Effect of Atrial Systole on Canine and Porcine Coronary Blood Flow

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SUMMARY A feature of phasic coronary flow patterns recorded in conscious chronically instrumented dogs is the atrial cove—a transient depression of arterial flow that occurs during atrial systole. The association between the hemodynamic effects of atrial systole and the atrial cove was studied in anesthetized dogs and pigs with complete heart block. Many atrial coves are available for study in these preparations because atrial activity continues unabated during the diastolic ventricular arrest that follows cessation of electrical pacing. The effect of atrial systole is to translate the pressure-flow relation found during diastole to a higher intercept pressure without change in slope. The increase in the intercept pressure equals the increase in intramyocardial pressure measured with microtransducers embedded in the left ventricular wall. The decrement in flow during the atrial cove is a direct function of the change in intramyocardial pressure and an inverse function of coronary vascular resistance. Each atrial systole is associated with a forward flow transient in the coronary veins, the peak of which occurs at the same instant as the nadir of atrial flow. These data suggest that the coronary vessels are acting as collapsible tubes and that the waterfall model of the coronary circulation is applicable. The following sequence is proposed to account for the atrial cove. Atrial systole ejects a bolus of blood into the left ventricle increasing both ventricular cavity and intramyocardial pressures. The increase in intramyocardial pressure raises the back pressure opposing coronary flow, reducing the arterial perfusion pressure gradient and causing flow to fall. Circ Res 49: 701–710, 1981.

Methods

A variety of acute experiments were performed upon anesthetized dogs and pigs to determine whether or not atrial coves were artifacts and, if not, to ascertain the relationship between the hemodynamic events associated with atrial systole and the decrement in coronary flow that occurs during the atrial cove. It was considered more efficacious to perform data analysis upon the many atrial coves that occur during the ventricular diastolic arrest which follows cessation of electrical pacing in dogs with complete heart block than to confine data collection to the single atrial cove that occurs per beat in dogs with normal conduction.

Most experimentation was done on dogs, the basic preparation being as follows. Dogs (n = 21; average weight 18.7 kg) were induced with 4% surital, intubated, and ventilated (Ohio Anesthesia Ventilator) with O2 and halothane (0.5–1%). Ventilatory parameters were adjusted to maintain normal blood gases and pH. Aortic root pressure was measured with either a Millar 5F catheter pressure transducer or a fluid-filled catheter inserted via the right carotid artery exposed in the neck. Fine wire electrodes were inserted into the vagi in some animals so that, when desired, the atria could be arrested by vagal stimulation (Grass S88 Stimulator and Grass SIUS Stimulus Isolation Unit; frequency 20–40 pulses/sec; 20–40 V). The right side of the chest was opened through an incision in the 4th intercostal space, the pericardium was incised, and a fluid-filled catheter or Millar 5F catheter pressure transducer was inserted through the atrial appendage into the right atrium. Electrodes were sutured
FIGURE 1 Typical atrial coves as they appear in a conscious, chronically instrumented dog. Panel A shows resting (right) and reactive hyperemia (left) beats recorded in the anterior descending branch of the left coronary artery (LAD). Vasodilation is associated with an increase in the decrement of flow during the cove. Panel B shows pressure (CIRP) and flow (CIRQ) recorded in the circumflex branch of the same animal. The flow tracing begins at the end of a short coronary occlusion which was used to establish a reference for zero flow. Flow during the atrial cove of the last beat appears to be zero. When diastolic flow prior to the atrial cove is plotted as a function of pressure, zero flow occurs at an arterial pressure of 45 mm Hg.

into the right ventricle so that the heart could be electrically paced when needed (rate 120-150 beats/min; Grass S9 Stimulator). The atrioventricular node was destroyed by direct injection of 10% formaldehyde (Steiner and Kovalik, 1968). In four animals in which this procedure was not successful, the superior and inferior vena cava were occluded, the right atrium was opened, and the region of the atrioventricular node was suture ligated. The atrium was closed and the circulation re-established after about 1 minute of arrest. It was found that the probability of obtaining diastolic arrests free from ventricular ectopy was enhanced by intravenous infusion of 10-20 mg of lidocaine prior to cessation of electrical pacing.

The right thoracotomy was closed, the animal was placed in the right lateral decubitus position, the left side of the heart was exposed through an incision in the 5th intercostal space, and the pericardium was incised. In five animals a Millar velocity/pressure transducer model VPC 684T was inserted via the left atrial appendage and positioned so that the distal pressure transducer was in the ventricular cavity and the proximal pressure transducer and velocity sensor were at the level of the mitral valve ring. A wide bore catheter was placed in the left atrium in five animals so that previously removed, warmed, and heparinized blood could be injected rapidly into the left heart.

Depending upon anatomical suitability, coronary flow was measured in either the circumflex or descending branch of the left coronary artery using Carolina Medical Electronics model EP406-410 RC electromagnetic flow probes and a Carolina Medical Electronics two-channel electromagnetic flowmeter. Considerable effort was made to ensure a stable and reproducible flow zero. To study the possible influence of contact between the electromagnetic flow probe and the underlying heart, an extracorporeal shunt fabricated from a 12-gauge Teflon Angiocath, 5-mm vinyl tubing, and a Carolina EP 608 flow probe was inserted between the left subclavian artery and the anterior descending branch in three animals. The total length of the shunt was about one-third of a meter. Prior to insertion of the shunt, the dogs were given heparin, 3 mg/kg. Flow in the great cardiac vein was measured in six animals. When a large vein embedded in fat was present, dissection was limited to the minimal amount that would allow contact between a Carolina model EP 108 flow probe and the vein wall. The surrounding fat was found to mechanically stabilize this
narrow, light weight flow probe. When the great cardiac vein was only loosely attached to the deeper structures, a Carolina model EP 406 RC flow probe was used. The broad jaws of this device enclosed a sufficient length of vein to allow the maintenance of a stable flow signal.

Intramyocardial pressure was measured in the left ventricular wall with a Millar 4F catheter pressure transducer. The catheter was inserted through a small epicardial incision into a tract made with a 1-mm probe. The catheter was passed obliquely into the myocardium in the distribution of the appropriate-artery and to the depth equal to about one-half of the full thickness of the wall. Exact wall position was determined at the end of the experiment. The catheter was usually sutured in place with a 5-0 suture. In three dogs, two catheters were employed, and an attempt was made to demonstrate regional and transmural differences in intramyocardial pressure during atrial systole. The calibration signal of the Millar transducer was compared to a mercury manometer before or after each experiment.

Coronary arterial and venous pressures were measured by using 22-gauge Teflon catheters positioned so that their orifices faced downstream. Pressure from fluid-filled catheters was measured with Statham P23ID strain gauges. Care was taken to ensure that the strain gauge was at the same height as the vissus in which pressure was measured. All data were recorded by a Honeywell Visicorder with a paper speed of 1.6–8 inches/sec.

Data collection was designed to study the relation between the decrement in flow during the atrial cove and such hemodynamic variables as atrial, ventricular, and intramyocardial pressures and coronary venous pressure and flow. In each preparation usually more than one variable was measured during the diastolic arrest that followed cessation of electrical pacing. In four dogs, experiments were performed specifically to measure the effect of rapid infusion of blood into the left heart during diastolic arrest. In three dogs, calcium chloride (10% solution, 200-mg bolus) was infused intravenously to determine the effect of an inotropic agent on the atrial cove. The exact relation between the decrement in flow and the change in intramyocardial pressure was obtained by constructing coronary pressure-flow relations. Coronary flow was plotted as a function of aortic pressure at the nadir of flow during the atrial cove and at two points between each cove. The pressure-flow relation constructed from data measured between coves is referred to as the "diastolic relation." Data were obtained in some preparations at both resting level flow and at the peak of the reactive hyperemia that followed a 15-second occlusion of the coronary artery. The ventricular manifestation of atrial systole is referred to in the text as the "atrial kick." The average difference in height between the base and apex of all atrial kicks occurring during a given arrest was used to calculate the change in intramyocardial pressure.

A final group of experiments was performed on four pigs (average weight 23.2 kg) to determine the important of left-sided venous return on the origin of the arterial cove. Pigs were premedicated with zylazine HCl, 0.5 mg/kg; ketamine, 2 mg/kg; and atropine, 1 mg/kg. They were then made to breathe halothane by mask until an endotracheal tube could be inserted. They were ventilated with O2 and anesthesia was maintained with an intravenous drip of 4% surital. A median sternotomy incision was made and steps were taken for the institution of cardiopulmonary bypass: heparin, 3 mg/kg; aortic root cannulation; two right heart canulas; left ventricular sump; Sarns roller pump model 5000; and a Bentley pediatric bubble oxygenator primed with blood. A Carolina electromagnetic flow probe of proper size was placed around the descending branch of the left coronary artery. A Millar 4F catheter pressure transducer was placed in the myocardium near the artery. The atrioventricular node was destroyed by injection of 10% formaldehyde and the heart electrically paced. With the pulse generator off, aortic pressure, coronary flow, intramyocardial pressure, and ECG were recorded when the circulation was supported by the beating heart and when the circulation was maintained by total cardiopulmonary bypass with the left ventricle decompressed.

**Results**

Several incidental observations pertinent to the origin of the atrial cove can be directly stated. First, during diastole in dogs with complete heart block, a forceful thump synchronous with each atrial systole is palpable at the ventricular apex. Second, pulling the left atrial appendage away from the circumflex coronary artery does not alter the shape of the atrial cove. Third, occlusion of small atrial arteries that pass from the circumflex to the left atrium is without effect on the shape of the atrial cove.

Figure 2A addresses the question of whether or not atrial coves are instrumental artifacts perhaps caused by the flow probe being jarred by the heart. Atrial coves are clearly apparent in flow tracings made in such a way that the flow probe is not in contact with the surface of the heart and are thus unlikely to be artifacts. Figure 2B, and C, shows that, for atrial coves to occur, atrial systole must be coupled to the flow of blood from atrium into ventricle. When a heart with complete atrioventricular block is maintained on total cardiopulmonary bypass (only bronchial artery flow enters the left heart), the ECG P wave is associated neither with atrial kicks in the ventricular intramyocardial pressure tracing nor atrial coves in the coronary flow tracing (Fig. 2C). These observations suggest that atrial coves are not the result of deformation of...
pressures. In contrast to the solitary atrial systole per ventricular beat in a normally conducting heart, in dogs with complete heart block, the multiple atrial systoles that occur during diastolic arrest are associated with a to-and-fro motion of blood, and probably net transfer is small. The atrial cove follows the rise in intramyocardial pressure by 10-15

![Figure 2](image-url)

**Figure 2** Panel A: Atrial coves are apparent in a flow tracing made with a cannulating flow probe inserted as part of a shunt between the left subclavian artery and the mid-anterior descending coronary artery in an anesthetized dog. The pressure increment during the atrial kick is about 2.5 mm Hg. Panels B and C: Anesthetized pig preparation. The following have been recorded: ECG, flow in the anterior descending branch of the coronary artery (LADQ), intramyocardial pressure measured in the anterior wall of the left ventricle (LVIMP), and pressure in the aortic root (AP). Panel B: The circulation is supported by a beating heart. After each ECG P wave, there is an atrial kick in the LVIMP tracing and a well-formed atrial cove in the coronary flow tracing. Panel C: The circulation is supported by total cardiopulmonary bypass and the left ventricle is vented to the atmosphere. Neither atrial kicks nor atrial coves are apparent, even though the ECG shows P waves.

coronary vessels caused by tugging of the contracting atrium upon the atrioventricular valve ring with associated distortion of contiguous myocardium.

Figure 3 shows some of the hemodynamic events that accompany the atrial cove. Left atrial systole follows the P wave of the ECG by about 30 msec. A bolus of blood is ejected through the mitral valve raising left ventricular cavitary and intramyocardial pressures.

![Figure 3](image-url)

**Figure 3** Hydraulic events associated with the atrial cove. Two cycles are shown from a reactive hyperemia experiment recorded in a dog. VELOCITY MITRAL = flow velocity at the mitral valve ring. Flow during ventricular systole is taken to be zero and an upward deflection indicates flow into the ventricle. LVP = left ventricular cavity pressure. LVIMP = intramyocardial pressure in the anterior wall of the left ventricle. LADQ = flow in the anterior descending branch of the left coronary artery. GCVQ = great cardiac vein flow. RAP = right atrial pressure. LADP = pressure in the anterior descending branch of the left coronary artery. See text for discussion.
msec. Simultaneous measurement of flow in the great cardiac vein showed a transient increase in flow, the peak of which corresponded to the nadir of arterial flow. Although not shown in Figure 3, a positive pressure wave occurred in the coronary veins which was synchronous with and identical in height to the atrial kick. This coronary venous pressure transient originates upstream to the right atrium since its shape was not altered by occlusion of the coronary sinus. A transient also was apparent in the coronary artery pressure tracing: arterial pressure increased at the same instant as the atrial kick, but the magnitude was always smaller than that of the atrial kick. This arterial pressure transient arose downstream from the epicardial arteries because it was still present when the coronary artery upstream to the catheter was occluded. A pressure transient synchronous with the atrial kick was also occasionally apparent in the aortic pressure tracing. This was of more complex shape and lesser magnitude than the coronary artery pressure transient. Atrial coves were not apparent when the coronary artery was occluded either upstream or downstream from the flow probe.

Because of the difficulty in measuring small volumes, it was not possible to compare the decrement in arterial flow and the increment in venous flow. In general, the phasic flow signal recorded during long diastoles in the coronary veins showed less cove-to-cove variation than did the complementary arterial flow signal.

One assumption made in this study was that at least some of the atrial coves seen in animals with complete heart block are equivalent to the solitary atrial cove of a normally conducted beat. Figure 4A shows that, in fact, only atrial coves occurring during the first several seconds of diastole were likely to satisfy this assumption. When aortic pressure had fallen to 30-40 mm Hg, the typical "V"-shaped depression was replaced by a bifid flow pattern—an initial decrement in flow followed by a forward flow transient. The latter feature suggests a capacitance event, i.e., refill of vessels emptied by the atrial kick. A similar pattern was found in the vasoconstricted bed at higher aortic pressure. When the nadir of flow during the cove reached the zero flow baseline, and especially after back flow had occurred several times, the coves became smaller and disappeared. Finally, when aortic pressure had fallen to the level of ventricular chamber pressure, atrial systole caused the equivalent of a tiny ventricular systole: a pressure spike in the aortic root and a forward flow wave in the coronary artery.

The remainder of Figure 4 shows data descriptive of the dependence of the atrial cove on intramyocardial pressure. Attempts were made to deepen the atrial cove by strengthening the force of atrial systole. Although intraventricular and intramyocardial pressures could be increased by more than 10 mm Hg by rapid infusion of blood during a diastolic arrest, the magnitude of the atrial kick changed only slightly. However, as is apparent in Figure 4C, infusion of Ca²⁺ resulted in an increase in the force of atrial systole and a corresponding increase in the decrement of flow during the atrial cove. The phasic flow pattern of the atrial cove was exquisitely sensitive to slight changes in intramyocardial pressure.
The peculiar "step" appearance of the atrial coves in Figure 4D probably was caused by the delay in the fall in intramyocardial pressure that was apparent after each atrial kick. Figure 4E shows a pattern that was consistently found in one animal. Intramyocardial pressure oscillated after each atrial systole with coronary flow following each perturbation. Finally, Figure 4F shows that, when the vagi were stimulated, the atrial coves disappeared as atrial systole weakened.

The exact relation between the magnitude of the atrial kick and the decrement in coronary flow was studied by correlating the change in intramyocardial tissue pressure with the displacement of the coronary pressure-flow relation. The basic prerequisite for this type of analysis is a reproducible and stable reference for zero flow. This condition, which is necessary if data from multiple arrests are to be combined, was realized in only four dogs. An example of an individual arrest from one of these animals is shown in Figure 5. Noteworthy is the fact that the increase in intramyocardial pressure during the atrial kicks and the decrement in coronary flow during the atrial coves appear constant throughout the 8 seconds of arrest.

Figure 6 shows the combined pressure-flow data from five diastolic arrests in the maximally vasodilated anterior descending bed of another animal. Below an aortic pressure of 70 mm Hg, neither relation showed a statistically discernible departure from linearity (Farley and Hinich, 1970) and both had statistically identical slopes. However, the extrapolated intercept pressures differed at the $P < 0.05$ level ([t]-test for equality of parameters (Neter and Wasserman, 1974)). When aortic pressure was less than 70 mm Hg, the average increment in pressure during the atrial kick was $4.0 \pm 0.2$ mm Hg, which was similar to the horizontal separation between the two pressure-flow relations. In the other three animals for which the preparation of a similar diagram was possible, identical increases in intramyocardial pressure and the zero flow pressure intercept of the pressure-flow relation were also found ($2.5, 3.0, \text{and } 4.5$ mm Hg).

The absolute level of intramyocardial pressure approached but never equaled the extrapolated or observed zero flow pressure intercept during arrest. No regional or transmural differences in the shape or magnitude of the atrial kick could be detected. Because the electronic pressure zero of the Millar catheters was not always reliable, it was necessary to establish an atmospheric pressure zero at the beginning and end of each experiment.

Although the pressure-flow relations shown in Figures 5 and 6 are nonlinear only in their initial extent and although the decrement in flow during the atrial coves was uniform throughout, these were not the findings in all preparations. There were two

![Figure 5](http://circres.ahajournals.org/)

**Figure 5** An example of the phasic pressure and flow data that were used to study the relationship between the atrial kick and the atrial cove. The bed was vasodilated by a 15-second occlusion of the coronary artery. The lower portion of the figure shows the relation between circumflex coronary blood flow (CIR0) and aortic pressure (AP). Unfilled circles are data measured between coves; filled circles are data measured at the nadir of flow during the cove. The horizontal separation between the two pressure-flow relations equals the average increment in intramyocardial pressure (IMP) during all the atrial kicks (~4.5 mm Hg).
circumstances in which curvilinearity was marked and atrial coves of nonuniform depth were common: (1) when diastolic arrest exceeds 10 seconds in a grossly dilated heart—especially noticeable when the heart had herniated through the pericardial incisions, and (2) when the heart was arrested prior to first obtaining maximum vasodilation in the coronary bed. In the first instance, pressure-flow relations were concave to the flow axis over most of their extent and arterial flow stopped at an arterial pressure that did not exceed right atrial pressure. The decrement in flow during the atrial cove in the grossly dilated heart became less pronounced as flow approached zero, and only part of this change could be accounted for by a weakening of atrial systole.

The size and shape of the atrial cove were also changed when the heart was arrested without first

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

**Figure 6.** Data from five diastolic arrests similar to the single arrest of Figure 5 have been combined. Flow was measured in the anterior descending branch (LAD) during reactive hyperemia. Regression equations were calculated for the data found below an aortic pressure of 70 mm Hg. The data of the atrial coves were fitted to the model \( y_k = -50.19 + 2.62 x_k \), \( r = 0.96 \). The data of diastole were fitted to the model \( y_k = -39.17 + 2.62 x_k \), \( r = 0.95 \). \( y_k \) is the \( k \)th flow measurement and \( x_k \) is the corresponding pressure measurement. At any given level of flow, the horizontal separation between the two pressure-flow reactions (42 mm Hg) equals the mean increase in intramyocardial pressure during the atrial kicks (4.0 ± 0.2 mm Hg). The average right atrial pressure at the end of the five arrests was 7 ± 1 mm Hg. Right atrial systole increased this pressure by an average of 3 ± 1 mm Hg.

**Figure 7.** The effect of vasodilation on the decrement of flow during the atrial coves. Pressure-flow relations and a portion of the phasic flow tracings are shown for the two beats: left side, high flow reactive hyperemia beat; right side, low flow resting beat. The atrial kicks were of identical magnitude in both beats. The phasic flow tracings show that the atrial coves were initially much deeper in the vasodilated bed. The similar horizontal separation between the two pairs of pressure-flow relations may result from the similar increases in intramyocardial pressure (\( \Delta IMP \)) that occurred during the atrial kicks. The vertical separation between the relations is the decrement in flow during the atrial coves (\( \Delta Q_{AC} \)) and is greater in the vasodilated bed because of the greater slope of this relation compared to the relation for the vasoconstricted bed. Note that the pressure-flow relation for the resting flow beat is curvilinear toward the end of the diastole and that the depth of the coves increases as aortic pressure falls. This change may signify autoregulation, i.e., a fall in vascular resistance.
vasodilating the coronary bed. Although the initial atrial coves in the vasoconstricted bed were smaller than those seen during reactive hyperemia (Fig. 7), as time passed in the arrest there was an increase in the depth of the cove, as well as a simultaneous departure of the pressure-flow relation from linearity. It was more difficult to study atrial coves in the vasoconstricted bed than when reactive hyperemia was present because pressure-flow relations in the vasoconstricted bed were not easily reproduced. Slight changes in vasomotor tone and small differences in the zero flow baseline from one arrest to the next caused such overlap of data that it was impossible to combine multiple arrests and to separate the pressure-flow relations describing diastole and the atrial coves.

Discussion

It has been known for some time that atrial systole has effects in addition to causing an increase in late diastolic ventricular volume. Piemme and Dexter (1963) have demonstrated the existence of low amplitude positive pressures waves during diastole in the aorta and have suggested that they are caused by vibration of the heart induced by atrial systole. Although such pressure perturbations were seen only infrequently in this study, it is pertinent to inquire whether or not they might have some connection with atrial coves. Pressure waves arising from the heart might be expected to propagate downstream, be reflected by the peripheral vascular bed, and reappear as a positive pressure wave in the aortic root where they might affect coronary flow. Be that as it may, such pressure perturbations are unlikely to be the source of the atrial cove because a positive pressure wave in the aorta at the level of the coronary ostium will cause a forward flow transient in the coronary artery regardless of whether the direction of wave propagation is antigrade or retrograde.

Another possible reason for the atrial cove is that it represents the direct inhibition of atrial blood flow by atrial systole. There are, however, several reasons why this is unlikely. First, atrial coves are observed downstream to the sites of origin of the atrial arteries. Second, the decrement in flow in some instances is constant over a wide range of perfusion pressures, meaning that autoregulation by atrial vessels must be perfect. Third, occlusion of accessible atrial arteries does not alter the appearance of the cove.

A more likely explanation for the atrial cove follows from the observation by VATNER et al. (1980) that atrial systole caused a substantial reduction in the diameter of the canine circumflex coronary artery. The decrease in this vessel's diameter may be a consequence of an increase in the cross-section of the heart at the level of the atrioventricular groove caused by 15-20% increase in ventricular volume caused by atrial systole. If other coronary vessels were similarly elongated and narrowed, a considerable increase in coronary vascular resistance would occur because of atrial systole. Since atrial systole also increases right atrial pressure, the atrial cove might occur because of both an increase in resistance and a decrease in the perfusion pressure gradient across the coronary bed. Nevertheless, it is by no means certain that the magnitude of these changes would be sufficient to explain the decrements in flow apparent in Figure 1.

An alternative explanation for the existence of atrial coves is suggested by data exemplified by Figure 5. Figure 5 shows that atrial systole displaced the diastolic pressure-flow relation to a higher intercept pressure without any change in slope. Downey and Kirk (1975) and, more recently, Ellis and Klocke (1980) have shown that a similar parallel displacement of pressure-flow relations occurs when ventricular pressure is elevated. These data may be understood by assuming that a segment of the coronary bed normally has the hydraulic properties of a collapsible tube operating over that portion of the vessel's tube law (Shapiro, 1977) in which a slight change in transmural pressure causes a large change in cross-sectional area. Transmural pressure in this situation is close to zero and a sudden increase in extravascular compression will upset the force balance across the wall of the collapsible segment, leading to a decrease in cross-sectional area and a slowing of the flow rate. Since the pressure gradient between the origin of the vessel and the collapsible segment is flow determined (pressure gradient balanced against viscous effects), a decrease in flow rate will cause intravascular pressure to rise until a new force balance is established in the collapsible segment with a transmural pressure again zero. The new flow rate will, however, be less than before, because the permissible pressure drop from the origin of the vessel to the collapsible segment has become smaller.

The following sequence of events, based on an analogy with collapsible tubes, appears to be a plausible explanation for the existence of atrial coves. Atrial systole ejects a volume of blood into the ventricular cavity causing simultaneous increases in ventricular cavity and intramyocardial pressures. The back pressure that opposes coronary artery flow is located in the collapsible segment at the point where transmural pressure is zero. Intravascular pressure at this location equals the collapsing pressure caused by a combination of vasomotor tone and extravascular compression (intramyocardial pressure). By increasing intramyocardial pressure, atrial systole increases the intravascular pressure necessary to prevent closure of the collapsible segment. This rise in back pressure decreases the arterial perfusion pressure gradient (aortic root pressure is unchanged), and the flow rate falls. Finally, the rise in intramyocardial pressure will affect other vessels, including intramyocardial veins. Blood displaced from collapsible ves-
sels will be expelled downstream and appear as a forward flow transient in the venous flow tracing.

Further support for the preceding explanation comes from the observation that the increase in intramyocardial pressure during the atrial kick and the increase in the zero flow pressure intercept of the pressure-flow relation were of similar magnitude. Although this suggests a possible cause-and-effect relationship, due caution is indicated because of the considerable doubt that exists regarding the feasibility of measuring intramyocardial tissue pressure. Since the report of Gregg and Eckstein (1941), it has been known that the size of the transducer used to measure systolic intramyocardial pressure affects the magnitude of the measurement. To what degree this limitation also applies to measurements made in diastole is unclear. Stein et al. (1980) have reported an extensive experience with the use of the Millar catheter pressure transducer to measure diastolic intramyocardial pressure. Certainly, in comparison with previous methods, the technical aspects of intramyocardial pressure measurements are greatly simplified by using this device. Nevertheless, care must be taken to recognize drift of the electronic reference for zero pressure because, otherwise, measurements will not be made relative to true atmospheric zero. The electronic calibration signal of the Millar transducer was found to be reliable. Thus, it is likely that the strength (<5 mm Hg) of the atrial kick reported here is accurate, even though it is somewhat less than the strength of atrial kicks seen in humans and in conscious dog preparations (5-8 mm Hg). This difference may be a consequence of the fact that, in the experiments reported here, the pericardium had been incised in two places and could not exert its normal restraint on myocardial distension. An additional factor that might weaken atrial systole is myocardial depression secondary to operative trauma and anesthesia.

Changes in diastolic coronary vascular resistance were also found to determine the size of the atrial cove: the greater the initial resistance, the smaller the decrement in flow, and vice versa. The following exercise in modeling shows why this is so. Let $P_a$, $P_d$, and $P_c$ be the arterial pressure, diastolic arterial pressure, and coronary arterial pressure opposing coronary artery flow during diastole and the atrial cove, respectively; $P_a$, $P_d$, and $P_c$ be the back pressure opposing coronary artery flow during diastole and the atrial cove, respectively; and $R$ and $\alpha R$ be the resistance during diastole and the atrial cove, respectively. Alpha is the fractional increase in resistance during atrial systole and will equal one if resistance does not change. Ignoring inertial and capacitance effects, arterial flow during diastole and the atrial cove will be $Q = (P_a - P_d)/R$ and $Q_c = (P_a - P_c)/\alpha R$, respectively; and the decrement in flow during the atrial cove will be $(P_a - P_d) - (P_c - P_d)/\alpha R$. Clearly, as resistance increases, the decrement in flow will become smaller.

The problem remains as to how to define resistance in the above equations. Several recent papers (Klocke et al., 1980; Bellamy, 1980) have discussed coronary vascular resistance in terms of two hemodynamic models: the classical model in which the coronary back pressure is taken to be venous pressure, and the waterfall model which is based on the known hydraulics of collapsible tubes (Lyons et al., 1980) and Permutt and Riley's concept of the vascular waterfall (Permutt and Riley, 1983). The latter model takes the coronary back pressure to be the arterial pressure at which flow in arteries stops. Resistance defined by the waterfall model is the reciprocal of the slope of the pressure-flow relation. Two relations that are parallel will therefore have the same resistance. When the classical model is applied to a pressure-flow relation which does not intercept the pressure axis at venous pressure, one finds that resistance progressively increases as flow falls. Thus, two relations that are parallel will have different resistances.

Since the pressure-flow relation shown in Figures 5 and 6 are parallel, resistances are the same when defined in terms of the waterfall model. Given this circumstance, the expression for the decrement in flow during the atrial cove reduces to change in back pressure/resistance. Since the measured change in back pressure (horizontal separation between the two relations) is constant, all atrial coves should have the same depth. Put in another way, given the observed constant change in back pressure and constant decrement in flow, the data shown in Figures 5 and 6 suggest that the coronary bed is behaving as if its resistance is also constant; i.e., the waterfall definition of resistance is applicable.

If venous pressure is taken to be the coronary back pressure, resistance must increase as the flow rate decreases. As shown previously, an increase in resistance should cause the atrial coves to become smaller. Clearly, this is not what Figures 5 and 6 show. The preceding argument suggests a sensitive experimental test that might be used to distinguish between the two hemodynamic models. The following question could be asked: given a succession of identical atrial kicks, does the decrement in flow follow changes in resistance defined in terms of the slope of the pressure-flow relation, or does the decrement in flow follow changes in resistance defined with venous pressure as the back pressure?

The conclusion of this paper as to the origin of the atrial cove is conveniently stated by using the phasic data of Figure 1B as an example. The back pressure opposing coronary artery flow is not venous or right atrial pressure, but a much higher pressure due to vasomotor tone and intramyocardial pressure acting on collapsible coronary microvessels. The effective coronary back pressure existing during the last beat in Figure 1B was ~45 mm Hg. Immediately prior to the atrial cove, the arterial perfusion pressure had fallen to ~50 mm Hg, and the actual gradient for arterial flow was therefore only ~5 mm Hg. Flow stopped during the atrial
because atrial systole, by increasing the intramyocardial pressure component of the back pressure by ~5 mm Hg, reduced the arterial perfusion pressure gradient to zero.

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