ESTIMATION OF CORONARY RESERVE IN LEFT ANTERIOR DESCENDING AND CIRCUMFLEX CORONARY ARTERIES BY REGIONAL THERMODILUTION TECHNIQUE

Akira Kurita, M.D.*, Jacques Azorin, M.D., Andre Granier, M.S.
and Martial G. Bourassa, M.D.

The present study was attempted to determine whether a reduction in regional venous maximal coronary flow can indicate the presence of significant coronary stenosis. The great cardiac vein flow and the coronary sinus outflow were measured simultaneously in 8 open-chest dogs by a continuous thermodilution technique using a triple thermister catheter or two separate thermister catheters. The left anterior descending and circumflex coronary inflows were recorded using electromagnetic flow probes. Successive 70% and 90% stenoses of the coronary artery were produced using a calibrated constrictor. Maximal coronary flow was obtained during reactive hyperemic response after coronary occlusion for 8 sec. Coronary flow reserve was expressed as the ratio of maximal coronary flow to resting one. During 70% coronary arterial stenosis maximal coronary flow and coronary reserve decreased significantly in the great cardiac vein and the coronary sinus. Significant correlations were found between the flows in the left anterior descending artery and in the great cardiac vein (r = 0.81) and between those in the circumflex artery and in the coronary sinus minus the great cardiac vein (r = 0.79) throughout the periods of preocclusion, occlusion and reactive hyperemic response. There were no significant changes in heart rate and hemodynamics. Using continuous thermodilution techniques, the inflows of the left anterior descending and the circumflex coronary arteries at a stenosis greater than 70% could be estimated from the changes in regional venous outflows.

Coronary arteriography is a widely used diagnostic method that provides excellent visualization of lesions in the larger coronary vessels and is an essential prerequisite for coronary bypass surgery. However, the interpretation of moderate lesions may be difficult and is subject to a rather wide inter-observer variability. Moreover, the potential clinical significance of these lesions may be difficult to define in the individual patient. Resting coronary blood flow is not altered until there occurs at least a 70% coronary stenosis and then even small increments in obstruction that may not be detectable by angiography result in marked changes in coronary flow.1,2 On the other hand, coronary vascular reserve can be compromised by much less significant stenosis. During the past decade the need to estimate regional myocardial perfusion

Key Words:
Coronary reserve
Regional thermodilution
Reactive hyperemia

(Received September 19, 1981; accepted March 30, 1982)
Department of Medicine, Montreal Heart Institute, University of Montreal, Montreal, Quebec, HIT 1C8, Canada
Supported in part by the Jean-Louis Lebesque Foundation.
*Present address and address for correspondence: Akira Kurita, M.D., National Defense Medical College, Saitama 359, Japan

964 Japanese Circulation Journal Vol. 46, September 1982
Coronary Reserve by Thermodilution Technique

Fig. 1. Method of producing reactive hyperemic response by #4 Ethicon silk and that inducing coronary artery stenosis by a constrictor. Coronary artery inflow was measured by an appropriately sized electromagnetic flow probe. EMF = electromagnetic flow probe

in addition to performing coronary arteriography has been fully recognized. Several quantitative techniques have been proposed and tested in experimental animals and in man. Myocardial imaging techniques using diffusible isotopes or radioactive microspheres with selective precordial sampling have been promising. However, these methods are limited by technical and economic considerations. A more simple, inexpensive and reproducible method is the use of selective venous sampling of He washout curves or continuous thermodilution curves.

The present study was attempted to validate the use of continuous regional thermodilution sampling for the assessment of regional myocardial perfusion. The usefulness of the method is tested at rest and during the stress of reactive hyperemia in the presence of significant and critical stenosis of the left anterior descending (LAD) and circumflex coronary arteries (LCx). This should provide relevant information concerning the applicability of the method to patients with obstructive disease of the left coronary system during coronary arteriography.

METHODS

Eight mongrel dogs (29 to 30 kg) were used. Anesthesia was induced with sodium pentobarbital, an initial dose of 25–30 mg per kg intravenously, and then an additional 60 mg as needed during the experiment. Respiration was controlled with a Harvard ventilator through a cuffed endotracheal tube. During positive pressure ventilation, the chest was opened through a left thoracotomy at the fourth intercostal space. The pericardium was opened and the LAD and the LCx arteries were carefully isolated. The external diameters of the proximal LAD and the LCx arteries were measured by a graduated caliper. An appropriately sized electromagnetic flow probe (North Carolina Medical Electronics, Model 501) was carefully placed on small distal to the septal branch of the LAD and the proximal portion of the LCx. Then a constrictor was placed distal to the flow probe. A No 4 Ethicon silk thread mounted on a snare was placed around the most proximal portion of the artery (Fig. 1).

Small branches between the constrictor and the flow probe were ligated. A No 5 specially designed Courand catheter having one internal and two external Wilton Webster's microtip thermisters was advanced to the great cardiac vein (GCV) through the coronary sinus (CS) under fluoroscopy (Fig. 2). When difficulties in positioning were encountered, the catheter was
guided manually from outside the pericardium. The position of the catheter was recognized by the injection of a small amount of contrast material which opacified the anterior interventricular vein and GCV without any restriction in flow. The tip of the catheter was positioned approximately one cm below the origin of the anterior interventricular vein. The second external thermistor was placed inside the ostium of the CS. The catheter was repositioned as needed during the experiment. All measurements were recorded on a Hewlett-Packard multichannel photographic recorder (Model 8890B) at paper speeds varying from 2.5 to 25 mm per second.

**Experimental Procedure**

GCV and CS flow were measured simultaneously using two Wheatstone bridges. Both flows were calculated by the following usual formula:

\[ \text{CSB} = F_1 \times \frac{(T_B - T_I)}{(T_B - T_M)} - 1 \times 1.08 \]

where \( F_1 \) represents the volume of the injectate (ml/min); \( T_B, T_I \) and \( T_M \) are the temperatures of blood, injectate and the mixture in CS, respectively; and 1.08 is a constant derived from the density and specific heat of the saline solution and blood. Amplifier gain was adjusted to provide approximately one cm recorder deflection for 1000 \( \Omega \) in the internal thermistor and 100 \( \Omega \) in the external thermistors. A normal physiologic saline solution at 22 to 25°C was infused into GCV with a calibrated Harvard infusion pump using 30 ml twin syringes at a rate of 30 ml/min. The changes of temperature during infusion of a normal physiologic saline were recorded by the external thermistor as an indicator and venous outflow was calculated by using these deflections as usual. All dogs were heparinized intravenously. Baseline resting flow was first recorded. Then the LAD or the LCx artery was occluded for a period of 8 sec by constriction of #4 Ethicon silk (Fig. 1). The reactive hyperemic response was recorded after releasing the occlusion. LAD or LCx artery inflow was measured with the electromagnetic flow probe and GCV and CS outflows were measured by continuous thermodilution using the external catheter thermistors. Aortic pressure obtained through an aortic catheter by a Statham P23Db pressure transducer were also recorded before occlusion, during occlusion and during reactive hyperemia. After stabilization of the physiologic parameters, a constrictor was gradually tightened to produce a 70% stenosis and all measurements were repeated. Then a critical 90% stenosis was induced and all measurements were again repeated.

The constrictor had 12 marks (Fig. 1) and the percent diameter of constricted artery was measured as follows: when the constrictor touched the surface of the artery, its value was read as 0 (contact). Then the number of rotations which resulted in complete occlusion of the artery was counted (occlusion). The number of rotations required to induce 70% or 90% stenosis could then be calculated as follows: the number of rotations to complete occlusion \( \times \) the degree of desired % stenosis/100.

In this experiment, general hemodynamics might be influenced by 90% stenosis because of a reduction of coronary blood flow. Therefore 90% stenosis obtained by this constrictor may be slightly different from that observed clinically.

The dogs were sacrificed at the end of the procedure. A Schlesinger's solution was injected into the coronary arteries. The heart was fixed in 10% formalin for 12 hours. Sections of stenosed and normal areas of the coronary arteries were obtained and planimetered after enlargement. A correlation coefficient and a linear regression equation between the anatomic results and the calculated degrees of stenosis were obtained.

The peak reactive hyperemic response was expressed by the ratio of hyperemic flow/basal flow in ml/min. Coronary vascular resistance was calculated by the ratio of mean aortic pressure/mean coronary arterial blood flow in units.

A Student's paired \( t \)-test was used for statistical analysis of the data. All data were analyzed with a Hewlett-Packard Model 9830A digitalizer computer.

**RESULTS**

Typical examples of records of coronary artery inflow and venous outflow under basal conditions and following a 90% coronary stenosis were illustrated in Figs. 3 to 6. Figure 3 represents a LAD occlusion and Fig. 5 a LCx occlusion. As shown in Fig. 3, absolute values for coronary inflow and venous outflow during reactive hyperemia under basal conditions were different: LAD flow increased from 45 to 117 ml/min, while GCV flow increased from 32 to 55 ml/min. However, both changes were parallel temporarily. Figure 4 shows a typical response during critical LAD stenosis: both coronary inflow and venous outflow increased only slight-
Fig.3. Occlusion of LAD for 8 sec without a pretreatment of coronary stenosis. GCV outflow (upper line) decreased from 32 to 14 ml/min following occlusion of LAD and increased to 55 ml/min during RHR. CS outflow (middle line) changed parallel with GCV outflow; decreased from 61 to 43 ml/min following occlusion of LAD and increased to 91 ml/min during RHR. LAD inflow (bottom line) decreased from 45 to 10.75 ml/min following occlusion and increased to 117 ml/min during RHR. In this figure, the deflection of the thermodilution curves (upper and middle lines) represent the decrease in flow, and the polarities of electromagnetic flow record (bottom line) and thermodilution curves are reversed. (These relations are the same in Fig. 3 to 6).

LAD = left anterior descending artery,
GCV = great cardiac vein,
CS = coronary sinus,
RHR = reactive hyperemic response

Fig.4. Occlusion of LAD following 90% coronary artery stenosis (the same dog as in Fig. 2). GCV outflow decreased from 27 to 13 ml/min following occlusion of LAD and increased to 34 ml/min during RHR. CS outflow decreased from 58 to 48 ml/min following occlusion and increased to 61 ml/min. LAD inflow decreased from 33 to zero ml/min following occlusion and increased to 48 ml/min during RHR. Abbreviations are the same as Fig. 3.

Fig.5. Occlusion of LCx for 8 sec without a pretreatment of coronary stenosis. GCV outflow (25 ml/min) was not influenced by the occlusion. CS outflow decreased from 62 to 37 ml/min following occlusion and increased to 79 ml/min during RHR. Therefore, there was a decrease in (CS-GCV) outflow from 39 to 12 ml/min following occlusion of LCx and an increase to 54 ml/min during RHR. LCx inflow decreased from 50 to zero ml/min following occlusion and increased to 140 ml/min during RHR. Other abbreviations are the same as Fig. 3. LCx = left circumflex coronary artery.

Occlusion of the Left Anterior Descending Artery
The relationships between LAD inflow measured by electromagnetic flow probe and GCV outflow measured by thermodilution technique were determined in 72 different series in 8 animals at 0, 70 and 90% of coronary stenosis,
Fig. 6. Occlusion of LCx following 90% coronary artery stenosis (the same dog as in Fig. 4). GCV outflow (24 ml/min) was not influenced by occlusion. There was a decrease in (CS-GCV) outflow from 48 to 32 ml/min during occlusion and an increase to 41 ml/min during RHR. LCx inflow decreased from 42 ml/min to zero during occlusion and increased to 39 ml/min during RHR.

respectively.

Figure 7 illustrates the relationship between LAD inflow and GCV outflow under basal conditions, during occlusion and hyperemic response at different degrees of stenosis. As shown in Fig. 7, there was still 17 ± 7 (mean ± SD) ml/min of venous outflow during complete occlusion, when LAD inflow showed no flow at all.

Table I shows LAD inflow and GCV outflow under basal conditions, their peak flow during reactive hyperemia and the ratio of hyperemic response in 8 animals. Under basal conditions, both LAD inflow and GCV outflow did not change significantly until 90% stenosis, as shown in this table. Of interest is the fact that both peak flow and hyperemic response significantly decreased even at 70% stenosis. LAD inflow decreased from 94 ± 32 to 59 ± 18 ml/min (p < 0.01), and GCV outflow decreased from 45 ± 11 to 33 ± 11 ml/min (p < 0.01). Values of hyperemic response of LAD inflow decreased from 2.9 ± 0.8 to 1.9 ± 0.3 (p < 0.01) and that of GCV outflow from 1.6 ± 0.3 to 1.2 ± 0.3 (p < 0.05).

Table II illustrates the changes in coronary vascular resistance in 7 experiments. Under basal condition, resistance to both LAD inflow and GCV outflow were slightly increased at 70% and 90% stenosis, but these changes were not statistically significant. During hyperemia, however, both inflow and outflow increased significantly even at 70% stenosis. Heart rate and blood pressure were not changed significantly at 70% and 90% stenosis.

Comparison of GCV outflow by thermodilution and LAD inflow by E M flow probe

$$r = 0.82 (p < 0.001)$$
$$y = 0.26x + 16.96$$

Fig. 7. Comparison of LAD inflow by electromagnetic flow probe and GCV outflow by the thermodilution method before and during occlusion and during reactive hyperemic response at 0, 70 and 90% coronary stenosis.
TABLE I  EFFECTS OF THE LEFT ANTERIOR DESCENDING ARTERY OCCLUSION FOR 8 SECONDS ON GREAT CARDIAC VEIN OUTFLOW AND THE LEFT ANTERIOR DESCENDING ARTERY INFLOW

<table>
<thead>
<tr>
<th></th>
<th>Basal flow</th>
<th>Peak flow during reactive hyperemia</th>
<th>Ratio of hyperemic response</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GCV outflow (ml/min)</td>
<td>LAD inflow (ml/min)</td>
<td>GCV outflow (ml/min)</td>
</tr>
<tr>
<td>No. of experiments</td>
<td>8</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Control</td>
<td>28 ± 4</td>
<td>32 ± 8</td>
<td>45 ± 11</td>
</tr>
<tr>
<td>70% stenosis</td>
<td>28 ± 8</td>
<td>30 ± 8</td>
<td>33 ± 11</td>
</tr>
<tr>
<td>90% stenosis</td>
<td>25 ± 7</td>
<td>26 ± 6</td>
<td>24 ± 9**</td>
</tr>
</tbody>
</table>

Values are shown as mean ± SD. GCV = great cardiac vein; LAD = left anterior descending artery; hyperemic response = ratio of hyperemic flow to basal flow. * = p < 0.05, ** = p < 0.01, *** = p < 0.001, as compared with the controls.

TABLE II  CHANGES OF CORONARY VASCULAR RESISTANCE [mmHg/ml/min] IN THE GREAT CARDIAC VEIN AND THE LEFT ANTERIOR DESCENDING ARTERY BEFORE AND 8 SECONDS OF OCCLUSION

<table>
<thead>
<tr>
<th></th>
<th>Basal resistance</th>
<th>Resistance during reactive hyperemia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GCV (mmHg/ml/min)</td>
<td>LAD (mmHg/ml/min)</td>
</tr>
<tr>
<td>No. of experiments</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Control</td>
<td>3.7 ± 0.7</td>
<td>3.4 ± 0.7</td>
</tr>
<tr>
<td>70% stenosis</td>
<td>3.9 ± 1.3</td>
<td>3.7 ± 0.7</td>
</tr>
<tr>
<td>90% stenosis</td>
<td>4.5 ± 1.3</td>
<td>4.3 ± 0.9</td>
</tr>
</tbody>
</table>

For symbols and abbreviations, see Table I.

Oclusion of the Left Circumflex Coronary Artery

This study was also carried out by the same protocol as that in the LAD occlusion.

In the dog heart, the right coronary artery does not notably contribute to coronary sinus flow and proximal CS flow mainly comes from both the LAD and the LCx. Therefore, as explained in the above section, we can consider LCx inflow equal to a difference between CS and GCV outflows [(CS–GCV) outflow].

The relationship between LCx inflow measured by electromagnetic flow probe and (CS–GCV) outflow measured by thermodilution technique was also studied in 72 different series in 8 animals (Fig. 8). As shown in Fig. 8, although there was still some venous pooling flow during complete occlusion of the LCx (19 ± 13 ml/min), a close correlation was also obtained similarly as that in the LAD occlusion.

Japanese Circulation Journal Vol. 46, September 1982

Table III illustrates basal flow, peak flow and the ratio of hyperemic response at 0, 70 and 90% coronary stenosis. Results analogous to those in the LAD occlusion were obtained throughout this study. Under basal conditions LCx inflow showed a statistically significant decrease at 90% stenosis (50 ± 9 to 42 ± 9 ml/min, p < 0.05), while (CS–GCV) outflow decreased only slightly (45 ± 17 to 42 ± 18 ml/min, NS). Peak flow and ratio of hyperemic response were changed significantly even at 70% of stenosis in both LCx inflow and (CS–GCV) outflow. The ratio of hyperemic response in LCx inflow decreased from 2.4 ± 0.3 to 2.5 ± 0.5 mmHg/ml/min (p < 0.05) and that in (CS–GCV) outflow decreased from 1.8 ± 0.3 to 1.5 ± 0.2 mmHg/ml/min (p < 0.01).

Table IV illustrates changes of coronary vascular resistance at different degrees of stenosis.
TABLE III  EFFECTS OF THE LEFT CIRCUMFLEX ARTERY OCCLUSION FOR 8 SECONDS ON (CS-GCV) VEIN OUTFLOW AND LEFT CIRCUMFLEX ARTERY INFLOW

<table>
<thead>
<tr>
<th></th>
<th>Basal flow</th>
<th>Peak flow during reactive hyperemia</th>
<th>Ratio of hyperemic response</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CS-GCV outflow (ml/min)</td>
<td>LCx inflow (ml/min)</td>
<td>CS-GCV outflow (ml/min)</td>
</tr>
<tr>
<td>No. of experiments</td>
<td>8</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Control</td>
<td>45 ± 17</td>
<td>50 ± 9</td>
<td>80 ± 32</td>
</tr>
<tr>
<td>70% stenosis</td>
<td>42 ± 19</td>
<td>46 ± 7</td>
<td>64 ± 23</td>
</tr>
<tr>
<td>90% stenosis</td>
<td>42 ± 18</td>
<td>42 ± 9</td>
<td>34 ± 15</td>
</tr>
</tbody>
</table>

Values are shown as mean ± SD. LCx = left circumflex artery. Other abbreviations are the same as in Table II.

TABLE IV  CHANGES OF CORONARY VASCULAR RESISTANCE (mmHg/ml/min) IN THE CS-GCV AND THE LEFT CIRCUMFLEX ARTERY BEFORE AND 8 SECONDS OF OCCLUSION

<table>
<thead>
<tr>
<th></th>
<th>Basal</th>
<th>Hyperemic response</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CS-GCV</td>
<td>LCx</td>
</tr>
<tr>
<td>No. of experiments</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Control</td>
<td>2.2 ± 1.0</td>
<td>2.1 ± 0.4</td>
</tr>
<tr>
<td>70% stenosis</td>
<td>2.5 ± 1.5</td>
<td>2.3 ± 0.5</td>
</tr>
<tr>
<td>90% stenosis</td>
<td>2.6 ± 1.4</td>
<td>2.6 ± 0.8</td>
</tr>
</tbody>
</table>

For symbols and abbreviations, see Table III.
Coronary Reserve by Thermodilution Technique

Under basal conditions, both the LCx inflow and the (CS–GCV) outflow resistances increased, but these changes were not statistically significant. During reactive hyperemia, coronary vascular resistance in the LCx increased from 0.7 ± 0.1 to 1.0 ± 0.30 at 75% (NS) and to 2.9 ± 1.8 mmHg/ml/min (p < 0.05) at 90% stenosis, and the (CS–GCV) outflow from 1.2 ± 0.5 to 1.6 ± 0.6 mmHg/ml/min (p < 0.05) at 70% of coronary stenosis.

Heart rate and blood pressure were not changed significantly throughout these procedures.

DISCUSSION

Recent visual techniques of coronary arteriography and left ventriculography have greatly contributed to the management of patients with ischemic heart disease and provided important prognostic information especially after coronary bypass surgery. Coronary blood flow measurements should theoretically give more relevant clinical information than an arteriogram, since an arteriographically significant lesion does not necessarily result in reduction in the flow when a decrease in arteriolar resistance can compensate for the effect of proximal stenosis. Therefore, the degree of stenosis and changes in flow do not show a close linear relationship, and the resting coronary blood flow and the regional flow distribution are little affected up to a critical level of stenosis. Recently radioactive microsphere techniques have been applied to this field and several investigators have measured regional myocardial blood flows in experimental animals as well as in humans. The method using 133Xe injected into different regions of the heart muscle at the time of thoracotomy has a limited clinical application, because this technique requires direct intramyocardial injections and also has the possibility of inducing local hyperemia following needle injection. Measurement of regional myocardial 133Xe washout with a scintillation camera was extensively carried out by Cannon et al. Although this may be a quite reliable method for quantitative measurements and can be performed at the time of coronary arteriography, this technique has the limitations in its use because of the high cost of the equipment and the isotope, as well as some methodological problems.

Nakazawa et al. estimated regional myocardial blood flow, using He by measuring myocardial washout courses simultaneously from the great cardiac vein (LAD area) and the coronary sinus (LAD and LCx area). Their results in humans suggest that 90–100% stenosis on arteriography are commonly associated with a reduction of local flow per unit weight even at rest, whereas stenosis of less than 90% is associated with either normal, intermediate or abnormally low resting flow. However, correlation with the values obtained by other techniques such as radioactive microspheres, electromagnetic flow probe or direct measurement was not analyzed.

In the human heart 90% of the small veins arising from left ventricular and intraventricular septal myocardium drain into the coronary sinus. Therefore, the changes of the coronary sinus blood flow might be considered to reflect those of the left ventricular efflux in human studies. For this reason, coronary sinus flow measurements have been always used in our institute. The results of the present study indicate that by the measurement of peak coronary inflow or outflow during hyperemia the significant changes of coronary reserve and coronary vascular resistance could be detected clearly at a level of 70% stenosis. On the other hand, coronary inflow measurement by an electromagnetic flow probe under basal conditions could estimate their changes only at a level of 90% stenosis, but coronary outflow measurement by thermodilution could not.

Bassan et al. showed that by an injection of a contrast material into the left coronary artery the flow rose by 70 ± 27% in the normal subjects and by 46 ± 25% in patients with coronary disease, while resistance fell by 44 ± 9% and 33 ± 11%, respectively, and they concluded that these changes were not sufficient to assess the degree of impairment in the coronary arterial bed in an individual patient. Miller et al. could separate diseased coronary artery completely from a normal one by injecting a 7 ml of contrast medium into the left coronary artery. In the present experiment, we used a temporary occlusion method to get peak reactive hyperemia, because peak hyperemic response can easily be estimated and its rate can reach a higher level than that by a Hypaque injection (4.3 ± 0.9 after occlusion and 3.9 ± 0.9 after Hypaque). Our results suggest that the response of the venous outflow reflects accurately arterial inflow, except in the case of complete occlusion (Figs. 7 and 8). Venous outflow of a few ml observed in this situation is probably due to a functional overlap of the collateral vessels between the adjacent coronary arteries or to a shunt between the
coronary arteries.  

Our data demonstrated that the increase in venous outflow was less than that in coronary inflow during reactive hyperemia, and the response of the arterial side may be different from that of the venous one. Reactive hyperemic flow should be calculated as described by Coffmann and Gregg using the following formula: reactive hyperemic flow = (integral of flow curve during reactive hyperemia) – (flow debt), where flow debt = (control flow rate) x (duration of occlusion), but we estimated the functional capacity of the coronary artery by measuring only the peak phasic flow during hyperemia. Since there are no similar experiments to those described here, the present results must await confirmation by further experimentation. In the present experiments the position of the thermistor catheter in the coronary venous system must be checked most carefully. A similar disadvantage of thermodilution technique has also been recognized in the He washout method because the positioning of the catheter opening is critical in order to avoid the blood admixture from other coronary arteries into the venous system.

Although there must be some approximation in the assessment of a selective physiological function using the thermodilution technique, our method is employed in ordinary catheterization studies.

Acknowledgement

We are grateful to Dr. William Ganz, Cedars Sinai Medical Center, Los Angeles, for critical review of the manuscript and illustrations.

REFERENCES

24. NAKAZAWA H, OLIVEROS RM, ORLICK AE, KLOCHE FJ: Evaluation of regional variations in
Coronary Reserve by Thermodilution Technique

coronary flow by simultaneous great cardial vein
and coronary sinus sampling (abstr). Circulation
49, 50 (Suppl III): 140, 1974
25. KLOCKE FJ: Quantitative evaluation of coronary
perfusion in man. Cath Cardiovasc Diag 1: 349,
1975
26. HOOD WB: Regional venous drainage of the
27. DRISCOL TE, ECKSTEIN RW: Coronary inflow
and outflow responses to coronary artery occlusion.

28. PRINZMETAL N, SIMKIN B, BERGMAN HC,
KRUGER HE: Studies on the coronary circulation
(II): Collateral circulation of the normal human
heart by coronary perfusion with radioactive
erthrocytes and glass spheres. Am Heart J 33:
420, 1947
29. COFFMANN JD, GREGG DE: Reactive hyperemia
characteristics of the myocardium. Am J Physiol
199: 1143, 1960