The Contribution of Blood Momentum to
Left Ventricular Ejection in the Dog

By Mark I. M. Noble, Ph.D., B.Sc., M.B., B.S.

ABSTRACT
In five conscious dogs and two anesthetized dogs, the pressure gradient between the left ventricle and the ascending aorta was measured with high fidelity micromanometers. Left ventricular pressure exceeded aortic pressure in the first 30 to 50% of the ejection period. Aortic pressure exceeded left ventricular pressure during the remainder of the ejection period. The magnitude of these differences varied from 5 to 20 mm Hg. This finding in late systole implies that at this time blood is flowing out of the ventricle under its own momentum and that the aorta is a pressure source for the ventricle. The ascending aorta was suddenly occluded in five dogs to verify this hypothesis. When occlusion occurred in the last third of the ejection period, there was an abrupt fall of left ventricular pressure, apparently confirming the momentum hypothesis.

ADDITIONAL KEY WORDS
left ventricular pressure  aortic pressure
pressure gradient  aortic occlusion  fluid inductance  late systole

Ambrosi and Starr (1) have said, "So many of our colleagues continue to think of the function of the heart as the Greeks thought of motion in general, e.g., the sun is drawn by Apollo's chariot because, like a wagon on earth, if not continually worked upon, its motion would stop. Similarly, many think that the heart must keep pushing out the blood up to the end of ejection. That this is not necessary has been clearly demonstrated by Spencer and Greiss (2); the data of Rushmer and associates (3) are also consistent with this view. The blood, once set into motion, will continue in motion because of its inertia until resistance stops it. So, after positive acceleration is over and maximum velocity has been attained, little if any cardiac effort will be required and this effort may cease well before the end of ejection." In previous studies of left ventricular ejection (4, 5), a similar suggestion was made. The present investigation was therefore designed to explore the contribution of momentum to left ventricular outflow.

The finding by Spencer and Greiss (2) that the pressure difference between the left ventricle and ascending aorta is negative in late systole would support the momentum concept, but the validity of their results has been questioned (6, 7). The first part of the present study was therefore devoted to a re-examination of this pressure difference.

If late systolic ejection results from the momentum of the blood, the heart, by implication, is not contributing greatly to this ejection or to the generation of left ventricular pressure; an alternative source of left ventricular pressure would be a retrograde transmission of aortic pressure. The second part of this study was therefore devoted to experiments in which the aorta was rapidly occluded during ejection. Under these circumstances, active generation of left ventricular pressure and attempted expulsion of blood against the occluded aorta would result in a rise of left ventricular pressure; in the case of retrograde transmission of aortic pressure, there would be a fall of left ventricular pressure when the aorta was occluded.

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The question of the contribution of momentum to ejection is of considerable importance because, if momentum contributes a sizable part of the stroke volume, the amount of shortening of isolated papillary muscle (8) cannot be considered analogous to stroke volume because, in the case of stroke volume, some of the shortening may result from blood flow due to momentum.

The insensitivity of cardiac output and stroke volume to changes in myocardial performance (5, 9) may be then explained by the hypothesis that changes in shortening predicted by isolated muscle experiments (8) are masked by opposite changes in the volume flow due to momentum (5).

Methods

PRESSURE GRADIENT

In seven dogs weighing 15 to 25 kg, a left thoracotomy was performed under aseptic conditions. A Microsystems 1017 pressure transducer was implanted through the anterior wall of the left ventricle and positioned as illustrated in Figure 1. An electromagnetic flowmeter transducer was implanted around the ascending aorta, and a polyvinyl or silastic catheter was inserted into the left atrium. The chest was closed and the dogs were allowed 1 to 2 weeks to recover.

The right brachial artery was then exposed under halothane (1% in nitrous oxide and oxygen in one dog), pentobarbital (24 mg/kg in one dog) or local (five dogs) anesthesia (the dogs were not disturbed or distressed by the procedure under local anesthesia). The anesthesia was continued as necessary for the duration of the study. A Statham SF-1 catheter-tip manometer was inserted into the artery and advanced under fluoroscopic control to the level of the 1017 transducer in the left ventricle. The SF-1 had a side-hole attachment at the tip as illustrated in Figure 1; in this way lateral pressure was recorded.

Continuous recordings of pressure and flow were made on a 7-channel Ampex tape recorder as the catheter-tip manometer was withdrawn to the ascending aorta (Fig. 1). The tape recording was played back onto an Electronics for Medicine photographic recorder. The gain and zero controls of this recorder were adjusted so that, at the beginning of the record when both transducers registered left ventricular pressure, the two pressure tracings during the period of isovolumic contraction were exactly superimposed (Fig. 2, A). It was then assumed that the pressure would be the same at the two transducers during this period of no flow. The gain and zero of the catheter-tip manometer were then matched to the 1017 transducer and, during the later part of the record when the SF-1 manometer was recording aortic pressure, the pressure difference between the left ventricle and aorta was demonstrated (Fig. 2, B). The withdrawal procedure was used only to match the two transducers; the pressures were always measured.
simultaneously with the left ventricular (1017) and aortic (SF-1) transducers (Figs. 1, B and 2, B). It was possible to reduce the noise on these records (Fig. 2, B) by using a tape speed for playback which was one quarter of that used for recording and then introducing a filter.

In one additional dog, two SF-1 manometers were advanced to the left ventricle so that the tip of one was just below the aortic valve and the other was 2.5 cm proximal in the outflow tract. The pressure tracings were displayed directly on an oscilloscope. The gain and zero were adjusted so that the phases during isovolumic contraction were identical on the two tracings. Recordings were then made by photographing the oscilloscope screen using a Cossor camera at a film speed of 10 inches/sec.

AORTIC OCCLUSION

In a separate series of experiments, the ascending aorta was occluded during ejection in three open-chest dogs anesthetized with pentobarbital, 90 mg/kg, and in two conscious dogs. Ventilation was maintained in the open-chest dogs with a Harvard intermittent positive-pressure respirator. A left thoracotomy was performed and a Microsystems 1017 pressure transducer was inserted into the left ventricular cavity. The ascending aorta was dissected and a stainless steel snare placed around it. The aorta was occluded by a large Guardian 115-volt a-c solenoid which was activated during ejection.

In one of the conscious dogs, the same snare, together with the pressure transducer, was inserted under aseptic conditions; the chest was closed leaving the end of the snare under the skin. On the following day, the end of the snare was exposed, the solenoid attached, and the experiment carried out in the same manner as for the open-chest dogs.

In the second conscious dog, a solenoid-operated shutter was placed around the ascending aorta. The entire device, including solenoid, was left within the chest. This solenoid (24 volts dc, Domneyer Industries) was 5 cm in length and 2.5 cm in diameter. Sufficient force to occlude the aorta rapidly was obtained by applying 125 volts dc for a short period. Both types of occluder (snare and shutter) required approximately 90 msec to close.

In all the aortic occlusion experiments, the upstroke of left ventricular pressure was used to generate a positive pulse which turned on a silicon controlled switch. The timing of this pulse was controlled by a delay circuit which allowed continuously variable timing of occlusion. Conduction through the silicon controlled switch allowed 15 volts dc to be applied to a relay or power-switching transistor which switched the power to the solenoid.

FLOW AND PRESSURE MEASUREMENTS

Two types of electromagnetic flowmeter were used, the Statham (Medicon) M 4001 and Biotronex BL 612. The frequency response of these instruments was measured using electrical modulation of the carrier wave (10). The amplitude frequency response of the Statham showed 18% attenuation at 20 cycles/sec and 30% at 30 cycles/sec. The phase-shift frequency response was linear with a transit time of 9.4 msec (for frequencies 1-50 cycles/sec). The amplitude frequency response of the Biotronex showed no attenuation at 20 cycles/sec and less than 3% at 30 cycles/sec. The phase-shift frequency response was linear with a transit time of 5.3 msec (constant for frequencies 1-50 cycles/sec).

Since the aortic flow signal contains frequencies up to 30 cycles/sec (11), the Medicon instrument introduced significant damping. Correction was made for the transit times when time relationships between pressure and flow were analyzed. Calibration was done at the end of the experiment by reopening the chest and inserting a cannulating flow transducer into the descending aorta. All branches of the aorta between the cuff and cannulating flow transducers were tied off so that the two were in series. Zero was obtained by arresting the heart with acetylcholine (12). The calibration of the cannulating transducer was determined by pumping blood or saline through it at known flow rates. Since both flow transducers were linear, the area under the flow curve of the cannulating transducer represented the same volume as the area under the flow curve of the cuff transducer. Knowing the time base of the recorder, the flow calibration of the cuff transducer was then obtained.

The pressure-sensitive diaphragm of the 1017 transducer was 6.5 mm in diameter and remained in the left ventricular cavity; there were therefore no catheter transmission problems. The gauge was linear from 0 to 300 mm Hg and showed no hysteresis. There was no detectable base-line shift over a period of 2 hours. An attempt was made to measure the natural frequency of the gauge using the step-pressure ("pop") technique (13, 14), and a resonant frequency of 3000 cycles/sec was recorded. However, this is probably the natural frequency of the test chamber, the SF-1 manometer also registered a resonant frequency of 3000 cycles/sec in the same chamber. The performance of the SF-1 has been described previously (11, 15) and was very similar to that of the 1017 transducer except that it was more temperature sensitive; adequate time was therefore allowed for complete temperature correction before the results were recorded.

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stabilization whenever the lumen was flushed. The problems involved in measuring lateral aortic pressure with an SF-1 manometer have been described previously (11). Calibration of both manometers was determined by matching the transducer output to a reference pressure recorded with an external Statham P23Dc gauge via the lumen of the SF-1 catheter (11). Alternatively, calibration of the 1017 manometer was performed in vitro at the end of the experiment in a water bath at 38.5°C with a mercury column as the reference.

Results

Pressure Gradient

In all the experiments, the left ventricular pressure was higher than aortic pressure (positive gradient) early in the ejection period, but aortic pressure was higher than left ventricular pressure (negative gradient) in late systole (Figs. 3 and 4, Table 1). The change from positive to negative gradient occurred at 30 to 50% of the duration of ejection (Table 1) and 20 to 30 msec after the time of peak aortic flow (Fig. 3). The magnitude of

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FIGURE 3

Record from conscious dog under resting conditions. AP = pressure in proximal ascending aorta; LVP = left ventricular pressure; AF = aortic flow. The flowmeter has a transit time of 0.4 msec.

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FIGURE 4

Record from dog anesthetized with 1% halothane. AP = pressure in distal ascending aorta; LVP = left ventricular pressure.

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### Table 1

<table>
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<tr>
<th>Size of art.</th>
<th>Heart rate</th>
<th>AP (mm Hg)</th>
<th>LVP (mm Hg)</th>
<th>VEDP (mm Hg)</th>
<th>T1 (msec)</th>
<th>T2 (msec)</th>
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LVP = left ventricular pressure; VEDP = left ventricular end-diastolic pressure. T1 = duration of period of positive difference divided by aortic pressure trace. T2 = the period of positive pressure of the PD (and VEDP) were measured with a 9.4 msec transit time.
the gradients was variable (Table 1). A similar pattern of pressure gradient was found between the proximal and distal left ventricular pressures recorded by two catheter-tip manometers (Fig. 5) and between the left ventricular pressures recorded by the 1017 and SF-1 transducers just before the latter was withdrawn through the aortic valve.

**AORTIC OCCLUSION**

During the time that the snare or shutter was closing, there was a spike or hump of left ventricular pressure while the aorta was being compressed. This was obscured by a rise in left ventricular pressure when the occlusion occurred in early systole and was followed by a fall in left ventricular pressure with late systolic occlusion (Figs. 6 and 7). Figure 7 shows that following occlusion in late systole, left ventricular pressure fell below the pressure at the corresponding time in the previous beat.

**Discussion**

In previous studies (4, 5) it has been suggested that when the left ventricle ejects blood at high peak velocity, the blood flow in the last part of the ejection period may result from the momentum of blood. The present study provides a certain measure of support for this view.

The longitudinal impedance along a large vessel tends to become dominated by fluid inductance as the diameter of the vessel and the frequency of the flow pulsations increase (14). As this happens, the pressure gradient along the vessel approaches a 90° phase shift ahead of flow and becomes nearly in phase with the acceleration of flow. These conditions apply to the ascending aorta of dog and man (16-19). The present study shows that similar considerations apply to the outflow tract of the left ventricle because the pressure gradient from left ventricle to aorta (Figs. 3 and 4) or along the left ventricular outflow tract (Fig. 5) is positive during acceleration and becomes negative shortly after deceleration begins.

Wiggers (20) pictured the left ventricular pressure and aortic pressure as identical during systole; only recently has it been possible to measure the small differences between them. Remington (21) and Driscoll and Eckstein (6) found a positive difference in early systole but thought that no difference was present in late systole. However, Spencer and Greiss (2) described a very brief early systolic spike of positive gradient followed by a negative gradient; the positive spike has been considered an artifact (7). These authors had only conventional manometers which required transmission of the pressures along catheters, needles or sounds with the attendant risk of waveform distortion. In the present study modern miniature manometers were used.

In a large vessel in which high frequency oscillations of flow occur, there is, as was stated before, a pressure gradient which is almost in phase with acceleration because fluid inductance dominates the longitudinal impedance. Thus, a positive pressure gradient is required to overcome the inertia of the
blood and accelerate it. The blood would then continue to flow under its own momentum since the viscous resistance is small. A negative pressure gradient is required to overcome the momentum of the blood and decelerate it. Similarly, it can be said that the early systolic pressure gradient between left ventricle and aorta accelerates the blood to a high peak velocity and peak momentum (mass times velocity). The blood may then continue to flow under its own momentum, and a negative pressure gradient will be required to decelerate it.

These considerations naturally raise the question, "If blood is flowing out of the ventricle under its own momentum, what is the ventricular muscle doing? Is it still actively ejecting blood?" Since there is still a high pressure in the ventricle at this time, one might suppose that it resulted from the contraction of the ventricle. However, the pressure in the aorta is higher than in the ventricle, and flow is continuing from ventricle to aorta by way of the aortic valve.

**FIGURE 6**
Records of left ventricular pressure obtained from a conscious dog, 1 day after left thoracotomy. A:

**FIGURE 7**
Superimposed tracings from an anesthetized, open-chest dog. A: Control beat. B: Subsequent occluded beat. Solid bar indicates time taken for snare to close.

Control beat. B: Aortic occlusion (in diastole) which was maintained throughout the subsequent systole to record left ventricular pressure for an isovolumic beat. C to H: Aortic occlusion during ejection at times indicated by the arrows. Width of arrows indicates the 20 msec required for the shutter to close and compress the aorta. From C to H, occlusion was timed for progressively later times in the ejection period. The rise in pressure produced in the obstructed ventricle diminished with later times of occlusion until occlusion in the last third of ejection resulted in a fall of left ventricular pressure (H).
aorta, keeping the aortic valve open. The aorta and ventricle are thus in free communication. There may be, therefore, transmission of pressure from the higher pressure source (the stretched aortic wall which recoils) to the ventricle.

The purpose of the aortic occlusion experiments was to determine whether this phenomenon is present and to determine its magnitude. If pressure in the ventricle is transmitted from the aorta, separation by aortic occlusion of the ventricle from the pressure source (the aorta) should result in a fall of pressure. If the contraction of the ventricle is responsible for the pressure, the introduction of a complete obstruction by aortic occlusion should result in a rise of pressure.

Unfortunately, it was not possible to perform the experiment in an ideal manner. It would have been preferable to occlude at the level of the aortic valve. The occluders used were actually distal in position to part of the hypothetical pressure source (proximal ascending aorta). It was not possible to design an occluding device which could occlude the aorta in less than 20 msec. The use of an anesthetized, open-chested preparation was unsatisfactory because the acceleration, deceleration, peak velocity, and peak momentum of the blood ejected from the left ventricle are reduced under these circumstances. Implantation of the occluder resulted in the collection of blood, fluid, and granulation tissue which prevented or restricted movement of the shutter. The experiment, therefore, had to be performed the day after surgery, at which time left ventricular performance (as judged by maximum rate of rise of left ventricular pressure and left ventricular end-diastolic pressure) remained impaired.

Despite all these factors working against the phenomenon being tested, a definite fall in left ventricular pressure was observed after occlusion of the aorta in the last third of systole. The phenomenon may be greater in the healthy conscious animal.

The suggestion that the ventricle is contributing little to the ejection of blood in late systole does not imply that the contractile element (22) is passive at this time. This cannot be so since the ventricle does support a high pressure and, therefore, considerable force in the wall (21). The contractile element must therefore be sufficiently active to maintain stretch of the series elastic element (22). Since the pressure falls following late systolic occlusion, it may be argued that prevention of further shortening at this time in the cardiac cycle produces a reduction in the active state (24) of the muscle. This possibility, which cannot be excluded in the absence of any method for measuring active state in the intact animal, would provide an alternative explanation for the results.

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