sectioned into five transverse pieces parallel to the mitral valve ring. Each piece was cut into six blocks, which were each sliced into three layers of equal thickness from the epicardium to the endocardium (fig. 1). Each myocardial specimen was weighed, placed in vials with 10% formaldehyde for counting, and examined histologically after measurement of tissue radioactivity.

Myocardial and blood reference samples were counted in an Autogamma Spectrometer System (Packard model 5130 NS-710) at window settings that corresponded to peak energies of each radionuclide by multichannel analyzer (Northern Inc., NS-700). The raw counts per minute recorded in each energy window were corrected for background activity and overlapping counts contributed by the accompanying isotopes with a digital computer (Nova-01, Data General). Blood flow to each myocardial specimen was computed using the formula Qm = Qr × Cm/Cr, where Qm = myocardial blood flow (ml/min), Qr = reference blood flow (ml/min), Cm = counts per minute of the myocardial specimen and Cr = counts per minute of

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**Figure 1. Instrumentation and tissue sampling procedures.** The left ventricular (LV) free wall was divided into 30 blocks. Shaded areas represent the sites of the ultrasonic crystals. Each block of transmural myocardium was divided into three layers of approximately equal thickness from endocardial (Endo) to epicardial (Epi) surfaces. LCX = left circumflex coronary artery; LAD = left anterior descending coronary artery.
the reference blood flow. The myocardial blood flow to each sample was divided by the sample weight and expressed as ml/min/g of the myocardium. The average sample weight was about 450 mg. When regional myocardial shortening was compared with regional myocardial blood flow, myocardial samples containing both crystals and those between a pair of crystals were averaged.

Experimental Protocols

Serial Measurements of Collateral Function

In 10 of 22 dogs, collateral function was measured serially. Starting about 5 days postoperatively, studies were conducted every 2–4 days for 11–53 days in conscious, unsedated dogs resting quietly on their right side in a dimly lit, quiet room. Basal hemodynamics, including regional segment lengths and coronary blood flow velocity, were measured. Temporary coronary occlusion was then induced by inflating the hydraulic cuff with water; zero flow was confirmed by Doppler flowmeter. Complete coronary occlusion was maintained for 3 minutes while segment lengths were monitored. Release of the cuff occlusion and the resultant reactive hyperemia was confirmed by Doppler flowmeter.

Two dogs died suddenly 18 and 22 days after instrumentation without showing any recovery of regional shortening during temporary coronary occlusion. Therefore, changes in collateral blood flow were evaluated in eight dogs using the tracer microsphere technique. Regional myocardial blood flows were measured at three periods defined according to regional hypokinesia of less than 20%, around 50% and above 90% of control during acute coronary occlusion respectively. In the present experiment, the first, second and third periods were noted 7 ± 1 (range 4–11), 19 ± 3 (8–34) and 25 ± 4 (11–50) days after instrumentation, respectively. After injection of the tracer microspheres, three of the dogs were lightly anesthetized with i.v. sodium pentobarbital, 20 mg/kg, and coronary cineangiography was carried out.

Regional Myocardial Blood Flow Before and During Coronary Occlusion

In 12 dogs instrumented with an ameroid cuff occluder, flow probe and dimension crystals in a manner similar to the dogs described above, regional myocardial blood flow was evaluated before and during coronary occlusion. The seven group A dogs were examined 10–14 days (mean 11 ± 1 days) after instrumentation and the five group B dogs were examined 15–37 days (mean 23 ± 4 days) after instrumentation, when there was no detectable regional reduction of shortening during temporary coronary occlusion. These dogs had received morphine, 10 mg subcutaneously, and the experiment was conducted under sedation. Tracer microspheres were injected in much the same way as in the previous experiment before and 60 seconds after coronary occlusion. At the end of the study, nine of the living dogs were lightly anesthetized with i.v. sodium pentobarbital, 20 mg/kg, and coronary cineangiography was carried out before and during coronary occlusion to document the grade of coronary stenosis and to obtain angiographic evidence of collateral channels.

Data Analysis

Hemodynamic variables were recorded on a multichannel data recorder (Sony BFR 3915) and played back on a direct-writing oscillograph (Nihon Koden Polygraph System). Measured data included mean aortic pressure, mean left atrial pressure, end-diastolic and end-systolic lengths of endocardial segments (EDL and ESL), percent shortening calculated by the formula (EDL – ESL)/EDL x 100. The values for segment lengths were normalized to an initial EDL of 10 mm by dividing the end-diastolic and end-systolic dimensions by the initial control EDL and multiplying by 1020. All data points were the average of at least 10 cardiac cycles.

Reactive hyperemia (%) was calculated as (peak velocity of reactive hyperemia/mean control blood flow velocity) x 100. Blood flow debt repayment was determined as described by Olsson and Gregg.24 Blood flow debt (ml) = control flow rate (ml/sec) x duration of occlusion (sec); reactive hyperemia (ml) = total flow during reactive hyperemia (ml) – control flow rate (ml/sec) x duration of reactive hyperemia (sec); blood flow debt repayment (%) = reactive hyperemic flow (ml)/blood flow debt (ml) x 100.

All values are presented as mean ± SEM. Data were analyzed by t test for paired data (control vs coronary occlusion). The differences between periods during gradual coronary constriction on changes of regional performance after abrupt coronary occlusion were evaluated by analysis of variance. When analysis of variance demonstrated a statistically significant result (p < 0.05), the Newman-Keuls method was used to identify the subgroup differences.25 In comparing EDL and regional shortening during coronary occlusion with those before abrupt coronary occlusion, the Tukey (α) test was used.25 Regression lines and associated correlation coefficients were computed by the least-squares method. The level of statistical significance was p < 0.05.

Results

Serial Changes in Regional Myocardial Function and Coronary Hemodynamics

Representative serial changes in regional myocardial shortening before and during temporary coronary occlusion are presented in figure 2. The EDL of the LAD area increased during coronary occlusion along with a simultaneous increase in percent shortening during systole by 5–8% up to 25 days; thereafter, EDL and percent shortening were unchanged (fig. 2). Percent shortening of the LCX area decreased to around 2% and showed no obvious amelioration for 25 days; thereafter, progressive recovery of regional hypokinesia was noted along with a decreased EDL during occlusion. Table 1 summarizes the time course of collateral development in 10 dogs.
Periods 1, 2 and 3 were recorded at 7 ± 1, 19 ± 3 and 25 ± 4 days, respectively, when regional myocardial shortening during occlusion was 3.1 ± 1.3% (16% of the preocclusive state), 9.1 ± 1.0% (53% of the preocclusive state) and 17.0 ± 1.8% (99% of the preocclusive state), respectively. The average duration from the attenuation of regional myocardial hypokinesia to full recovery (period 3) during a bout of occlusion was 14 ± 3 days (range 5–29 days). Fifty percent recovery (period 2) to full recovery (period 3) was 7 ± 2 days (range 4–16 days).

During 3 minutes of coronary occlusion, a stable severe (period 1) or moderate (period 2) hypokinesia was observed within 1 minute and was stable for 3 succeeding minutes (fig. 3). A summary of hemodynamic variables is shown in table 2.

Figure 4 left is a summary of mean coronary flow velocity before coronary occlusion and at the peak of reactive hyperemia after reperfusion. Resting coronary flow velocity decreased progressively from the first to the third period (p < 0.01) along with a further exaggerated reduction in peak reactive hyperemia (p < 0.05). At the third period, both peak reactive hyperemic and resting flow velocities were low and the same. Flow debt repayment similarly decreased from 1.52 ± 0.26 at the first to 1.06 ± 0.07 and 0.84 ± 0.04 at the second and third periods, respectively (p < 0.01, first vs third and second vs third periods). A summary of regional myocardial shortening before and during coronary occlusion at the LCx area is also presented in figure 4. Regional myocardial shortening before coronary occlusion was maintained fairly constant during the entire experimental period and that during occlusion decreased at the first period and recovered gradually with time. At the third period, regional shortening of the LCx was unchanged before and after coronary occlusion.

Serial Changes in Regional Collateral Flow and Regional Myocardial Function

Figure 5 is a summary of the transmural distribution of myocardial blood flow. One minute after coronary occlusion, regional myocardial blood flow in the endocardial layer in the LCx was reduced to 0.42 ± 0.07 ml/min/g (p < 0.01) and that of the epicardial layer was reduced to 0.72 ± 0.07 ml/min/g (p < 0.01), at the first period as compared with the LAD region. At the second and third periods, regional myocardial blood flow to the endo- and epicardial layers increased progressively. Regional myocardial blood flow in the area supplied by the LCx was consistently lower than that supplied by the LAD (p < 0.05).

In dogs of group A, in which the study was conduct-
ed about 11 days after instrumentation, regional myocardial blood flow at the LCx endocardial region decreased strikingly, from 1.21 ± 0.28 ml/min/g (Ac in fig. 6) before occlusion to 0.28 ± 0.07 ml/min/g (p < 0.01) during the LCx occlusion (Ao in fig. 6), while regional myocardial blood flow at the LAD endocardial region tended to increase from 1.07 ± 0.31 ml/min/g before coronary occlusion to 1.12 ± 0.20 ml/min/g during coronary occlusion (NS). In group B, in which the study was conducted about the twenty-third day of the same instrumentation, regional myocardial blood flow in the LAD or LCx area was unchanged before and during coronary occlusion (Bc vs Bo in fig. 6).

As demonstrated in figure 6, the correlation between regional myocardial shortening and regional myocardial flow at the same site of shortening measurement during coronary occlusion is fairly linear at regional blood flows of 0.3–1.0 ml/min/g (y = 31x − 9, r = 0.99). A similar linear relation was noted between regional myocardial shortening at the endocardial site and mean transmural blood flow at the LCx area (y = 34x − 14, r = 0.99). The myocardium in which the collateral vessels developed did not exhibit significant fibrosis, although there was some fibrosis in the core of posterior papillary muscle.

**Relationship between Angiographic Coronary Stenosis and Collateral Function**

Coronary cineangiography was performed before and during temporary coronary occlusion. With the development of collaterals, the LCx distal to the occlusion was opacified from the intact LAD. Collateral channels were noted angiographically when the LCx stenosis exceeded 75%, and the resting and peak reactive hyperemic flow dropped markedly in proportion to the progression of coronary stenosis (fig. 7).

**Discussion**

In the present study, regional myocardial blood flow and segmental myocardial shortening during acute coronary occlusion were determined repeatedly in a canine model of gradual coronary stenosis. During induced progressive stenosis of the LCx, collateral development was investigated in terms of functional reserve, i.e., reactive hyperemia, regional collateral flow and regional segmental shortening, and the grade of coronary stenosis and the opacification of distal LCx through the collateral channel were documented by cineangiography.

Previous studies were concerned with the role of collateral vessels in peripheral coronary pressure, 1 retrograde flow 2 and regional myocardial blood flow es-

**Figure 3. Changes in end-diastolic segment length (EDL) and percent shortening (%ΔL) during 3 minutes of coronary occlusion at the three periods. LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery. *p < 0.05, **p < 0.01 vs control (before coronary occlusion).**

**Table 2. Hemodynamics Before and During Coronary Occlusion**

<table>
<thead>
<tr>
<th>Time after ameroid implantation (days)</th>
<th>Heart rate (beats/min) Before</th>
<th>Heart rate (beats/min) During</th>
<th>SBP (mm Hg) Before</th>
<th>SBP (mm Hg) During</th>
<th>LVEDP or mLAP (mm Hg) Before</th>
<th>LVEDP or mLAP (mm Hg) During</th>
<th>Mean AoP (mm Hg) Before</th>
<th>Mean AoP (mm Hg) During</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>11 ± 1</td>
<td>95 ± 4</td>
<td>121 ± 9*</td>
<td>122 ± 8</td>
<td>112 ± 8</td>
<td>8.1 ± 0.9</td>
<td>13.4 ± 2.2*</td>
<td>95 ± 5</td>
</tr>
<tr>
<td>Period 1</td>
<td>7 ± 1</td>
<td>108 ± 3</td>
<td>124 ± 12</td>
<td>114 ± 2</td>
<td>114 ± 4</td>
<td>6.5 ± 0.6</td>
<td>14.0 ± 2.0*</td>
<td>91 ± 4</td>
</tr>
<tr>
<td>Period 2</td>
<td>19 ± 3</td>
<td>110 ± 7</td>
<td>120 ± 8</td>
<td>110 ± 2</td>
<td>115 ± 7</td>
<td>7.5 ± 1.5</td>
<td>10.6 ± 1.4*</td>
<td>89 ± 2</td>
</tr>
<tr>
<td>Period 3</td>
<td>25 ± 4</td>
<td>115 ± 11</td>
<td>117 ± 16</td>
<td>109 ± 9</td>
<td>112 ± 8</td>
<td>5.8 ± 1.5</td>
<td>6.4 ± 1.7</td>
<td>92 ± 7</td>
</tr>
<tr>
<td>Group B</td>
<td>23 ± 4</td>
<td>87 ± 3</td>
<td>87 ± 5</td>
<td>126 ± 4</td>
<td>125 ± 5</td>
<td>10.7 ± 1.8</td>
<td>11.1 ± 2.3</td>
<td>103 ± 6</td>
</tr>
</tbody>
</table>

Values are mean ± SEM.

*p < 0.05.

Abbreviations: SBP = peak systolic pressure; LVEDP = left ventricular end-diastolic pressure; mLAP = mean left atrial pressure; AoP = aortic pressure.
timed by $^{86}$Rb uptake or tracer microspheres. However, the question of whether collateral flow immediately available after coronary artery occlusion has any protective effect might not be answered using these methods. Recently, it has been accepted that regional myocardial shortening during abrupt coronary occlusion or coronary stenosis provides direct and quantitative information concerning the functional derangement in cases of myocardial ischemia.

Thus, regional myocardial shortening during temporary coronary occlusion is a qualitative index of nutritional supply by collateral blood flow. Using the same ultrasonic dimension gauge technique, we confirmed that the collaterals that developed during gradual coronary stenosis progressively attenuated both the grade of regional hypokinesia of the LCx area and hyperfunction of the LAD area induced by an abrupt coronary occlusion. The reasons for slight hyperfunction in normal regions during coronary occlusion have been attributed to the Frank-Starling mechanism and to the reduced net afterload on the normal segment. These beneficial effects of collaterals on myocardial function are in accord with findings in experimental and clinical studies.

In addition, we correlated regional shortening to regional blood flow and grade of coronary stenosis during chronic coronary stenosis. We found that in the conscious dog with an ameroid constrictor, regional myocardial shortening at the endocardium changed linearly with respect to regional flow. Vatner measured both endocardial flow and endocardial segment length during partial coronary stenosis and found a

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**Figure 4.** Coronary flow velocity (left) and regional myocardial performance (right) during progression of collateral development in the three periods. (Left) Mean flow velocity before coronary occlusion (CO) and during peak reactive hyperemia (RH). (Right) Regional shortening in the left circumflex coronary artery (LCX) at rest and during coronary occlusion. *p < 0.05, **p < 0.01 vs control (before coronary occlusion).

**Figure 5.** Transmural distribution of regional myocardial blood flow during temporary coronary occlusion as a function of the period. LAD = left anterior descending coronary arterial area as a control; LCX = left circumflex coronary arterial area as an ischemic site; CO = coronary occlusion.
nonlinear relationship between reductions in regional flow and hypokinesia. However, myocardial segments grouped according to percent decreases in flow show direct linear decreases in regional myocardial function. Studies of segmental work and wall thickness provided data in accord with our present observations. Thus, the dependency of regional myocardial performance on regional flow was confirmed in the case of chronic increases of collateral flow as it was in cases of acute reduction of antegrade coronary flow.

Collateral flow measurement at the LCx area during coronary occlusion inevitably involves serious artifacts due to inclusion of a small amount of normally perfused myocardium in the sample. In the present study, normally perfused flow at the LAD area remained unchanged during progressive coronary stenosis. Therefore, even if the LCx myocardial sample included a small amount of normal myocardium, changes in the measured regional blood flow of the LCx endocardium would still represent incremental flow through the collateral channels.

We calculated the endocardial flow of the LCx area in which regional shortening was measured. The amount of myocardium was relatively large (about 1.0 g). Accordingly, the finding that the flow value at the first period was relatively high for a dog in which collateral vessels were not present may be explained if there was contamination of normally perfused myocardium in the LCx sample.

We observed a time delay (for example, 25 days in figure 2) in the day-to-day recovery of regional dysfunction at rest during temporary coronary occlusion. In the angiographic study, collateral channels were visible when the LCx stenosis exceeded 75% (fig. 7). Thus, there may be a delay between the time the LCx stenosis exceeds 75% and when the inherent collaterals have been functionally stimulated by the resultant pressure gradient or the hypoxic gradient. The time between the initial recovery of regional dysfunction and full recovery at rest was 14 ± 3 days, and the time course of this functional recovery was fairly constant. However, the grade of collateral development at 25 days may not be sufficient to maintain normal regional function during strenuous running. Kumada et al. noted that exercise-induced myocardial dysfunction remained even after postspacing myocardial dysfunction, a sensitive index for detecting limited coronary flow, was eliminated by further collateral development. The time dependency of collateral function in our study is in good accord with serial changes of reactive hyperemia as well as with serial changes in collateral resistance after amiodaroid implantation.

However, because coronary stenosis affects the extent of reactive hyperemia, it might be reasonable to evaluate whether collateral flow or coronary stenosis determines the grade of reactive hyperemia. In the present study, peak reactive hyperemia decreased progressively from the first to the third period with a marked reduction in flow debt repayment. These find-
ings may represent a concomitant progression of coronary stenosis and collateral development to overcome the myocardial ischemia. Reactive hyperemia was virtually absent above 75% coronary stenosis, and a similar finding has been described in acute studies. Thus, the relationship between reactive hyperemia and the grade of stenosis can be determined on the same physical basis in acute and chronic stenosis. It is also necessary to further evaluate the interdependence of the grade of peak reactive hyperemia and flow debt repayment. We propose that the reduction in regional shortening during ischemic intervention be considered when attempting to evaluate growth patterns of collateral vessels.

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