The Effects of the Coronary Capacitance on the Interpretation of Diastolic Pressure-Flow Relationships

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SUMMARY. The effects of coronary capacitance on instantaneous pressure-flow (P/F) relationships were analyzed using a theoretical model of coronary flow during diastole that included capacitance. The magnitude of the discrepancy between actual intramural and instantaneously derived P/F relationships was predicted to be dependent on the ratio of two natural decay constants (central aortic decay constant/intrinsic coronary decay constant). The effects of coronary capacitance are eliminated using constant pressure conditions. The instantaneous (dynamic) and constant pressure (static) P/F relationships were compared experimentally using a reservoir to provide constant pressure perfusion during prolonged diastoles in heart blocked dogs. In the presence of coronary tone, zero flow pressure intercepts (Pzf) of 27.1 ± 6.6 and 11.0 ± 3.0 mm Hg were obtained under dynamic and constant pressure conditions respectively, P < 0.001. After maximal vasodilation, Pzf of 14.2 ± 4.5 mmHg and 10.7 ± 2.4 mmHg were obtained under dynamic and constant pressure conditions, respectively, P = NS. Pzf derived under constant pressure conditions were independent of the state of coronary vasomotor tone with a value about 11 mmHg. The slopes of the dynamic P/F relationships tended to be greater than those derived from constant pressure conditions. This may suggest an additional component of increasing coronary resistance during diastole that could not be readily assessed under dynamic conditions. We conclude that coronary capacitive effects and resistance changes during diastole severely limit the interpretation of instantaneous dynamic P/F relationships. Diastolic coronary perfusion ceases at about 11 mm Hg and is independent of coronary tone when capacitive effects are eliminated. (Circ Res 50: 334-341, 1982)

PREVIOUSLY, coronary pressure-flow relationships (P/F) have been considered only in terms of assessing resistance characteristics. However, recent evidence suggests that the zero flow pressure intercept (Pzf) is of significant magnitude and must also be considered. Bellamy (1978) analyzed the instantaneous coronary P/F relationships during diastole in chronically instrumented dogs. He found P/F relationships to be linear with Pzf as high as 50 mm Hg in the presence of coronary tone. During reactive hyperemia or adenosine induced vasodilation, the instantaneous P/F relationships had a greater slope (interpreted as an increase in coronary conductance) and a decrease in the Pzf to about 20 mm Hg. These results have been confirmed in open-chest anesthetized preparations (Canty and Klocke, 1979).

The question remains as to the correct interpretation and physiological significance of these high diastolic pressure intercepts. If coronary inflow correctly represents the flow in the microcirculation at every instant of time, then these high pressure intercepts could reflect the downstream back pressure of a vascular waterfall (Permutt and Riley, 1963). Critical closing pressures of this magnitude during diastole would certainly play a major role in the regulation and distribution of coronary flow (Hoffman, 1978). However, the assumed instantaneous equivalence of coronary inflow and intramural flow cannot be made in the presence of reactive components of the coronary system (capacitance and inertia). In particular, capacitive properties of the coronary system could provide continued intramural flow despite zero coronary inflow as measured by a flow meter. This underestimation of intramural flow can result in an overestimation of the true zero flow pressure intercept. In this study, the consequences of coronary capacitance on the instantaneous P/F relationships are analyzed by means of a simple theoretical model of coronary flow during diastole. Experimental data is presented to test the validity and predictions of the model. Specifically, P/F relationships are obtained under steady state (constant pressure) conditions and compared to those obtained under dynamic (declining pressure) conditions.

Methods

Theoretical Model

In the electrical analog representation of the coronary system in Figure 1, Pd denotes the aortic driving pressure, Po the back pressure which can represent either the coronary sinus pressure, intramyocardial pressure, or critical closing pressure, whichever is larger. The coronary capacitance, C, is in parallel to the small vessel resistance, Rs. Although the resistance of large vessels is ignored in the analysis, it can be shown that this does not affect the overall interpretation of results (see Appendix). The intramural flow, iwm, is the sum of the coronary inflow, i, and the flow contributed by the discharging capacitor, ic. Assuming (1) a constant resistance,
artifactually decreased and the actual intramural back pressure, Po, is artifactually approaches unity. Thus, after vasodilation, the model predicts that the actual intramural conductance \(1/R_B\) is

\[i = \frac{1}{(1/a_c)} \left( \frac{P_A}{P_0} - \frac{1}{1 - a_c} \right)\]  

FIGURE 1. Theoretical model of coronary blood flow during diastole (see text). The analysis indicates that coronary capacitance significantly affects the slope and zero flow pressure axis intercept of the instantaneous P/F relationship. This effect is graphically demonstrated at the bottom of the figure.

\[R_a\) during diastole, (2) a linear capacitor, \(C\), and (3) an exponentially decaying aortic driving pressure of the form \(P_A = k \exp(-\alpha t)\), an instantaneous coronary P/F relationship can be calculated:

\[i = \left( \frac{1}{R_a} - \alpha C \right) \left( \frac{P_A}{P_0} - \frac{1}{1 - \alpha C R_a} \right)\]  

This equation can be arranged into a more easily interpretable form by defining the entity, \(a_c = 1/(R_s C)\), the instantaneous P/F relationship becomes:

\[i = \left( \frac{1}{R_a} \right) \left( 1 - a_c \right) \left( \frac{P_A}{P_0} - \frac{1}{1 - a_c} \right)\]  

where \(\alpha\) is the central aortic decay constant and \(a_c\) is the intrinsic coronary decay constant. This coronary decay constant is theoretically and practically obtained by occlusion of the coronary artery and measuring the subsequent peripheral coronary pressure decay rate.

Equation 2 indicates that correlation of instantaneous coronary inflow (i) vs. pressure (P_A) would yield a linear relationship with a slope of \(1/(R_a) \left( 1 - a/a_c \right)\) and a zero flow pressures axis intercept, \(P'_A = P_0/(1 - a/a_c)\). The actual conductance, \(1/R_a\), and the actual back pressure, \(P_0\), are modified by the factor \(1 - a/a_c\) if measurements are performed under dynamic conditions. Thus, the ratio of two natural decay constants, \(a/a_c\), determines the deviation of the P/F relationship obtained under dynamic conditions from the actual intramural P/F relationship. If the two decay constants were of comparable magnitude, then this model predicts that the actual intramural conductance \(1/R_a\) is artificially decreased and the actual intramural back pressure, \(P_0\), is artificially magnified using dynamic measurements.

The effects of capacitance can be eliminated by constructing P/F relationships under conditions of constant pressure (\(\alpha = 0\)) and thus arrive at the actual intramural conductance and pressure intercept (Figure 1). Furthermore, if \(a_c\) is made large by coronary vasodilation, then the factor \(1 - a/a_c\) approaches unity. Thus, after vasodilation, the model predicts that the dynamic P/F relationship will approach the actual intramural relationship. Based on this theoretical model, experimental preparations were studied in which diastolic coronary P/F relationships were compared using dynamic (decaying pressure) and static (constant pressure) conditions. The behavior of the dynamic P/F relationship relative to the static P/F relationship during maximal coronary vasodilation also was determined.

Animal Experiments

Thirteen mongrel dogs of either sex weighing between 25 and 34 kg were anesthetized with sodium pentobarbital, 30 mg/kg, intravenously. Additional doses were given as required. After endotracheal intubation, ventilation was provided by an intermittent positive pressure respirator with 100% oxygen. A left thoracotomy was performed on the 4th left intercostal space and the heart was exposed after the pericardium was incised. After administration of 10,000 units of heparin, a polyvinyl catheter was inserted into the femoral artery and passed up into the ascending aorta to measure systemic pressure and another catheter was inserted into the femoral vein for fluid and drug administration. A bipolar electrical pacing electrode was sewn onto the surface of the right ventricle and the heart was electrically paced by a Grass S53 stimulator at a heart rate of 130 beats/min. An electrocautery tip was inserted through a stab wound in the right atrial appendage, secured with suture, and the area of the AV node cauterized. Successful destruction of the AV node resulted in prolonged diastoles upon cessation of electrical pacing. The electrocautery tip was then removed. A 15- or 18-gauge metal cannula attached to a 6 inches of stiff polyvinyl tubing (0.07 inch i.d.) was inserted into the second or third marginal branch of the left circumflex artery to measure coronary pressure. A polyvinyl catheter was inserted into the left atrial appendage and passed into the left ventricular cavity to measure ventricular pressure. In five dogs, an additional catheter was inserted into the right atrium to measure right atrial pressure after the electrocautery tip was removed. Pressures were measured with Statham P23Db transducers, with zero reference at the level of the right atrium. All pressure and electromagnetic flow measurements were recorded on a Beckman type SII multichannel recorder.

In four dogs, the thoracic aorta was cannulated with a large bore T-tube that was connected to a reservoir (Fig. 2). This aortic reservoir was filled with autologous oxygenated blood from a donor. The reservoir, when opened, buffered aortic pressure and was able to maintain aortic pressure constant during the measurement period in diastole. Different levels of constant pressure perfusion were obtained by varying the height of the reservoir. In these experiments, the proximal left circumflex artery was dissected free. A flow transducer of 1.5 to 2.5 mm i.d. (series D, In Vivo Metrics) was placed on the proximal left circumflex artery and was connected to a sine wave electromagnetic flow meter (Model BL410 Biotronex Laboratories). An occluder was placed just distal to the flow transducer for mechanical zero flow. Both flow transducer and occluder were proximal to the cannulated branch measuring coronary pressure.

In nine dogs, the left main coronary artery was cannulated with a brass Gregg cannula inserted via the subclavian artery and perfused by tubing connected to the left carotid artery (Fig. 2). A reservoir (left main reservoir) was connected to the perfusion line to provide constant pressure perfusion of the left main coronary artery. This reservoir was filled with blood drawn from the femoral artery prior to any measurements. An on-line flow transducer (model...
vascular status at the beginning of diastole. In each dog, P/F was abolished after a 5- to 10-second occlusion.

Dilation was achieved when the reactive hyperemic response pressure conditions before and after maximal coronary F relationships were obtained under dynamic and constant diastole (within 0.2 second) as most representative of the P/F relationships were taken immediately after induction of some cases, the coronary flow was noted to decrease slightly as the duration of diastole progressed. This was interpreted as an autoregulatory response during diastole since this may represent an autoregulatory response (increasing resistance). The least square regression line was fitted to the pressure-flow data yielding the slope, the extrapolated zero flow pressure axis intercept (P_a), and the correlation coefficient of the individual P/F relationships. Differences between P_a obtained under aortic vs. left main preparations and dynamic vs. constant pressure conditions were assessed by a two-way analysis of variance with repeated measures method. Differences among groups were tested by the Scheffe multiple comparison test with a P < 0.05 considered to be a significant difference. Differences between P/F slopes were tested using Student's paired t-test. Hemodynamic parameters were compared using analysis of variance. Summary data are expressed as the mean ± sd.

**Results**

Figure 4 illustrates the P/F relationships obtained in one experiment under four different conditions. In the presence of coronary tone, the P/F relationships obtained under dynamic and constant pressure conditions differ in both slope and zero flow intercept. After vasodilation, the dynamic P/F relationship tends to be more similar to that obtained under constant pressure conditions. The zero flow intercept obtained under constant pressure conditions are of similar magnitude and indicates that vasodilation affects only the slope of the static P/F relationship. This is in contrast to the dynamic pressure flow relationships, where vasodilation affects both the slope and zero flow pressure intercept. Figure 5 isolates the P/F relationships obtained under constant pressure conditions from another experiment. The P/F relationships are quite linear over a wide representative diastolic pressure range and suggest a common pressure intercept at about 10 to 11 mm Hg unaffected by the state of coronary vasomotor tone.

**Data Analysis**

Dynamic diastolic P/F relationships were obtained by switching the pacemaker off. During these prolonged diastoles, the simultaneous declining coronary pressure and flow were recorded. The dynamic P/F relationships were obtained by correlation of the instantaneous data at 0.1-second intervals 0.2 second after initiation of diastole, as previously described (Bellamy, 1978). A dynamic P/F relationship was repeated immediately after static P/F relationship data were obtained to assess for reproducibility. P/F relationships under constant pressure conditions were obtained by simultaneously switching to both reservoir perfusion (reservoir clamp off) and diastole (pacemaker off) (Fig. 3). This procedure was repeated to obtain a series of pressure (and the associated flow) points by varying the height of the reservoir. A minimum of six points (mean of eight points) were obtained to construct each P/F relationship under constant pressure conditions. Although the reservoir provided constant pressure perfusion during diastole, the coronary flow was noted to decrease slightly as the duration of diastole progressed. This was interpreted as an autoregulatory response during diastole since this phenomenon was observed neither after maximal vasodilation nor at constant perfusion at pressures lower than 30 mm Hg in the presence of coronary tone. In these cases, flow values were taken immediately after induction of diastole (within 0.2 second) as most representative of the vascular status at the beginning of diastole. In each dog, P/F relationships were obtained under dynamic and constant pressure conditions before and after maximal coronary vasodilation with carbochromen 8 mg/kg, iv. Maximal vasodilation was achieved when the reactive hyperemic response was abolished after a 5- to 10-second occlusion.
FIGURE 4. Static and dynamic P/F relationships obtained from a left main preparation. In the presence of coronary tone, P/F relationships under static (constant pressure reservoir perfusion) and dynamic (declining pressure perfusion) conditions differ in both slope and pressure axis intercept. After vasodilation, the dynamic P/F relationship approximates the static P/F relationship. These results are predicted by the theoretical model.

The mean aortic pressure of the experimental preparations was 90.6 ± 13.6 mm Hg and significantly decreased to 69.0 ± 14.3 mm Hg after vasodilation with carbochromen. The linearity of the P/F data obtained under the various conditions was excellent. The mean correlation coefficient of all 52 P/F relationships was 0.98 ± 0.01. Instantaneous dynamic P/F relationships were reproducible. Dynamic P/F relationships done before and immediately after obtaining the static P/F relationship data points averaged less than a 5% difference in extrapolated Prf values and were not significantly different. Table 1 lists the Prf obtained under the various conditions. There was no significant difference between the Prf obtained by aortic and left main experimental preparations. Prf obtained under dynamic conditions (27.1 ± 6.6 mm Hg) was significantly higher than when obtained under constant pressure conditions (11.0 ± 3.0 mm Hg) in the presence of coronary tone (P < 0.001). After coronary vasodilation, the Prf obtained under dynamic conditions (14.2 ± 4.5 mm Hg) although higher than that obtained under constant pressure conditions (10.7 ± 2.4 mm Hg) was not significantly different, P > 0.22. Prf obtained under constant pressure conditions with (11.0 ± 3.0 mm Hg) and without (10.7 ± 2.4 mm Hg) coronary tone were not different.

Table 2 lists the slopes of the pressure-flow relationships obtained under the various conditions. The slopes obtained from the aortic experimental preparations was about one-half of the values obtained from the left main preparation, as was expected, since only the left circumflex vascular distribution was assessed in the aortic preparation. The greater overall variability in the slope data is due in part to the differences in mass of the perfused myocardium among the 13 experiments. In the presence of coronary tone, slopes of 1.14 ± 0.32 (dynamic) vs. 1.01 ± 0.45 (static) ml/min per mm Hg were obtained with aortic pressure perfusion and 3.54 ± 1.28 (dynamic) vs. 2.84 ± 0.87 (static) ml/min per mm Hg with the

![Graph](http://circres.ahajournals.org/)

**FIGURE 5.** P/F relationships under constant pressure conditions in a left main preparation. The state of coronary vasomotor tone does not appear to affect the pressure axis intercept. Rather, the effect of coronary vasodilation is a pure slope (conductance) change. A family of P/F relationships are possible bounded by the relationships representing maximum and minimum conductance and all with a common pressure intercept at about 10 mm Hg. This picture is consistent with the traditional view of autoregulation as a pure resistance change.

![Graph](http://circres.ahajournals.org/)

**TABLE 1**

<table>
<thead>
<tr>
<th>Zero Flow Pressure Intercepts (Prf)</th>
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<tbody>
<tr>
<td><strong>Coronary tone</strong></td>
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<tr>
<td>(n = 9)</td>
</tr>
<tr>
<td><strong>1. Declining pressure</strong></td>
</tr>
<tr>
<td><strong>2. Constant pressure</strong></td>
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<tr>
<td><strong>Vasodilation</strong></td>
</tr>
<tr>
<td><strong>3. Declining pressure</strong></td>
</tr>
</tbody>
</table>

* Data in the form of mean (mm Hg) ± so.
† P < 0.001 compared to (1).
‡ P = not significant compared to (4).

**TABLE 2**

<table>
<thead>
<tr>
<th>Slopes of Pressure-Flow Relationships</th>
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<tbody>
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<td><strong>Coronary tone</strong></td>
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</tbody>
</table>

* Data in the form of mean (ml/min per mm Hg) ± so.
P < 0.025 compared to declining pressure.
Table 3

<table>
<thead>
<tr>
<th>Hemodynamic Parameters</th>
<th>mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reservoir pressure intercept (n = 26)</td>
<td>10.9 ± 2.7</td>
</tr>
<tr>
<td>Left ventricular diastolic pressure (n = 26)</td>
<td>6.5 ± 3.5*</td>
</tr>
<tr>
<td>Right atrial pressure (n = 10)</td>
<td>3.4 ± 1.8*</td>
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</tbody>
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Data in the form of: mean ± SD.

* P < 0.001 compared to reservoir pressure intercept.

Discussion

Capacitive Effects on Measurements

Although the effects of the reactive components of the coronary system are not readily apparent, they can exert a major influence on the interpretation of certain experimental relationships. That the capacitive properties of the vascular system significantly affects the interpretation of blood flow measurements is illustrated by the characteristic blood flow pattern in the ascending aorta. Measured aortic blood flow effectively ceases at end-systolic pressure and through-out diastole. However, peripheral blood flow continues despite the zero measured blood flow proximally. This study confirms that this capacitive phenomenon also occurs in the coronary system. The instantaneous diastolic coronary P/F relationship as previously analyzed (Bellamy, 1978) uses dynamic measurements of coronary inflow and pressure. A theoretical analysis of the coronary system that includes capacitance indicates that the underestimation of intramural coronary flow due to capacitive effects can result in both an underestimation of the true conductance and over-estimation of the true intramural zero flow pressure intercept.

Referring to Equation 2, both the slope and zero flow pressure intercept are modified by the term (1 - α/c). Thus, the instantaneous P/F relationship is dependent on the ratio of two natural decay constants. Although the absolute magnitude of coronary capacitance (C) is relatively small compared to the central system, the magnitude of coronary conductance (1/Rc) is also relatively small compared to the central system. Thus, it is quite possible that two vascular beds (coronary and central systems) may have decay constants (1/RC) that are comparable in magnitude despite large differences in their respective absolute resistance and capacitance values. Hence, coronary capacitance, although small in magnitude, cannot be ignored when dynamic measurements are analyzed.

In the presence of coronary tone, αc is difficult to quantitate accurately, since analysis of the diastolic coronary pressure decay rate after occlusion of the left main coronary artery undoubtedly is affected by changing resistance due to autoregulation. However, as a gross approximation, we have analyzed several coronary decay rates and estimated an intrinsic coronary decay rate of about one-half the central aortic decay rate in the presence of coronary tone (Fig. 6). From the theoretical model, this would result in a doubling of the true zero flow pressure intercept using...

![Figure 6](https://example.com/fig6.png)

**Figure 6.** Left main coronary occlusion in mid-diastole. A prolonged diastole was produced by turning the pacemaker off. The initial coronary pressure decay represents the central aortic pressure decay rate (α = 0.252 sec⁻¹) in mid-diastole; the left main coronary was occluded and the subsequent pressure decay represents the intrinsic coronary decay rate (αc = 0.090 sec⁻¹). The ratio α/αc = 0.37. In this experiment, the zero flow pressure intercept under constant pressure conditions was 13.0 mm Hg. The theoretical model would predict a Pzf = 21.4 mm Hg under dynamic conditions. In fact, the experimental dynamic Pzf = 25.4 mm Hg.
dynamic measurements. We suspect that this ratio varies depending on the basal state of the experimental preparation.

Equation 2 also predicts that if $\alpha > \alpha_c$, as could occur in the rapid aortic pressure decay of aortic insufficiency, there would be negative coronary inflow (coronary reflux) during diastole. This has been demonstrated experimentally (Folts and Rowe, 1974) and has been reported in angiographic studies of patients with aortic insufficiency (Carroll and Falsetti, 1976). Although the coronary reflux was attributed to mechanical properties of the aortic valve and/or the Bernoulli effect, a discharging coronary capacitance is a likely explanation. Theoretically, the dynamically derived $P_d$ could be manipulated arbitrarily. This is demonstrated in Figure 5 from the article by Folts and Rowe (1974). During production of experimental aortic insufficiency, and thus a manipulated increase in the aortic decay rate ($\alpha$), the diastolic pressure at which zero coronary flow occurs ($P_{zf}$) is seen to be about 75 mm Hg. Several beats later, without aortic insufficiency, significant flow occurs at a pressure of 75 mm Hg indicating a lower $P_{zf}$ at the slower decay rate.

If $\alpha_c > \alpha$, as would occur after full coronary vasodilation, Equation 2 predicts that the dynamic P/F relationship would tend to be similar to the P/F relationship under constant pressure conditions. Our results confirm this prediction in that both the slopes and the $P_{zf}$ were not significantly different under dynamic compared to constant pressure conditions after full coronary vasodilation.

Assumptions and Experimental Limitations

In testing the capacitive model using experimental data the coronary resistance, $R_s$, is assumed to be constant during diastole. This assumption probably is appropriate after maximal coronary vasodilation. However, in the presence of normal coronary vaso-motor tone, resistance may change during the prolonged diastole. In several experiments, coronary flow was noted to decrease with time despite constant pressure perfusion (Fig. 3). This is interpreted as an autoregulatory phenomenon (increasing resistance) in response to a decreased metabolic demand, since it was never observed after full vasodilation. For this reason, we chose the earliest flow value after switching to reservoir perfusion as most representative of the status of coronary tone upon induction of diastole. Of interest, an increase in flow (decreasing resistance) was not observed in our experiments using constant pressure perfusion even in the low pressure range where it might be expected to occur.

Resistance changes during the dynamic measurements are more difficult to assess. Dynamic P/F relationships may still appear linear despite changes in resistance (Spann, 1979). Figure 7 illustrates the effect of an increasing resistance during diastole on the dynamic P/F relationship. The net effect of increasing resistance during diastole is to give an apparent P/F relationship with a greater slope and a greater $P_d$ than if resistance were constant. The apparent P/F relationship obtained from dynamic measurements could therefore yield slopes greater than would be predicted on the assumption of constant coronary resistance, and might even exceed the slopes obtained under constant pressure conditions. Thus, a greater slope seen under dynamic conditions provides indirect evidence of resistance change during diastole. Furthermore, resistance changes during diastole may result in a more overestimation of actual $P_d$ over and above the effect due to capacitive effects alone.

Another source of experimental error includes the extrapolation of data to obtain the $P_d$. The P/F relationships as a group were quite linear (mean $r = 0.98 \pm 0.01$), similar to reported results (Bellamy, 1978). However, among the P/F relationships obtained under constant pressure conditions, nonlinearity in the low pressure (less than 20 mm Hg) and flow range cannot be excluded. Thus, if there exists a transmural gradient of diastolic closing pressures within the myocardium, curvature would be expected at the point of the highest closing pressure (Downey and Kirk, 1975). In this case, it can be shown that the extrapolated $P_d$ represents the regional conductance weighted mean transmural value. In the experiments involving left main coronary cannulation, the compliance of the perfusion tubing could have affected the results under dynamic conditions. In addition, collateral perfusion from the right coronary artery could theoretically elevate the estimated $P_d$. However, results using the aortic reservoir preparation were not different from the results obtained with left main preparations, suggesting that these effects were minimal. A nonlinear capacitance would theoretically produce nonlinear instantaneous P/F relationships. Although the experimental P/F relationships were fitted to linear relation-
ships over a limited pressure range, curvature may well be obtained over a larger pressure range. However, constant pressure P/F relationships should be unaffected by a nonlinear capacitor, since the capacitance effect is eliminated.

Diastolic Coronary Pressure-Flow Relationships

In the presence of coronary tone, the Pd obtained under dynamic conditions was significantly higher than under constant pressure conditions (27.1 vs. 11.0 mm Hg). This overestimation of Pd using dynamic measurements is due to capacitive effects with a possible contribution of resistance change as noted above. After vasodilation, the Pd obtained under dynamic and constant pressure conditions were not significantly different (14.2 vs. 10.7 mm Hg). The Pd obtained under constant pressure conditions with and without tone were not different (11.0 mm Hg vs. 10.7 mm Hg), suggesting that the actual zero flow pressure intercept is independent of coronary tone. These results are consistent with the simple capacitive model. Although the Pd obtained under constant pressure conditions was about 11 mm Hg, it was still significantly higher than the simultaneous left ventricular diastolic and right atrial pressures. This is compatible with a critical closing phenomenon due to a vascular waterfall mechanism during diastole (Permutt and Riley, 1963).

It has been demonstrated (Bellamy, 1978) that the minimal Pd obtained under dynamic conditions during reactive hyperemia and full vasodilation are similar (about 20 mm Hg), suggesting that this may represent the effective back pressure in ischemic myocardium. In contrast, our mean critical closing pressure of about 11 mm Hg should be compared to the diastolic peripheral coronary pressures seen after acute coronary occlusion which range from 10 to 20 mm Hg. Since it is difficult to envision how diastolic critical closing pressure could exceed the diastolic peripheral coronary pressure, the reported Pd obtained under dynamic conditions appear to be inconsistent with, or leave little room for, diastolic collateral perfusion of ischemic myocardium.

In the presence of coronary tone, the slopes of the P/F relationships under dynamic conditions were not less than under constant pressure conditions, as the theoretical model predicted. As discussed above, these results can be explained by resistance change (increasing) during diastole that could not be directly assessed under dynamic conditions (Fig. 7). However, after coronary vasodilation, dynamic and constant pressure slopes were not significantly different as predicted by the model. The P/F relationships obtained under constant pressure conditions revealed that the effect of coronary vasodilation was an increase in conductance (slope) without a change in the zero flow pressure intercept (Fig. 5). This is consistent with the traditional view of autoregulation as a pure change in resistance rather than changes in both resistance and Pd, as suggested by Canty and Klocke (1979). Thus it is possible that there exists a family of relationships that are bounded by the two P/F relationships representing minimum and maximum conductance, all with a common pressure intercept.

End-diastolic coronary pressure divided by end-diastolic flow is commonly used to calculate coronary resistance. It has been suggested that it is the most reliable index of coronary resistance minimizing the reactive components (Gregg, 1950). Yet, because it is an instantaneous dynamic measurement, end-diastolic intramural flow is underestimated, resulting in an overestimation of coronary resistance. In addition, neglecting Pd, although small (11 mm Hg), results in an additional overestimation of actual resistance. Furthermore, the use of this index to assess the directional resistance changes from various interventions could fail as well. Depending on the effect of an intervention on the relative decay rates of the central and coronary systems, calculated directional coronary resistance changes may not reflect true change. For instance, a drug that predominantly affects the central system (selectively altering the central pressure decay rate with little effect on the coronary decay constant) would be incorrectly interpreted to have coronary vasomotor effects. Thus, some of our present concepts of coronary resistance and directional resistance changes due to interventions may require reassessment.

Appendix

The addition of large vessel resistance, Rf, between PA and the capacitance C (C placed in parallel between RL and Rf) can be analyzed in a manner similar to that shown in Figure 1.

The solution for instantaneous coronary inflow, i, involves a linear first order differential equation:

\[ \frac{di}{dt} + \frac{1}{CRfRr}i = \frac{1}{CRfRr} \left( P_{A} + CRf \frac{dP_{A}}{dt} - P_{0} \right). \]

Assuming an aortic driving pressure during diastole of the form \( P_{A} = ke^{-\alpha t} \) (a is the central aortic decay constant) and defining \( \alpha_{c} = 1/(RC_{C}) \), the differential equation (3) can be solved exactly. Experimentally, instantaneous P/F relationships are quite linear. This imposes a boundary condition that effectively makes negligible the homogeneous solution of Equation 3. Thus, the solution for coronary inflow involves only the particular solution. The instantaneous P/F relationship becomes:

\[ i = \frac{(1 - \alpha_{c}/\alpha_{c})}{(1 - \alpha_{c}/\alpha_{c})R_{c} + R_{e}} \left( P_{A} - \frac{1}{\alpha_{c}} \frac{R_{c}}{R_{c} + R_{e}} \left( P_{d} - \frac{R_{c}}{R_{c} + R_{e}} \right) \right). \]

This equation reduces to Equation 2 (in text) for \( \alpha_{c} = 0 \). Instantaneous P/F relationships yield slopes and Pd that are affected by the relative magnitudes of the coronary and central aortic decay constants.

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