The Role of Vascular Capacitance in the Coronary Arteries

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SUMMARY. When the left coronary artery was perfused with nonpulsatile pressure, the onset of diastole was accompanied by a capacitance overshoot in flow with an exponential decay back to a steady state. Time constant for that decay ranged from 55 msec when tone was present to 105 msec with maximal dilation. Since the transient resulted from a fall in tissue pressure, this represents an estimation of intramural arterial capacitance only. Transients in perfusion pressure, which would also affect epicardial arteries, yielded similar time constants. We concluded that most of the coronary capacitance resides in the small intramural vessels. Analysis of transients yielded a value for capacitance of between 0.01 and 0.05 ml/mm Hg per 100 g. We then used the data from the transients to construct coronary pressure flow curves which were free of any back flow from capacitance. When coronary tone was present, the curves indicated that flow ceased at 30 mm Hg. With maximal dilation, flow ceased at only 18 mm Hg. Long diastoles in those same hearts indicated that flow ceased at about 10 mm Hg higher pressure. Although capacitance causes critical closing pressure as determined by a long diastole to be artificially high, critical closing pressure is still appreciable in the heart, and tone dependent. Finally, three computer models were built, one of which included only small vessel capacitances, the second, only vascular waterfalls, and the third, both of the above. Only model 3 was capable of reproducing the flow patterns which were actually seen. (Circ Res 55: 751-762, 1984)

It has been known for some time that the walls of the coronary arteries are elastic and that their volume is a function of the internal pressure. This distensibility, often termed capacitance, was traditionally thought to reside in the epicardial coronary arteries. Recently, however, Spaan et al. (1981a, 1981b) have presented evidence that this capacitance is much larger than previously estimated (Douglas and Greenfield, 1970), with much of it residing in the small intramural vessels.

The ramifications of the theories of Spaan et al. are far reaching. For example, they present evidence that the time constant with which this capacitance charges and discharges is several seconds long. Since intramyocardial pressure represents the pressure surrounding the capacitance vessel site, the pressure inside the microvessels should rise and fall by the same increment as intramyocardial pressure. This would have to occur, because the time required for filling or emptying these vessels would be much longer than one beat. Vascular waterfall formation, which has been proposed to represent the mechanism whereby cardiac contraction inhibits regional coronary flow (Downey and Kirk, 1975), would be theoretically impossible, since the pressure gradient across the microcirculation would obviously not be diminished in systole. Inhibition of flow by the waterfall mechanism could occur only if the time constant for discharge of the small vessel capacitance were significantly shorter than one beat.

Another aspect of the study by Spaan et al. deals with the recent reports of a high critical closing pressure in the coronary system. Bellamy (1978) reported that the pressure-flow relationship for the coronary artery was linear but had a zero flow intercept which could be as high as 50 mm Hg. These high critical closing pressures were reported to be a function of coronary artery tone and reportedly fell to values of 15-20 mm Hg when the coronary artery was maximally dilated. Since Bellamy's measurements were made at a time when the perfusion pressure was falling during a long diastole, it has been argued that a concealed back flow was associated with the discharge of coronary artery capacitance (Eng et al., 1981). When the forward flow through the microcirculation equaled the capacitive back flow, it would appear that inflow had stopped and a critical-closing phenomenon had occurred, even though forward flow still would be present at the microcirculatory level.

The aim of this study was to try to determine accurately both the site and the magnitude of the coronary arterial capacitance. Not only would this allow us to determine whether vascular waterfall formation is precluded in the coronary bed, but, also, we could determine to what extent coronary capacitance distorts pressure-flow curves determined by the long diastole method. Toward this end, we examined flow transients associated with step changes in coronary perfusion pressure analyzed in terms of resistance capacitance (R-C) networks, and these were compared with similar tran-
sients associated with the onset of systole and diastole. Second, we analyzed the pressure-flow data in such a way that the capacitive flow component could be eliminated to provide an estimate of the zero flow intercept for the dog’s coronary artery which would be free of capacitance artifact. Finally, we constructed computer models of the proposed capacitive arrangements in the coronary system and examined their ability to reproduce the phasic blood flow patterns actually observed in the coronary artery.

**Methods**

**Animal Models**

Mongrel dogs of either sex were used for these studies. They were anesthetized with 30 mg/kg of sodium pentobarbital, iv. Their chests were opened with a thoracotomy in the 4th left intercostal space. They were ventilated with room air under positive pressure while their chests were open. The hearts were exposed and the main left coronary artery was dissected free at its juncture with the aorta, and a ligature was passed around it. We then produced a complete atrioventricular (AV) block through a right thoracotomy by injecting 0.1 ml of 40% formalin into the region of the atrioventricular (AV) node (Steiner and Kovalik, 1968). Once the heart block had been achieved, a pair of silver pacing electrodes were sewn onto the free wall of the right ventricle. The hearts were paced electrically with 7-V pulses of 1 msec duration and 120 beats/min.

A double-lumen cannula (Cohen et al., 1973) was then introduced through the subclavian artery and passed retrograde into the coronary ostium where the tip was tied in place with the ligature. Arterial blood was drawn from the outer lumen of the cannula which resided in the aortic wall. This was propelled through the tygon tubing circuit by a Harvard Apparatus peristaltic pump. Pulsations in the circuit were damped by a 100-ml Plexiglas Windkessel reservoir in the perfusion circuit which was filled with air. A section of tubing connected this reservoir with a second, air-filled, 2-liter reservoir. By adjusting the second reservoir’s pressure to the desired level, and then abruptly removing the clamp on the tubing, the pressure in the first reservoir, and, thus, the perfusion circuit, could be caused to change suddenly (time constant <10 msec) to any level desired. A Carolina Medical Electronics extracorporeal blood flow probe, 4 mm i.d., was used to measure blood flow in the circuit and was positioned as near the coronary cannula as possible. The low-pass filter on the flowmeter was set at 100 cps, and frequency response was tested by a sudden occlusion of the perfusion tubing just proximal to the coronary cannula. The fall-time was less than 5 msec. With the clamp in place, a 50 mm Hg step in perfusion pressure caused a negligible flow increment, indicating that the capacitance of the perfusion line was negligible. Perfusion pressure was measured from a branch of the circuit with a Statham P23DB pressure transducer. Ten thousand units of heparin were given intravenously to prevent clot formation in the perfusion circuit.

**Phasic Flow under Constant Pressure**

To observe the phasic nature of the coronary blood flow, we perfused the coronary with a constant pressure while blood flow was recorded. Because we could not eliminate atrial coves, respiratory influences, or all pump noises from the perfusion pressure, we signal averaged 10 consecutive beats with a Digital Equipment PDP12 computer. The computer then did an analysis of the exponential decay portion following the early diastolic overshoot in flow, and printed out a calculated time constant for the decay. This was done by standard exponential least squares fit algorithm. Points were sampled from the flow record at 1-msec intervals by the computer through a 10-bit A to D converter. Care was taken to be sure that pulsations in the perfusion pressure were kept to a minimum for this protocol by using a 2-liter air-filled buffer bottle in the perfusion system. As a result, coronary pulse pressure in the seven dogs averaged only 8.3 ± 2.7 (SEM) mm Hg. Finally, adenosine was infused into the perfusion line at 1 mg/min to induce a state of maximal dilation when required.

**Pressure Transients**

When the step transient protocol in coronary perfusion pressure was performed, the heart was first stopped by switching off the pacing signal. Shortly after the heart had stopped beating, the clamp was removed from the tubing connecting the two reservoirs. The flow was allowed to stabilize for several seconds and then the pacing was resumed and the clamp replaced. If the transient had dropped to a lower pressure, the primary reservoir would have accumulated an additional volume of blood. This was removed by stopping the pump and pumping air into the reservoir until it had been displaced. Similarly, if the transient were increased to a higher pressure, blood would have been lost. The volume was replaced by letting air out of the reservoir and increasing the rate of the peristaltic pump.

**Pressure Flow Studies: Determination of Pf=0**

Using the above procedure for creating transients, we were able to derive a pressure-flow curve for the coronary arteries, free of capacitance artifact and for a single state of coronary tone. The protocol was as follows: the pump was set to deliver coronary flow into the first reservoir at a fixed rate. The pump was adjusted so that a pressure of between 100 and 150 mm Hg perfusion pressure was realized. When a steady state had been reached, the second reservoir was pressurized to a pressure different from the perfusion pressure. The heart was then stopped and a pressure transient was performed as described above. Blood flow measurements were taken 500 msec after the transient, since the capacitive overshoot was finished at that time, but autoregulatory changes in tone had not yet begun. The pacing was then resumed and the tubing between the two reservoirs was reclamped. With the return to the pretransient flow rate, perfusion pressure would usually reestablish itself within 20–30 seconds. After recovery, the procedure was repeated, but with a different pressure in the second reservoir. After 5–10 repetitions, the flow measurements were plotted against the perfusion pressures to generate a pressure flow curve. The pressure axis intercept (Pf=0) was then noted. This procedure was repeated in each animal but with a starting pressure of only 50 mm Hg to produce maximal dilation of the coronary bed (Downey et al., 1976). Loss of tone was verified by the absence of hyperemia following a 20-μg bolus of adenosine into the coronary perfusate.

We also measured coronary pressure-flow curves in

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Circulation Research /Vol. 55, No. 6, December 1984
these animals with a falling pressure method, as was done by Bellamy (1978). This was accomplished by stopping the heart and then opening a small valve in the top of the Windkessel reservoir so that pressure would fall at an initial rate of 25 mm Hg per second. This yielded a rate of decline similar to the fall in aortic pressure with cardiac arrest which Bellamy employed. Flow points during the falling pressure phase were then plotted against the perfusion pressure to generate a second set of pressure-flow curves for these hearts. This protocol was repeated both in the presence and the absence of coronary tone.

**Estimation of Capacitance**

Figure 1A shows a simple model of the coronary capacitance. The model assumes that all capacitance can be lumped into one site, C, with a proximal resistance, R1, and a distal resistance, R2. Figure 1B shows that this model will respond to a transient change in perfusion pressure, P1 to P2, with an overshoot in flow, Q2, and a decay back to a stable value, Q3, with a time constant, T. Panel C of Figure 1 indicates that there are unique solutions for R1, R2, P_{n=0}, and C based on the parameters in panel B.

The derivation of the equations is as follows. Flow across R1 will be equal to (P1-P_{n=0})/R1. Under steady state conditions, this will be equal to Q1. When pressure is abruptly increased, as shown in Figure 1, P_{n=0} is incapable of changing instantly. Thus, Q2 reflects a new gradient across R1 (P2-P_{n=0}). R1 can then be solved by solving the following two simultaneous equations for R1.

\[
Q1 = \frac{(P1-P_{n=0})}{R1} \\
Q2 = \frac{(P2-P_{n=0})}{R1}
\]

P_{n=0} subsequently rises as the capacitance charges. A zero flow intercept, P_{n=0}, can then be calculated from the steady state flow, Q1 and Q3, and P1 and P2. Finally, R2 is calculated by determining the overall resistance, (P1-P_{n=0})/Q1, and subtracting R1.

The records from the pressure transient experiments described above were analyzed to determine the values of R1, R2, P_{n=0}, and C by this method. It has been our experience (see Fig. 5), and that of others (Bellamy, 1978; Klocke et al., 1981; Dole and Bishop, 1982), that the dynamic pressure-flow curves in the coronary artery are slightly convex to the pressure axis, rather than perfectly straight. The equations in Figure 1 assume a linear relationship, and, therefore, the P_{n=0} values calculated by this protocol will consistently be somewhat overestimated. That imprecision is estimating P_{n=0} will have only a small effect on the estimate of capacitance (a 100% overestimate of P_{n=0} would cause only a 20% overestimate of C). We therefore feel that this simplification is justified for deriving a first order approximation of coronary capacitance.

**Constant Flow Perfusion**

Several experiments were performed under conditions of constant flow. This was accomplished by placing a screw clamp on the perfusion line between the Windkessel and the coronary cannula. With the pump engaged, the clamp was tightened until pressure in the Windkessel exceeded 300 mm Hg. Under these conditions, flow into the coronary cannula had less than 5% variation between systole and diastole.

**Computer Models**

To determine the role of capacitance in the coronary system better, we performed computer simulations of the coronary bed to see if we could duplicate the phasic blood flow profiles which were obtained from the animal experiments. These simulations were executed on a Commodore PET microcomputer programmed in BASIC. Graphic results were output through a D to A converter (Downey and Rodgers, 1981) driving a storage oscilloscope. Figure 2 shows the basis of the model. Ten legs represent blood vessels at different depths in the myocardium. R1 represents resistance proximal to the capacitance site and R2 represents resistance distal to it. C is the capacitance and is between the junction of R1 and R2. The battery on the other side of the capacitor represents the intramyocardial pressure at that depth, since this is the pressure surrounding the vessel. The voltage of the battery at time t was set to equal K \times P_v(t). K is a constant ranging from 0 at the outer leg to 1 at the inner leg and calculates intramyocardial pressure at that depth, since this is the pressure surrounding the vessel. The voltage of the battery at time t was set to equal K \times P_v(t). K is a constant ranging from 0 at the outer leg to 1 at the inner leg and calculates intramyocardial pressure as a fixed percentage of ventricular pressure, P_v(t) in each depth. The outflow of R2 passes through a diode and is connected to the battery, as well, to simulate a vascular waterfall (Downey and Kirk, 1975).

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**Figure 1.** Panel A: the coronary system is assumed to approximate an R-C-R network where R1 is the resistance proximal to the capacitance site and R2 the distal resistance. Panel B: a record showing the coronary flow response to a step change in perfusion pressure for the above model. Panel C: R1, R2, and C can be solved by these equations from the parameters obtained in panel B.
A more sophisticated model of the coronary capacitance incorporating multiple parallel legs, each experiencing a different tissue pressure. Both vascular waterfalls and capacitance are incorporated into each leg of the model.

The model was examined in three basic forms. In the first form, C was set to zero so that only vascular waterfalls would be present. This is the model originally presented by Downey and Kirk (1975). In the second form, the outflow of R2 was set to zero to eliminate waterfalls. This is essentially the model presented by Spaan et al. (1981a, 1981b). In the third form, the model was run as shown in Figure 2 so that both capacitance and vascular waterfalls would be present in a hybrid model.

Results

Animal Group Assignments

Twenty-eight dogs were used in this study. Table 1 shows the blood pressure for these dogs (systolic/diastolic) and to which protocols each dog contributed data. Eight of the dogs were technically unsatisfactory or died prematurely and thus did not contribute data.

Phasic Coronary Flow in the Beating Heart

When we perfused the coronary vessels with a nonpulsatile perfusion pressure, a flow profile similar to that shown in panel A of Figure 3 resulted. The onset of systole was associated with a transient period of reduced or retrograde flow with a recovery toward a positive value late in systole. When perfusion pressure was lowered to 60 or 70 mm Hg, this retrograde transient was exaggerated. The fact that this flow can become retrograde means that at least part of the negative transient that occurs during systole must be related to capacitance, since vascular

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P/F = pressure-flow, CAP = capacitance measurement, Occl = sudden occlusion, AOP = aortic pressure (systolic/diastolic) in mm Hg.

* x indicates protocols to which each dog contributed data. (Eight animals were technically unsatisfactory or died prematurely.)
Panel A shows the phasic coronary flow which results when the coronary artery is perfused with a nonpulsatile pressure. Note the transients in flow which occur in early systole and early diastole. Panel B shows signal-averaged flow tracings from three different heart rates superimposed. Note that the diastolic flow profile is independent of heart rate.

Coronary Flow in Response to Pressure Transients

In the above experiment, blood flow transients related to vascular capacitance were a result of changes in the intramyocardial pressure which limits the response to the smaller intramural blood vessels. To see the extent to which the large epicardial arteries contribute to the total coronary capacitance, we measured the blood flow response to a step in perfusion pressure, since that would involve both vessel types. Any differences could then be attributable to the epicardial artery compliance. Figure 4 shows a typical experiment. In this experiment, the heart was stopped and perfusion pressure abruptly changed. Note that the abrupt fall in pressure was associated with a retrograde blood flow which then exponentially decayed to a steady state. Note also that the rate of the decay in this record was virtually identical to the decay seen during diastole while the heart was beating.

In these experiments the pressure transients were both negative-going and positive-going. When tone was present, there was no difference between time constants calculated with positive-going transients, 77.44 ± 4.5 msec, and negative-going transients, 76.4 ± 4.1 msec. These data are based on 17 observations in four dogs. With maximal dilation, however, there did seem to be a difference. Transients toward higher pressures resulted in time constants of 67.4 ± 3.8 msec, whereas those toward lower pressures yielded time constants which were higher, 124.7 ± 14.6 msec. These data are based on 18 observations in four dogs. With maximal dilation, at least, the time constant seemed to be greater when lower perfusion pressures were involved.

These time constants were in the same range as those associated with diastole in the beating heart, so our conclusion is that the epicardial vessels contribute little to the overall coronary artery compliance.
Pressure-Flow Curves

Pressure-flow curves were obtained as outlined in the methods from seven dogs. Curves generated by the step method indicated a zero flow pressure ($P_f=0$) of 29.6 ± 2.85 mm Hg when tone was present. This fell to 18.44 ± 1.31 mm Hg in the presence of adenosine dilation or hypoperfusion. A paired t-test indicated that these differences were highly significant, $P < 0.01$. When $P_f=0$ was estimated by the falling pressure method, the values were much higher. $P_f=0$ by that method was 41.8 ± 5.1 mm Hg when tone was present, and fell to 23.6 ± 1.14 mm Hg when tone was absent. The falling pressure measurements were, without exception, 5–15 mm Hg higher ($P < 0.02$) than those measured by the step method in the same heart. Figure 5 shows a typical experiment. The solid figures are determinations by the step method, while the open figures are derived by the falling pressure method.

Estimates of Capacitance

Perfusion pressure transients were analyzed by the equations presented in Methods, to determine proximal resistance, $R_1$, distal resistance, $R_2$, $P_f=0$, and capacitance, $C$. Eighty measurements were made in 13 dogs, 40 when tone was present and 40 while tone was abolished.

Measurements were made with both rising and falling pressures and are reported separately in

![Figure 4. An original record in which coronary perfusion pressure was abruptly dropped from $P_1$ to $P_2$. Note the transient undershoot in flow.](http://circres.ahajournals.org/)

![Figure 5. Pressure-flow curves from the coronary artery of a typical experiment. The solid symbols represent points taken following step changes in pressure after the flow transients had decayed. The open symbols represent those derived from 25 mm Hg/sec ramps in perfusion pressure. The displacement of the two curves is thought to be due to a capacitance artifact inherent in the ramp method.](http://circres.ahajournals.org/)
 capacitors were made small enough to give a time constant less than one beat. Although this and R2. Capacitance was made small enough to give a time constant less than one beat. Although this was determined in the previous section where the entire curve was constructed. Table 2 indicates, however, that capacitance was tone dependent on the direction of the pressure transient and averaged about 0.0065 ml/mm Hg per 100 g. The capacitance value approximately doubled with maximal dilation, and the magnitude seemed to be dependent on the direction of the pressure transient, although only marginal significance was determined for that difference. The Pf=0 values in Table 2 were calculated from only two points on the pressure flow curve and were linearly extrapolated to the pressure axis. Since we find these curves to be consistently convex to the pressure axis (see Fig. 5), Pf=0 determined by this procedure will consistently overestimate the Pf=0 as determined in the previous section where the entire curve was constructed. Table 2 indicates, however, that reasonable values for Pf=0 were used for the capacitance calculations, although they were somewhat higher than were determined in the previous section. It is interesting to note that the Pf=0 determined by this less accurate method were, nevertheless, significantly higher when coronary tone was present than when it was absent. The site of the capacitance was also tone dependent. With tone present, the downstream resistance R2 accounted for about 60% of the total resistance. With dilation, however, R2 decreased more than R1, so that it accounted for only 40% of the resistance.

Table 2. When one dog yielded more than one value, these values were averaged such that n equaled the number of dogs studied for the statistics. Since all 13 dogs did not contribute to all four data groups (only seven contributed to all four groups) an unpaired t-test was used to test for significance between the groups. The results appear in Table 2. Note that capacitance with tone present was independent of the direction of the pressure transient and averaged about 0.0065 ml/mm Hg per 100 g. The capacitance value approximately doubled with maximal dilation, and the magnitude seemed to be dependent on the direction of the pressure transient, although only marginal significance was determined for that difference. The Pf=0 values in Table 2 were calculated from only two points on the pressure flow curve and were linearly extrapolated to the pressure axis. Since we find these curves to be consistently convex to the pressure axis (see Fig. 5), Pf=0 determined by this procedure will consistently overestimate the Pf=0 as determined in the previous section where the entire curve was constructed. Table 2 indicates, however, that reasonable values for Pf=0 were used for the capacitance calculations, although they were somewhat higher than were determined in the previous section. It is interesting to note that the Pf=0 determined by this less accurate method were, nevertheless, significantly higher when coronary tone was present than when it was absent. The site of the capacitance was also tone dependent. With tone present, the downstream resistance R2 accounted for about 60% of the total resistance. With dilation, however, R2 decreased more than R1, so that it accounted for only 40% of the resistance.

Results with the Model—Phasic Flow Prediction

Three forms of the model were examined to see which, if any, could predict a phasic coronary flow profile similar to that actually observed when the coronaries were perfused with a nonpulsatile pressure. Figure 6A shows the capacitance-only model, using a small capacitance and equal values of R1 and R2. Capacitance was made small enough to give a time constant less than one beat. Although this profile is similar to the real coronary flow profile, it is different in that both the systolic and the diastolic overshoots decay back to the same base line. When perfusion pressure was lowered (not shown), the systolic flows could become retrograde, as was seen in the animals. Figure 6C shows the result of increasing the time constant to 3 seconds as suggested by the reports of Spaan et al. (1981a, 1981b). Because no appreciable decay can occur during systole or diastole, the flow profile becomes a mirror image of the ventricular pressure, and the transients are completely absent. Thus, a pure capacitance model was incapable of duplicating the phasic flow profile, regardless of the magnitude of the capacitance.

When only waterfalls were incorporated in the model, the flow profile seen in Figure 6B resulted. In this case, no transients were ever seen, and flow during systole was always positive, regardless of the perfusion pressure.

Figure 6D shows the results when both waterfalls and capacitance are present in the model. Note that the overshoots now decay back to a plateau late in diastole. Furthermore, the asymptote for the systolic decay phase is different from that for the diastolic phase. In addition, retrograde flows occurred in systole when perfusion pressure was lowered (not shown). When the time constant was lengthened to 3 seconds in this model, the flow profile seen in the pure capacitance model with a long time constant (Fig. 6C) was recreated. Furthermore, examination of flows through individual legs revealed that systole did not selectively inhibit mean flow to the inner legs. Only the waterfall-capacitance model with short time constants faithfully reproduced the phasic flow profile seen in the dogs.

Results with the Model—Load Line Analysis

In their original paper, Spaan et al. (1981a) used a load line analysis to estimate the magnitude of R1 and R2 and to support the existence of intravascular capacitance linked to intramyocardial pressure. Basically, the phasic component of the flow signal was plotted against the phasic component of the pressure signal. This established a load line for the upstream resistance, R1. When we plotted load lines from the
two models, we found that both capacitance and waterfall models produced load lines compatible with the animal data. One difference was seen, however, between these two divergent models. The load line for the pure capacitance model was independent of the mean perfusion pressure, whereas that for a waterfall model was not. As mean perfusion pressure fell below the peak tissue pressure, a family of load lines was generated, as shown in upper panel A of Figure 7. Panel B shows what a plot of phasic perfusion pressure under nonpulsatile flow conditions (the pressure axis intercept in the upper graph) would look like when plotted against mean perfusion pressure for the two models. Notice that the phasic pressure is dependent on perfusion pressure only in the waterfall model. To test this, we perfused the dog's coronary arteries under constant flow conditions, as outlined in Methods. The pump speed then was gradually decreased so that the mean flow rate and perfusion pressure fell off as a ramp (see Fig. 8). When this was done, the phasic perfusion pressure signal was always (observations from six dogs) seen to narrow markedly as mean pressure fell below peak ventricular pressure, as is shown in Figure 8. This result is compatible only with a model incorporating vascular waterfalls in addition to capacitance.

**Discussion**

We believe that the blood flow transients which we describe are capacitance related because: (1) retrograde flow could be observed in the systole transient when perfusion pressure was low, (2) the diastolic decay approximated an exponential as would be predicted by a capacitive discharge, and (3) the diastolic transient had a time constant exactly equal to that resulting from a step change in perfusion pressure, an unambiguous capacitive response. It is surprising to us that the capacitive component in the phasic coronary blood flow, which we have described here, has gone unnoticed for so many years. The reason for this probably lies in the fact that these transients are normally masked when the coronaries are perfused with aortic pressure. That is because, during both early diastole and early systole, the rate of change of the blood pressure is high and in a direction opposite to the capacitive transients. It is only when the coronary vessels are perfused from a nonpulsatile source that these transients are evident. Spaan et al. (1981a, 1981b) perfused the coronary arteries with a nonpulsatile pressure for their studies, and their published records clearly reveal overshoots identical to ours.

A proper understanding of coronary capacitance is vital to proper interpretation of many aspects of coronary hemodynamics. For example, Dennison and Green (1958) suggested that true coronary resistance, and thus the degree of coronary tone, could best be estimated by dividing late diastolic aortic pressure by late diastolic coronary blood flow. Unfortunately, that concept may have two fundamental flaws in it. First, several reports indicate that the pressure-flow curve for the coronary system has a non-zero intercept (Bellamy, 1978; Dole and Bishop, 1982; Klocke et al., 1981; Sherman et al., 1980). If that were the case, coronary resistance would obviously be dependent on both the perfusion pressure
FIGURE 7. Panel A shows load line plots of the phasic coronary pressure vs. phasic coronary flow. Whereas the load line for a pure capacitance system is independent of the mean perfusion pressure, a waterfall system generates a family of load lines as mean perfusion pressure falls below the peak-tissue pressure. Panel B shows what a plot of phasic coronary pressure (nonpulsatile flow conditions) would look like when plotted against mean coronary pressure for the two systems.

and the state of tone. Such a quantity would have little practical use. Second, it has been suggested that the phasic variation between systolic and diastolic flow rates derives entirely from coronary capacitance (Spaan et al., 1981a). If that were true, then the relatively high inflow rate during diastole would represent filling of some capacitance sites rather than resistive flow across the coronary capillaries.

The second objection seems to be obviated by our data. Although diastole does seem to be a time of capacitance filling, this process is nearly complete by the end of diastole, even at heart rates in excess of 150 beats/min. When diastole was made indefinitely long by cardiac arrest, no longer time constants could be found.

Our data also shed light on the former objection, as well. Bellamy (1978) originally presented data that indicated that, during diastole, the pressure-flow relationship for the coronary system had a positive pressure intercept which was an appreciable fraction of the blood pressure. Furthermore, this intercept was reported to be in proportion to coronary tone. That concept has recently been challenged by Eng et al. (1981). They argued that the falling perfusion pressure employed by Bellamy to produce a range of pressure-flow values was associated with a concealed back flow from the coronary capacitance. Thus, when forward capillary flow equaled capacitive back flow, flow in the coronary ostium would have ceased, even though flow in vessels distal to the capacitance would continue. Three groups, Eng et al. (1981), Klocke et al. (1981), and Dole et al. (1982), have employed a step pressure technique similar to the one reported here to determine $P_f=0$ in a manner that would be free of capacitance artifact. Interestingly enough, they have arrived at divergent results. Whereas Eng et al. (1981) found $P_f=0$ to be small and independent of tone, the findings of the latter two groups were virtually identical to ours.

The study by Klocke et al. (1981) has been criti-
cized on the grounds that they perfused only the left circumflex branch (Eng and Kirk, 1982b). Collaterals from the LAD and circumflux branches could have provided a peripheral coronary pressure which could have been confused with the $P_{f=0}$. The present protocol avoids that objection, since the entire left coronary artery was cannulated, making the small right coronary the only possible source of collateral flow. We have no explanation for why the Eng et al. (1981) observation is at such variance. However, in our laboratory, like that of Klocke et al. (1981) and Dole and Bishop (1982), the pressure intercepts were both high and clearly tone dependent.

Our results would indicate that there is only about a 10 mm Hg artifact in estimating the pressure intercept by Bellamy’s falling pressure method. Recent papers by Canty et al. (1982) and Kirkeeide et al. (1981) would corroborate that finding. We therefore conclude that the resistance derived by late diastolic pressure–flow calculations will not only be pressure dependent, but tone dependent as well, and, therefore, virtually impossible to interpret.

All of the present data strongly point to a capacitive time constant in the coronary system of 100 msec or less. Why then did Spaan et al. (1981a) arrive at such a large figure? Those figures were derived from experiments in which the coronary artery was suddenly occluded. Occlusion produced a rapid fall in peripheral coronary pressure to about 30–40 mm Hg, and then a gradual decline over the next few seconds. The original interpretation of this response was that, on occlusion, the peripheral coronary pressure would immediately fall to the pressure at the node between the capacitor and the resistors. The capacitance would then discharge into the veins with a time constant of the capacitance times the distal resistance. Consider another possibility, however. Suppose the rapid component included the short capacitive time constant. In that case, peripheral coronary pressure would fall to the critical closing pressure $P_{f=0}$ rather than $P_N$. The ensuing ischemia would cause a rapid dilation over the next few seconds such that the $P_{f=0}$ would progressively fall. Peripheral coronary pressure would then follow this pressure over that period. We, therefore, must consider the possibility that the time constant measured by Spaan et al. (1981a) reflected a smooth muscle event rather than discharge of capacitance.

One must finally ask, then, what is the magnitude and location of the coronary capacitance. Spaan et al. (1981a) put the capacitance far down in the microcirculation using load line analysis such that R1 was two-thirds of the total coronary resistance. Those calculations may contain some error, how-
ever, since their model assumed that all of the pressure variations observed were from capacitance and none from waterfalls. Similarly, Canty et al. (1982) assumed that most capacitance is in the large coronary arteries, but their analysis did not really allow for a proximal resistance. The concept that the epicardial vessels are the major site of the coronary capacitance is disputed by two of the present observations.

First, capacitance-related flows result from a change in the pressure across the walls of the capacitance vessels. When perfusion pressure was held constant, capacitance-related transients were seen both in the early part of diastole and systole. We propose that these transients result from sudden changes in the intramyocardial pressure surrounding the capacitance vessels associated with the onset of systole and diastole. Because the epicardial vessels are physically located outside of the ventricular muscle, the pressure surrounding them should be unchanged through the cardiac cycle. Thus, the epicardial vessels should not have contributed to the capacitive transients seen with constant pressure perfusion. When the coronary pressure was abruptly changed, however, all of the coronary vessels should contribute. Yet, the time constant for decay following a pressure step was identical to that seen in diastole with constant pressure perfusion. We, therefore, conclude that the epicardial capacitance is small compared to that in the deep vessels.

Second, the transient pressure analysis also indicated that the epicardial vessels are not the major site of the coronary arterial capacitance. Our transient pressure method of solving for R1 and R2 places the capacitance about midway in the coronary circulation. Whether our assumption of lumping all of the capacitance, some of which must exist in every size vessel, into one site can be justified remains to be seen. Clearly, the epicardial coronary vessels do possess some capacitance, as has been demonstrated by Chilian and Marcus (1982). The magnitude of the epicardial capacitance must be considerably less than that in the intramural vessels, however, not to be detected by the present studies. A great deal of capacitance probably resides in the coronary veins, as well. Unfortunately, venous level capacitance is difficult to measure with our methods, since the transients in perfusion pressure will be severely attenuated in the veins. This insensitivity to capacitance at the distal reaches of the coronary circulation is admittedly a shortcoming of the present method. For now, however, a midlevel capacitance site seems to be a good first approximation. We conclude, then, that Spaan et al. were correct in placing the capacitance site deep in the microcirculation.

As for the magnitude of the capacitance, again, there is confusion in the literature. Whereas Spaan (1982) reports a value of 0.1–0.25 mg/mm Hg per 100 g, Canty et al. (1982) report a much lower value of about 0.001–0.011. That is two orders of magni-
unexplainable. For example, a pure capacitance system cannot reduce mean flow, it can only make it pulsatile. Thus, the observation that coronary flow abruptly increases when the heart is arrested (Sabiston and Gregg, 1957) is very difficult to explain with a pure capacitance system. It is compatible, however, with a waterfall capacitance model. Similarly, whenever diastolic perfusion is selectively reduced, blood flow to the subendocardium is preferentially curtained (Buckberg et al. 1972; Downey and Kirk, 1974). A capacitance system with a time constant of 3 seconds would not allow the phasic perfusion variations between systole and diastole to be transmitted to the microcirculation. Rather, they would be damped out by the long time constant. Numerous other examples exist. Only when Spaan's system is modified to incorporate shorter time constants and vascular waterfalls is it compatible with these observations.

In conclusion, it appears that an appreciable capacitance of about 0.005 to 0.03 ml/mm Hg per 100 g of myocardium exists midway between the coronary ostium and the coronary sinus. This capacitance is very sensitive to the intramyocardial pressure, and it charges and discharges with each beat, because the time constant for charging this capacitance is short (<100 msec). However, the coronary vessels are near volume equilibrium most of the time. Such a short time constant means that vascular waterfalls have to be important determinants of coronary blood flow.

We wish to thank Cindy Simmons for her technical help and Sandy Worley, Leigh Cooper, and Patricia Gragg for their help in preparing this manuscript.

Supported by a Grant-in-Aid from the American Heart Association. Address for reprints: James M. Downey, Ph.D., Department of Physiology, College of Medicine, University of South Alabama, Mobile, Alabama 36688.

Received January 10, 1983; accepted for publication August 22, 1984.

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INDEX TERMS: Capacitance • Vascular waterfall • Critical closing pressure • Load Line • Coronary hemodynamics • Intramyocardial pressure
The role of vascular capacitance in the coronary arteries.

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Circ Res. 1984;55:751-762
doi: 10.1161/01.RES.55.6.751

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