Flow into Ischemic Myocardium and across Coronary Collateral Vessels is Modulated by a Waterfall Mechanism

Calvin Eng and Edward S. Kirk
From the Cardiovascular Center, Division of Cardiology, Albert Einstein College of Medicine, Bronx, New York

SUMMARY. If a coronary artery is ligated and the distal end cannulated, blood flows retrograde from the cannula when vented to the atmosphere. By varying the height of the outflow tubing, and thereby changing the outflow pressure, pressure-flow relationships can be constructed. We used this technique in eight dogs to assess the characteristics of blood flow into ischemic myocardium. Above a back pressure of 10 mm Hg, increasing back pressure resulted in a decrease of retrograde blood flow. However, below a back pressure of about 10 mm Hg (10.7 ± 2.7 mm Hg), alterations in back pressure did not result in changes in retrograde blood flow (back pressure-independent region). The transition at 10 mm Hg is interpreted as the critical waterfall pressure in ischemic myocardium. In another group of eight dogs, the ischemic bed was completely embolized with 25-μm sized microspheres to prevent RBF from entering the tissue as back pressure was raised. Pressure-flow relationships performed in this group revealed a back pressure-independent region that extended to approximately 20 mm Hg (23.0 ± 2.5 mm Hg). This behavior of the pressure-flow relationship is consistent with a waterfall phenomenon on the collateral vessels. To the extent that collateral vessels in the dog are mainly epicardial in location, the findings suggest that extravascular pressures of 20 mm Hg can occur in the more superficial layers of the heart. In addition, the waterfall on the collaterals indicates that this mechanism can operate on nonvenous vessels. Our results suggest separate waterfall phenomena operating on the collateral vessels (20 mm Hg) and on the vessels in the ischemic myocardium (10 mm Hg). (Circ Res 55:10-17, 1984)

THERE has been considerable recent interest in the "back pressure" as a major determinant of blood flow in the heart. This back pressure is felt to be due to the intramyocardial external forces exerted on the coronary vasculature during cardiac contraction tending to compress and collapse the vessels. Permutt et al. (1962) described the flow through a collapsible vessel surrounded by an external pressure and likened the flow behavior to that of a waterfall. Downey and Kirk (1975) demonstrated that systole inhibited coronary blood flow by a waterfall mechanism. Although the back pressure to coronary flow should be a consideration during physiological conditions, its magnitude under ischemic conditions is of considerable importance. In the ischemic bed, the driving pressure is low and, thus, the magnitude of the back pressure becomes a major determinant of blood flow into the ischemic myocardium. This study was performed to assess for the existence and the magnitude of a back pressure in ischemic myocardium. If a coronary artery is ligated and the distal portion of the vessel is cannulated and vented to the atmosphere, blood flows retrograde out of the cannula. This retrograde blood flow (RBF) is an index of the collateral flow that would have entered the ischemic myocardium if it had not been diverted (Kattus and Gregg, 1959). This technique was employed to assess the effect of raising the outflow pressure or peripheral coronary pressure (PCP) on the amount of RBF collected. As a result of the study, evidence was obtained that suggests a waterfall mechanism operating on both the collateral vessels as well as on the vessels in the ischemic myocardium. These appear to be two separate and distinct vascular sluices.

Methods

A total of 16 mongrel dogs of either sex weighing between 17 and 34 kg were anesthetized with pentobarbital, 30 mg/kg, intravenously. After endotracheal intubation, the animals were ventilated with an intermittent positive pressure respirator and 100% oxygen. A cutdown was performed on the right groin, and stiff polyvinyl catheters were inserted into the femoral artery and the femoral vein. The femoral artery catheter was passed retrograde up to the ascending aorta to measure the aortic pressure. A thoracotomy was performed at the 5th left intercostal space, and the heart was exposed by incising the pericardium. A catheter was inserted into the left atrial appendage and passed into the ventricular cavity to measure left ventricular pressure. A site on the proximal third of the left anterior descending (LAD) artery was dissected free and a small branch 5–10 mm distal to the dissection site was also dissected free, if available. Heparin (5000 U)
was then given intravenously. The small branch was cannulated in a retrograde fashion with a small bore polyvinyl catheter (8" of PE90, 0.05" o.d., 0.034" i.d.) which was connected to a pressure transducer to measure coronary pressure. This was performed in five dogs. In 10 dogs, the coronary pressure was measured at the cannula tip. In one dog, the coronary pressure was measured simultaneously from a branch and the cannula tip. The LAD artery was then ligated and cannulated with a 13-gauge metal cannula. Connected to this cannula was a segment of tubing which was used for collecting the RBF and also for varying the height of the column of blood in the tubing (Fig. 1). All pressures were measured using Statham P23Db transducers and recorded on a Beckman type SIII multichannel recorder.

### Relationship between Retrograde Blood Flow and the Peripheral Coronary Pressure

The experimental preparation to obtain this relationship is illustrated in Figure 1 and was performed on eight dogs. After ligation of the left anterior descending artery and commencement of RBF, the hemodynamics were allowed to stabilize for at least a 5- to 10-minute period. Arrhythmias were treated with 2% lidocaine, 1–2 ml, intravenously, as required. Since the aortic pressure is an important determinant of collateral blood flow (Kattus and Gregg, 1959), data were collected when the aortic pressure was stable. Timed collections of RBF were performed with graduated cylinders and estimated to the nearest 0.1 ml. Collections were performed over a 1-minute period. In two animals with a relatively large RBF, collections were obtained over a 30-second period. The mean peripheral coronary pressure (PCP) was correlated to the amount of RBF. By adjusting the height of the outflow tubing, the column of blood in the tubing was changed, resulting in a different PCP or back pressure exerted on the retrograde blood flow. While raising or lowering the tubing, we allowed a period of approximately 10–15 seconds for the capacitive charging or discharging transients to dissipate, as monitored by the coronary pressure tracing. Thus, a series of data points (RBF and its associated mean PCP) was obtained. A RBF collection was also obtained at or near 0 mm Hg to be used for normalizing pooled data to this reference value (RBF0). Data were collected in a continuous manner while observing for a changing trend in the aortic pressure by more than 5 mm Hg. If this occurred, data collection was discontinued. The hemodynamic tracings were reviewed after the experiment, and the subset of data collected within a 5 mm Hg aortic pressure range tolerance was considered for further analysis. Data acquired during a period containing ventricular ectopy was excluded from analysis. Lost blood from retrograde bleeding was returned to the animal.

### Evaluation of the Collateral Waterfall

The relationship of RBF as a function of the PCP strongly suggested a nonlinear property of the coronary collateral vessels. This series of experiments done on a different group of dogs (n = 8) was performed to further document and characterize this phenomenon. The experimental procedure was similar to that described above, except that the LAD artery was cannulated and perfused by tubing connected to the left carotid artery. To prevent RBF from entering the ischemic myocardium as the PCP was raised, it was necessary to eliminate this alternative pathway. Nonradioactive 25-μm (25.2 ± 2.1, 3M Company) microspheres were injected in approximately four divided doses into the LAD perfusion tubing. Enough microspheres were injected to mobilize a retrograde flow into the LAD myocardium completely. This procedure has been employed previously by Schulz et al. (1973) and, more recently, by Wyatt et al. (1982). One dog had ventricular fibrillation during the embolization procedure which was successfully converted by DC shock.

An assessment of the completeness of blockade was performed visually, using Evans blue dye injected into the tubing. Absent or minimal streaming of the dye was empirically chosen as an end point. It should be noted that absolute antegrade zero flow theoretically cannot be achieved, since a small amount of antegrade LAD flow will pass across the collateral vessels despite complete obliteration of the native bed. Presumably, the pressure at the portion of the vasculature where the collaterals enter the LAD system increases toward the aortic pressure as the distal vascular bed is progressively emobilized. This would result in flow across the collaterals away from the LAD region as long as the LAD system is pressurized with the prevailing aortic pressure. Fortunately, the actual data obtained will prove to be the most critical check on the adequacy of complete blockade. After achieving blockade, the perfusion tubing from the carotid artery was clamped. A connecting T-side arm to this carotid tubing (distal to clamp site) allowed for RBF collection when opened to atmosphere. Again, the height of this tubing was adjustable, and data to determine the relationship of RBF as a function of PCP were obtained, as described previously.

### Data Analysis

The data describing the relationship between RBF and mean PCP was graphically plotted for each animal. By inspection, it was clear that the relationship consisted of two regions (Figs. 2 and 4): (1) a PCP-independent region (plateau) and (2) a PCP-dependent region. The two regions intersect, forming a presumed "breakpoint." This relationship is difficult to describe statistically. In the nonembolized group, the breakpoint appeared to occur at approximately 10 mm Hg, and for the embolized group the breakpoint occurred at about 20 mm Hg. We arbitrarily chose to perform linear regression analysis on all points equal to and below 10 mm Hg and 20 mm Hg for the nonembolized and embolized groups, respectively. Regression analysis was also performed on all data points above 10 and 20 mm Hg in the nonembolized and embolized groups, respectively. The intersection of the regressions of the plateau region and the PCP-dependent
region was considered the breakpoint or waterfall pressure for the individual experiment. An alternative method to view the plateau region and the breakpoint involves normalizing all the data from each group. Since RBF values at PCP = 0 mm Hg differed between animals, all RBF values from each animal were normalized to the value of the RBF at PCP = 0 mm Hg (RBF₀). This RBF:RBF₀ ratio as a function of the PCP allowed data from all animals in each group to be pooled (Figs. 3 and 5). The plateau portion of the relationship was assessed using linear regression analysis to test for a significant slope (or correlation). Summary data are presented as mean ± SD.

Results

The hemodynamics of the embolized and nonembolized groups are summarized in Table 1. The heart rate, left ventricular diastolic pressure, retrograde blood flow at zero back pressure (RBF₀) were not significantly different between the embolized vs. nonembolized groups. The mean aortic pressure in the embolized group was less than in the nonembolized group: 85 ± 12 mm Hg vs. 101 ± 13 mm Hg. The mean right atrial pressure was only measured in the nonembolized group and had a value of 2.1 ± 1.4 mm Hg. The mean PCP in the nonembolized group measured 25.2 ± 3.8 mm Hg. Pressure-flow measurements derived from measurements at the cannula tip or from an LAD branch were similar. The one dog in which the PCP was measured simultaneously from the cannula tip and an LAD branch demonstrated no pressure difference. Analysis of RBF measurements within 2 mm Hg (1.1 ± 0.5, n = 9) of each other indicated reproducibility within 3.7 ± 3.0%.

Pressure-Flow Relationship into Ischemic Myocardium (Nonembolized)

Figure 2 is a representative plot of the RBF as a function of the back pressure. As the back pressure is progressively decreased from 21 mm Hg, the amount of RBF collected increases in a reasonably linear manner. The mean slope of the PCP-dependent region was −0.38 ± 0.23 ml/min per mm Hg with a mean correlation coefficient of r = −0.98 ± 0.03. At approximately 8–10 mm Hg, further decreases in the back pressure do not result in an increase of RBF. The RBF collected at back pressures below 8–10 mm Hg appears independent of the back pressure. The mean estimated breakpoint derived from the intersection of the regressions from the plateau region and the back pressure-dependent region was 10.7 ± 2.7 mm Hg. The mean slope of the PCP-independent region was −0.018 ± 0.025 ml/min per mm Hg with a mean correlation coefficient of r = −0.37 ± 0.47. To assess this plateau region further, the data from all eight nonembolized preparations were pooled and are presented in Figure 3. Since the RBF differed among the dogs, the RBF...
measurements in each individual experiment was normalized to the RBF value at 0 mm Hg (RBF0). This RBF:RBF0 ratio is plotted vs. the back pressure. The RBF:RBF0 ratio is seen to be reasonably independent of the back pressure below approximately 8-10 mm Hg. Table 2 summarizes the linear regression results of all points below a given PCP (back pressure) range. There is no significant positive or negative correlation between the RBF:RBF0 and the back pressure until all points inclusive of 12 mm Hg is included in the analysis. Analysis excluding the negative PCP values did not alter the general results. A significant negative correlation criteria is probably too insensitive to detect the inflection point (or breakpoint), and probably overestimates it. However, all points inclusive of 8 mm Hg yield a regression that is almost parallel to the abscissa. The actual inflection or breakpoint is likely to reside in the range of 8-12 mm Hg.

Pressure-Flow Relationships across the Coronary Collaterals (Embolized)

A mean of 7.0 ± 2.1 million 25-μm beads were used to embolize the LAD bed completely. The embolized preparations gave a mean stem pressure (Pstem) of 68 ± 11 mm Hg (Table 1). Conceptually, Pstem is the lumped representation of the pressure at the origin or the stem portion of the collaterals (Fig. 6). In our study, this value averaged 79.9 ± 6.1% of the simultaneous aortic pressure, confirming the results of previous studies (Diemer et al., 1977; Wyatt et al., 1982). A mean pressure drop of 17 ± 6 mm Hg occurred from the systemic aortic pressure down to the presumed origin or proximal stem of the collaterals due to the 'large' vessel resistance, Rv (Fig. 6). Figure 4 plots the RBF as a function of the back pressure in an embolized preparation. There is a linear increase of RBF as the back pressure is progressively reduced until about 25 mm Hg, when further reductions of the back pressure do not result in any significant changes in the amount of RBF collected. The mean slope of the PCP-dependent region was −0.13 ± 0.07 ml/min per mm Hg, with a mean linear correlation coefficient of \( r = -0.99 \pm 0.01 \). The mean estimated breakpoint derived from the intersection of the plateau region with the PCP-dependent region was 23.0 ± 2.5 mm Hg. The positive value of the slope of the PCP-dependent region is considered to be a measure of the coronary collateral conductance (Wyatt et al., 1982). The mean slope of PCP-independent region was 0.0037 ± 0.0075 ml/min per mm Hg, with a mean correlation coefficient of \( r = 0.024 \pm 0.58 \). The pooled data for eight experiments are illustrated in Figure 5, where again, the RBF in each individual experiment is normalized to the RBF at 0 mm Hg (RBF0). The RBF:RBF0 ratio is seen to be independent of the back pressure below approximately 20 mm Hg. Table 3 summarizes the linear regression analysis of all the data points up to a given back pressure. Although a significant negative correlation between RBF:RBF0 and the back pressure is achieved when points inclusive of 27.5 mm Hg are analyzed, a more conservative estimate of the breakpoint would be in the range of 20-25 mm Hg. Thus, RBF:RBF0 does not significantly correlate with the back pressure below 20-25 mm Hg.

**Discussion**

The hemodynamic criteria for waterfall behavior were described by Permutt et al. (1962) and later formalized (Lopez-Muniz et al., 1968). They can be briefly summarized as follows (where \( P_i, P_s, P_o \) are the inflow, surround, and outflow pressures, respectively, and \( R \) is the resistance of the collapsible tube): (1) if \( P_i < P_s \); flow = 0 (the vessels are entirely collapsed), (2) if \( P_i > P_s > P_o \); flow = \( (P_i-P_s)/R \) (flow is independent of the outflow pressure), and (3) if \( P_i > P_o > P_s \); flow = \( (P_i-P_o)/R \) (flow is independent of the surround pressure).

In this study, we have employed the load line analysis of Wyatt et al. (1982) to demonstrate each of these three waterfall criteria. In the RBF vs. PCP relationship (nonembolized) illustrated in Figures 2 and 3, RBF appears to be independent of the PCP up until a PCP of approximately 10 mm Hg. Above this critical back pressure, RBF declines in a linear fashion as the PCP is progressively increased. We interpret this breakpoint at approximately 10 mm Hg as the back pressure or waterfall pressure of ischemic myocardium (\( P'_0 \) in Fig. 6). Below a PCP of 10 mm Hg, it appears that none of the divertible

### Table 2

Regression Analysis of Pooled Data (Nonembolized Group)

<table>
<thead>
<tr>
<th>PCP range (mm Hg)</th>
<th>n</th>
<th>RBF:RBF0*</th>
<th>Slope</th>
<th>Y intercept</th>
<th>r</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Through 2</td>
<td>18</td>
<td>0.993 ± 0.025</td>
<td>0.0046</td>
<td>0.999</td>
<td>0.477</td>
<td>0.043</td>
</tr>
<tr>
<td>Through 4</td>
<td>25</td>
<td>0.990 ± 0.027</td>
<td>0.0014</td>
<td>0.990</td>
<td>0.164</td>
<td>0.562</td>
</tr>
<tr>
<td>Through 6</td>
<td>31</td>
<td>0.994 ± 0.031</td>
<td>0.0022</td>
<td>0.992</td>
<td>0.251</td>
<td>0.171</td>
</tr>
<tr>
<td>Through 8</td>
<td>36</td>
<td>0.991 ± 0.042</td>
<td>-0.000007</td>
<td>0.991</td>
<td>-0.0007</td>
<td>0.992</td>
</tr>
<tr>
<td>Through 10</td>
<td>43</td>
<td>0.984 ± 0.044</td>
<td>-0.0020</td>
<td>0.990</td>
<td>-0.209</td>
<td>0.176</td>
</tr>
<tr>
<td>Through 12</td>
<td>50</td>
<td>0.969 ± 0.064</td>
<td>-0.0054</td>
<td>0.993</td>
<td>-0.443</td>
<td>0.002</td>
</tr>
</tbody>
</table>

\( r = \) correlation coefficient; \( P = \) significance of the correlation; \( n = \) cumulative number of data points

* Mean ± s.d.
by Wyatt et al. (1982), except that a PCP-independent region was not noted. This critical pressure of 10 mm Hg in the ischemic myocardium is quite similar to the zero flow pressure axis intercepts derived from diastolic pressure-flow relationships reported from this laboratory (Eng et al., 1982) and elsewhere (Kirkeeide et al., 1981) under nonischemic vasodilated conditions. Thus, it appears that acute ischemia does not alter this effective back pressure to coronary flow. The slope of the PCP-dependent region may reflect the conductance of the intramural vasculature ($R_e$ in Fig. 6). However, it is an indirect assessment. The second and third criteria for waterfall behavior are demonstrated by the pressure-flow relationships in Figures 4 and 5. RBF is independent of the outflow or back pressure until a pressure of about 20 mm Hg (criterion 2). This plateau region was actually alluded to by Kattus and Gregg (1959), who showed in one experiment that raising or lowering the collection tubing 10 cm from heart level did not affect the amount of RBF collected. The plateau region extends to approximately 20 mm Hg, and then "breaks" to become a monotonically decreasing relationship. The "breakpoint" represents the waterfall pressure on the collateral vessels. At higher back pressures, RBF is determined by the difference between the $P_{stem}$ and the back pressure $P_{BP}$ (criterion 3). The waterfall pressure on the collateral vessels demonstrated in Figure 4 is evidence that this phenomenon can occur in nonvenous vessels. Schaper (1971) has found that collaterals in dogs are located mainly in the epicardial regions of the myocardium. This suggests that an effective extravascular pressure of approximately 20 mm Hg can occur in the more superficial layers of the heart. The undeveloped wall structure of acute coronary collaterals may also be a factor in their sensitivity to extravascular pressures.

**Potential Limitations**

The site of back pressure measurement was made either at the cannula tip or from a small cannulated branch distal to the cannula ($P'_{CP}$ in Fig. 6). The desired site of pressure measurement should be at the distal end of the collaterals, which is physically inaccessible. However, an estimation of the pressure

**Table 3**

<table>
<thead>
<tr>
<th>PCP range (mm Hg)</th>
<th>$n$</th>
<th>RBF:RBF$_0$</th>
<th>Slope</th>
<th>Y intercept</th>
<th>$r$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Through 10</td>
<td>26</td>
<td>1.010 ± 0.023</td>
<td>0.0014</td>
<td>1.004</td>
<td>0.245</td>
<td>0.226</td>
</tr>
<tr>
<td>Through 15</td>
<td>34</td>
<td>1.012 ± 0.024</td>
<td>0.0012</td>
<td>1.004</td>
<td>0.264</td>
<td>0.129</td>
</tr>
<tr>
<td>Through 20</td>
<td>44</td>
<td>1.011 ± 0.026</td>
<td>0.0002</td>
<td>1.009</td>
<td>0.070</td>
<td>0.658</td>
</tr>
<tr>
<td>Through 22.5</td>
<td>47</td>
<td>1.007 ± 0.030</td>
<td>-0.0007</td>
<td>1.014</td>
<td>-0.169</td>
<td>0.256</td>
</tr>
<tr>
<td>Through 25</td>
<td>50</td>
<td>1.005 ± 0.030</td>
<td>-0.0009</td>
<td>1.015</td>
<td>-0.248</td>
<td>0.078</td>
</tr>
<tr>
<td>Through 27.5</td>
<td>54</td>
<td>0.996 ± 0.045</td>
<td>-0.0027</td>
<td>1.027</td>
<td>-0.515</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Abbreviations as in footnote to Table 2.

* Mean ± SD.
Analog of the experimental preparation. The left and right sides of the analog represent the nonischemic and ischemic (left anterior descending artery) regions, respectively. $P_A$ is the aortic pressure. $P_{swm}$ is the lumped representation of the pressure at the origin or stem portion of the collaterals. $P_o$ and $P'_o$ are the waterfall pressures in the nonischemic and ischemic beds, respectively. $P_{cp}$ represents the peripheral coronary pressure at the outflow end of the collaterals. $P'_{cp}$ is the peripheral coronary pressure that is actually measured. $P_{bp}$ is the imposed back pressure to retrograde bleeding produced by varying the height of the outflow tubing. $R_L$, $R'_L$, and $R_R$, $R'_R$ represent the large and small vessel resistance in their respective beds. $R_c$ is the collateral resistance. The flow across the collaterals is represented by $i_c$. This flow divides into $i_{rbf}$, the retrograde blood flow diverted out to be collected, and $i_{tm}$ the collateral flow that enters the ischemic myocardium. Note that in the embolized group: (1) $i_{tm} = 0$, (2) $i_c = i_{rbf}$, and (3) when the outflow tubing is clamped to prevent RBF diversion, $P'_{cp} = P_{cp} = P_{stem}$.

Although the plateau region and the PCP-dependent region can be statistically demonstrated to behave differently, the question arises as to whether these regions are linear. Despite the high linear correlation coefficients in the PCP-dependent region, certain arguments would suggest that the PCP-dependent region should not be linear, at least for the nonembolized preparations. The waterfall model of Downey and Kirk (1975) predicted that, at perfusion pressures below the systolic ventricular pressure, the pressure-flow relationship should be nonlinear because of the transmural variation in waterfall pressures. This should be applicable to the nonembolized preparations. Thus a continuum of "breakpoints" are expected over a certain PCP region. Our analysis should not suggest that a unique breakpoint occurs. Rather, the analysis was performed to assess for the minimum waterfall pressure value and to demonstrate that regions of the pressure-flow relationship behave differently. It is likely that the transition value of approximately 10 mm Hg represents the point of initial perfusion of the superficial layers during diastole. As discussed previously, this pressure value is consistent with values derived from antegrade pressure-flow relationships, and appear internally consistent.

An important aspect of the data presented is the interpretation of the plateau portion of the RBF vs. back pressure relationship. Is there complete independence of the RBF from the back pressure below a certain back pressure value? This is the most specific aspect of waterfall behavior among the three criteria since zero flow intercepts (criterion 1) may be produced by "critical closure," independent of extravascular factors. Although independence of the plateau region is not statistically proven, it is fair to state that RBF:RBF$_0$ does not significantly correlate with back pressure below a given value. It is possible that the region is not parallel to the abscissa, but is concave toward the abscissa. From a theoretical viewpoint, concavity in this "plateau" region is strong evidence for vessel compliance factors. Wyatt et al. (1982) suggested this effect in their data. The different interpretations between the present study and their study may be due to the particular attention given to data analysis in the plateau region in the present study. Indeed, collateral load line data from Figure 3 in their paper could also be interpreted as a plateau. We do not imply that collateral vessels...
are indistensible, however. An integral aspect of waterfall behavior involves a lumenal adjustment at some point in the vessel which modulates flow (Permutt and Riley, 1963). Thus, vessel compliance and waterfall behavior are not mutually exclusive, and are related in a yet undefined manner. The determinants of whether flow through vessels is modulated via pure passive compliance and/or waterfall effects are not known. Interestingly, the original paper presented as evidence of waterfall behavior shows evidence of vascular compliance effects (Permutt et al., 1962). However, strictly flat plateau data have been collected (Scharf et al., 1971; Traysman, 1971; Uhlig et al., 1981). Although some individual pressure-flow relationships in our data did demonstrate a degree of concavity in the plateau region, a consistent pattern of curvilinearity could not be discerned. Several relationships yielded multiple identical RBF measurements in the plateau region. These findings are not obviously explainable by compliance effects, and are readily predicted by waterfall hemodynamics.

Considerations for Perfusion of Ischemic Myocardium

There is still a significant controversy regarding whether retrograde blood flow underestimates (Downey et al., 1973; Schulz et al., 1973), overestimates (Eckstein, 1954), or is equivalent (Kirk et al., 1978; Kirk, 1980) to tissue collateral blood flow. This study cannot address this issue. Nevertheless, several new hemodynamic considerations appear to be necessary when analyzing collateral perfusion after coronary occlusion. The "usual" mean PCP measured after acute coronary occlusion in dogs range from 15 to 30 mm Hg. If this natural PCP is less than the collateral waterfall pressure (approximately 20 mm Hg), then the PCP does not participate as a determinant of collateral flow. Rather, the determinant of flow across the collaterals and into the ischemic tissue is the difference between \( P_{\text{stem}} \) and the collateral waterfall (criterion 2). In this sense, the PCP would be a measure of the "backup" of blood below the collateral waterfall, the magnitude of which is dependent on the ischemic tissue conductance and the tissue waterfall pressure. However, if the PCP is greater or rises above the magnitude of the collateral waterfall pressure, the amount of collateral flow would be dependent on the difference between \( P_{\text{stem}} \) and the ischemic tissue waterfall pressure. The collateral waterfall no longer modulates flow. In any event, as suggested by Schaper (1971), the PCP clearly cannot be a very reliable index of collateral flow. This notion becomes apparent when measuring the PCP after complete embolization. The PCP rises up to approximately 80% of the aortic pressure (i.e., \( P_{\text{stem}} \)) when, presumably, minimal collateral flow would be entering the ischemic tissue. Accordingly, the clinical implications of the PCP, as measured during the percutaneous transluminal coronary angioplasty procedure, should be tempered with these considerations.

The load line analysis illustrates the importance of the stem pressure or the pressure at the origin of the collaterals in providing flow across the collaterals. Although the \( P_{\text{stem}} \) in our studies averaged approximately 80% of the aortic pressure, this pressure can be altered by vasodilation of the nonembolized tissue (Diemer et al., 1977), and demonstrates the key hemodynamic event in the "coronary steal" phenomenon (Fam and McGregor, 1964). In addition to alterations in \( P_{\text{stem}} \), changes in the collateral conductance (slope of the load line) and collateral and ischemic tissue waterfall pressures may be possible. Ellis and Klocke (1980) found that changes in ventricular preload affected the tissue zero flow pressures. The effects of preload and drugs on the collateral conductance and waterfall pressure would be of considerable interest, as well.

References


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