Analysis of the Characteristics of the Flow Velocity Waveforms in Left Atrial Small Arteries and Veins in the Dog

Fumihiko Kajiya, Katsuhiko Tsujioka, Yasuo Ogasawara, Osamu Hiramatsu, Yoshifumi Wada, Masami Goto, and Masao Yanaka

To clarify the characteristics of the phasic blood velocity pattern in small arteries and veins on the left atrial surface, we used our newly developed fiber-optic laser Doppler velocimeter. We intended particularly to examine the influence of atrial contraction and relaxation on velocity waveforms to obtain some insight into the nature of the mechanical force acting on the atrial intramyocardial vascular beds. In 14 anesthetized open-chest dogs, the left atrial appendage was gently displaced to expose small branches of the artery and vein. Vessels with an outer diameter of about 150–500 μm were chosen for the measurements because their walls are transparent to laser light. The fiber tip (velocity sensor) was fixed on the vessel surface with a drop of cyanoacrylate when good-quality Doppler signals were consistently observed. Additional experiments with three dogs were performed to observe the blood velocities in the atrial artery and vein during arrhythmia. The blood velocity waveform in the artery was similar to the pattern of aortic pressure during ventricular ejection (peak velocity, 18.8±7.8 cm/sec) but was characterized by a pronounced dip during atrial contraction. The temporal coincidence between the dip formation and atrial contraction was confirmed during atrial flutter with an atrioventricular block. After isoproterenol administration (2 μg i.v.), the acceleration rate of the forward flow velocity increased by 176% (p<0.05), and reverse flow appeared during atrial contraction in five cases out of eight (p=0.013). The blood flow velocity in atrial small veins, on the other hand, was predominant during atrial contraction (peak velocity, 15.6±5.8 cm/sec). Isoproterenol increased the acceleration rate of this forward flow velocity by 121% (p<0.01). Nitroglycerin did not change the blood velocity waveform significantly in atrial arteries or in veins. The phase opposition between arterial inflow into and venous outflow from the atrial myocardium indicates that a large portion of the coronary inflow to the atrial myocardium may be stored due to the presence of atrial myocardial vascular capacitance. We conclude that atrial myocardial contraction impedes atrial inflow and promotes venous outflow from atrial capacitance vessels. (Circulation Research 1989;65:1172–1181)

The blood flow in the coronary circulation has a unique phasic nature. The distal coronary artery flow of the left ventricle is almost exclusively diastolic,1,2 whereas venous outflow occurs predominantly during systole.3–6 This phase opposition between the coronary artery and the vein flow of the left ventricle can only be explained by the diastolic storage of arterial inflow in intramyocardial capacitance vessels and subsequent displacement into the coronary veins during ventricular contraction. Intramyocardial capacitance has been estimated to be 0.08–0.25 ml/mm Hg/100 g left ventricular myocardium by simultaneous measurement of coronary artery and vein flows in the dog.7–9 However, the mechanical characteristics of the compressive force acting on the intramyocardial capacitance vessels during ventricular contraction have not yet been well clarified. To better understand the nature of this compressive force, comparison of atrial artery and vein velocity waveforms during atrial contraction and relaxation with those of the left ventricle seems necessary. However, studies on the phasic atrial flow velocities have been few because of the technical difficulties of measurement. Therefore, reports have been limited to evaluation of the flow in...
the right sinus node artery and the microcirculation of the left atrium.

The present study was undertaken to determine the characteristics of the flow velocity waveforms in small arteries and veins on the left atrial surface using our laser Doppler velocimeter (LDV) with an optical fiber. This LDV has the following advantages over conventional methods: 1) fine accessibility of the optical fiber probe to small vessels on the surface of the beating heart, 2) high accuracy of instantaneous velocity measurements for both forward and backward flows, and 3) excellent baseline stability for zero velocity. We intended particularly to examine the influence of atrial contraction on atrial artery and vein velocity waveforms to obtain some insight into the nature of the mechanical force acting on the intramyocardial vascular beds.

Materials and Methods

Laser Doppler Blood Flow Velocimeter With An Optical Fiber

Measurements of blood flow velocity were made with an LDV with an optical fiber, previously described in detail by Kajiya and colleagues and Nishihiara et al. In short, a helium-neon laser beam (632.8 nm, 5 mW) is divided into incident and reference beams by a beam splitter. The incident beam is directed onto the vascular surface through an optical fiber (external diameter, 62.5 μm; core diameter, 50 μm) and then introduced into the vascular lumen through the vessel wall. Part of the light that is backscattered through the vascular wall by flowing erythrocytes is collected by the same fiber and transmitted back. The other light that is divided by the beam splitter is used as the reference beam. A frequency shifter (40 MHz) is interposed in the path of the reference beam to differentiate forward from reverse flow. Photocurrent from an avalanche photodiode is fed into a spectrum analyzer to detect Doppler shift frequencies. The Doppler shift frequency Δf is given by

\[ \Delta f = 2nV \cos \theta / \lambda \]  

where \( V \) is the blood flow velocity, \( n \) is the refractive index of blood (approximately 1.33), \( \theta \) is the angle between the fiber and the blood stream axis, and \( \lambda \) is the laser wavelength (632.8 nm). The maximum Doppler shift frequency in the sample volume, that is, the maximum velocity, was detected automatically.


We have already reported on the high level of accuracy of our method, which was evaluated by immersing the fiber tip in blood and measuring the known blood velocity in the circular groove of a turntable rotated at various speeds (correlation coefficient between known and measured velocities, 0.998). In this study, we examined the accuracy of blood velocity measurements in a small vessel by using four dogs. The animals were killed at the end of the experiments by intravenous pentobarbital followed by potassium chloride. The heart was removed; then the left anterior descending coronary artery or the left circumflex coronary artery was cannulated, and the cannula end was connected to a reservoir filled with autologous blood. The fiber tip (velocity sensor) was placed on a small branch (about 500 μm o.d.) of the left anterior descending coronary artery or the left circumflex coronary artery, and the blood velocities were measured by the LDV at a range of different perfusion pressures. The vascular portion distal to the velocity-measuring section was cut, and the blood effluent was collected with a graduated cylinder and a stopwatch. In two dogs, the vascular diameter was measured by a radiograph of the vessel after intracoronary injection of barium sulfate–gelatin mixtures. The blood volume flow was calculated from the vascular diameter, and the blood velocity was measured by the LDV (blood volume flow = \( 1/2 \times \pi \times \text{diameter}^2 \times \text{maximum velocity} \)). It was assumed that the flow obeys the Poiseuille law; that is, it follows a parabolic velocity profile across the vessel. The calculated blood flow was compared with the blood flow determined by timed blood collection. In the other two dogs, the timed-collected blood flow was compared with the LDV-measured blood velocity.

Animal Preparation

Fourteen adult mongrel dogs of both sexes (15–25 kg) were anesthetized with sodium pentobarbital (25 mg/kg i.v.). After intubation, the animals were ventilated by a respirator pump (Harvard Apparatus, South Natick, Massachusetts) with room air, which was supplemented with 100% oxygen at a rate sufficient to maintain arterial oxygen tension at a physiological level. A left thoracotomy was performed at the fourth and fifth intercostal space. The heart was exposed and suspended in a pericardial cradle.

Electrocardiograms were recorded from standard leads. Stiff polyvinyl catheters were inserted into the ascending aorta through the carotid artery and into the right atrium through the right atrial appendage. Pressure measurements were taken with a fluid-filled catheter and a pressure transducer (model DHC, Nippon-Koden, Tokyo, Japan). Left ventricular pressure was measured by a catheter tip micromanometer (model PC-470, Millar, Houston, Texas). Left atrial pressure was measured in some cases with a catheter tip micromanometer (model SPR-230, Millar). All measurements were recorded on a data recorder (model R-81, TEAC, Tokyo, Japan), and direct writing was made by a multichannel recorder (model RIJ 5608, Nippon-Koden).
Measurements of Blood Flow Velocities in Atrial Small Arteries and Veins

The left atrial appendage was gently displaced to expose small branches of the artery and/or vein. The artery is a branch of the left circumflex coronary artery, and the vein drains into the great cardiac vein. Vessels with an outer diameter of about 150–500 μm were chosen for the measurements because their vascular walls are transparent to laser light. Since the maximum detectable length for backscattered light in the blood was found to be around 300 μm by our previous study, the sample volume may include the central axial region in the vessel. The fiber tip was placed on the vessel surface and was traversed manually perpendicular to the vascular axis. When large and good-quality Doppler signals were consistently observed, the fiber tip was fixed at that position with a drop of cyanoacrylate. Then, the fiber was gently supported so that it did not compress the vessel. This method is especially useful for the measurement of small atrial artery and vein flow velocity since these vessels move with cardiac motion and the vein in particular is easily deformed. The angle $\theta$ between the fiber and the vascular axis was measured to allow blood flow velocity to be calculated from Equation 1. The blood flow velocity was measured under control conditions, after intravenous administration of nitroglycerin (0.5 mg i.v.), and after isoproterenol administration (2 μg i.v.). After blood flow velocity measurement of an artery (or vein), the fiber tip was moved onto a vein (or an artery if the first measurement was of a vein) when a vessel of suitable size and at a suitable position was found. Then, the above-mentioned procedure was repeated. Additional experiments with three mongrel dogs (15–16.5 kg) were performed to observe the blood velocity waveforms in the left atrial small arteries and veins during atrial fibrillation or flutter and ventricular premature beats induced by electrical stimulation, mechanical stimulation, or by applying aconitine on the left atrium.

Statistical Analysis

Results were expressed as mean±SD. The differences between two means were compared by the paired $t$ test. The relation between two parameters was evaluated by a simple correlation analysis. The differences between two ratios were compared by Fisher’s exact test. The criterion for statistical significance was $p<0.05$.

Results

Accuracy of Blood Flow Velocity Measurements in Small Vessels by the Laser Doppler Method

Figure 1 shows the relation between the blood volume flow rate calculated from the measured velocity by our LDV method and vascular diameter and blood flow rate determined by timed collection in one dog. An excellent correlation was found between the two values in two animals ($r=0.99$, $p<0.01$; $r=0.97$, $p<0.01$). However, the absolute volume flow rate was underestimated by about 10%. The correlation coefficients between the blood velocity measured by the LDV method and the timed-collected blood flow rate in the other two dogs were also significantly high ($r=0.99$, $p<0.01$; $r=0.96$, $p<0.01$). These results indicate that our method accurately measures blood flow velocity in small vessels with thin walls.

Blood Velocity Waveforms in Small Arteries on the Left Atrium

Figure 2 shows a representative tracing of the blood flow velocity in a left atrial small artery under control conditions. The velocity increased with the aortic pressure and decreased gradually after reaching a peak in mid systole. The blood flow velocity waveform showed a dicrotic notch, as seen in the aortic pressure waveform. Thus, the outline of blood velocity waveform resembled the pattern of aortic pressure during ventricular ejection. However, a sharp transient decrease in flow velocity, that is, a dip formation, corresponding to the atrial contraction phase was always observed in late ventricular diastole. The dip was initiated with the rise in the left atrial pressure and ceased at the end of left ventricular isovolumic contraction (also see Figure 7).

Figure 3 shows a typical example of the effect of isoproterenol administration on the blood flow velocity in a small atrial artery. Compared with the control tracing, isoproterenol caused an increased rate of acceleration of the systolic velocity wave ($p<0.05$) without a significant change in the peak flow velocity (Figure 4 and Table 1). Another characteristic of the blood flow velocity waveform after isoproterenol administration was the first appear-
FIGURE 2. A representative tracing of the blood flow velocity in a left atrial (LA) small artery under control conditions. The outline of the blood velocity waveform resembled the pattern of aortic pressure (AoP) during ventricular ejection, but a sharp transient decrease in flow velocity corresponding to the atrial contraction phase was observed in end diastole. LVP, left ventricular pressure, ΔV, velocity change; Δt, time in 0.1-second increments. The maximum acceleration rate (ΔV/Δt) of the forward flow in Table 1 and Figure 5 was read as shown in this figure.

Table 1. Comparison of Hemodynamic Values and the Dimensions of Velocity Waveforms in Atrial Small Arteries and Veins Under Control Conditions and After Administration of Isoproterenol and Nitroglycerin

<table>
<thead>
<tr>
<th></th>
<th>Heart rate (beats/min)</th>
<th>Aortic pressure (mm Hg)</th>
<th>Right atrial pressure (mm Hg)</th>
<th>Peak velocity (cm/sec)</th>
<th>Maximum velocity after aortic valve closure (cm/sec)</th>
<th>Maximum velocity before atrial contraction (cm/sec)</th>
<th>Maximum acceleration rate (cm/sec/0.1 sec)</th>
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<tbody>
<tr>
<td>Left atrial artery (n=8)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>105±25</td>
<td>109±21/86±25</td>
<td>3.6±1.1</td>
<td>18.8±7.8</td>
<td>15.5±7.9</td>
<td></td>
<td>25.7±12.9</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>138±25*</td>
<td>95±26/63±22</td>
<td>4.8±2.2</td>
<td>20.7±9.6</td>
<td>12.3±7.8</td>
<td></td>
<td>71.0±43.4*</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>101±21</td>
<td>97±13/72±14</td>
<td>3.4±1.0</td>
<td>22.5±7.1</td>
<td>17.3±6.5</td>
<td></td>
<td>39.6±22.0</td>
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<tr>
<td>Left atrial vein (n=9)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>112±22</td>
<td>122±18/104±16</td>
<td>2.8±1.2</td>
<td>15.6±5.8</td>
<td>2.8±4.2</td>
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<td>34.5±22.5</td>
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<tr>
<td>Isoproterenol</td>
<td>130±13</td>
<td>103±15/86±14</td>
<td>3.8±1.6</td>
<td>19.2±5.3</td>
<td>1.4±1.4</td>
<td></td>
<td>76.3±25.9†</td>
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<tr>
<td>Nitroglycerin</td>
<td>116±22</td>
<td>93±16/71±14</td>
<td>3.0±1.5</td>
<td>16.7±8.7</td>
<td>3.3±2.5</td>
<td></td>
<td>46.6±29.5†</td>
</tr>
</tbody>
</table>

Values are mean±SD. Maximum acceleration rate is velocity change per time (0.1-second increments). Aortic pressure, systole/diastole.

*p<0.05 vs. control.

†p<0.01 vs. control.

‡p<0.001 vs. control.
shape. Figure 6 shows an example of the blood velocity waveform during ventricular premature contraction. The dip appeared with the increase in left atrial pressure during sinus rhythms and ventricular premature contractions. However, the depth of the dip after ventricular premature contraction was smaller than that of sinus rhythms although the developed left atrial pressure was greater after ventricular premature beats.

Blood Velocity Waveform in Small Veins

The blood flow velocity waveform in small veins on the left atrial surface was always characterized by a prominent atrial systolic velocity wave. Figure 7 shows both left atrial small artery and vein flow velocities under control conditions simultaneously to indicate the temporal relation between the two velocity waveforms. In this case, artery blood velocimetry was performed before the vein blood velocimetry, and the artery velocity waveform was added to the tracings during vein blood velocity measurement since the heart rate, left ventricular pressure, and left atrial pressure were almost identical for artery and vein blood velocity measurements. The vein blood velocity increased rapidly with atrial contraction and decreased with atrial relaxation. After a midystolic nadir, the blood flow velocity increased gradually before the onset of the atrial contraction phase. The major forward velocity wave...
of the vein showed a reciprocal relation to the dip of the artery velocity waveform.

Isoproterenol administration increased the rate of the rise in the venous velocity during atrial contraction by 121% (p<0.01) compared with the rate of rise under control conditions and increased the peak blood flow velocity in the atrial small vein by 23% (Figure 8 and Table 1). This latter change, however, was not significant. The systolic and diastolic aortic pressures decreased significantly, but the heart rate and right atrial pressure did not change significantly. Nitroglycerin administration increased the acceleration rate of the forward flow velocity during atrial contraction by 35% and the magnitude of the velocity component, with its earlier resumption during ventricular diastole, before atrial contraction by 18% compared with those values under control conditions. These changes, however, were not significant statistically (Figure 8 and Table 1). The peak blood flow velocity was similar to the control value.

Figure 9 shows an example of the atrial vein blood velocity waveform during a ventricular premature beat. The outline of the major forward velocity during ventricular premature contraction did not differ from that of sinus rhythms. However, the peak velocity was lower than that of sinus rhythms although the peak left atrial pressure was higher during the ventricular premature beat. During atrial fibrillation, small multiple forward velocity components appeared temporally at random, varying in shape and size.

![Figure 7](image1.png)

**Figure 7.** A representative recording of the blood flow velocity in a left atrial (LA) small vein under control conditions. A recording of small artery blood flow velocity is displayed simultaneously (see the text for details). The atrial vein velocity waveform was characterized by a prominent atrial systolic flow wave that showed a reciprocal relation to the dip of the arterial flow velocity. AoP, aortic pressure; LVP, left ventricular pressure; LAP, left atrial pressure; AV, velocity change; Δt, time in 0.1-second increments. The maximum acceleration rate (ΔV/Δt) in Table 1 and Figure 8 was read as shown in this figure.

![Figure 8](image2.png)

**Figure 8.** Bar graphs showing comparison of blood flow velocity waveforms in left atrial small veins among those under control conditions (CNT), after isoproterenol administration (ISP), and after nitroglycerin administration (NTG). The maximum acceleration rate is the velocity change per time (0.1-second increments). *Statistical significant at p<0.01.

![Figure 9](image3.png)

**Figure 9.** Tracings of the blood flow velocity in a left atrial (LA) small vein during ventricular premature contraction (arrow). AoP, aortic pressure; LVP, left ventricular pressure; LAP, left atrial pressure.
Discussion

This investigation provides the first systematic study of the blood velocity waveforms of atrial myocardial arterial inflow and venous outflow. The main observations were 1) that the velocity waveform in the artery flow resembled the aortic pressure pattern except during the atrial contraction phase, 2) that atrial contraction caused a transient sharp decrease in arterial flow velocity, and 3) that the velocity waveform in the atrial veins was characterized by a prominent atrial systolic velocity wave.

For this study we used the LDV with an optical fiber developed in our own laboratory. In previous studies, we have shown that the blood flow velocities in the proximal and distal coronary arteries and in the great cardiac vein can be successfully measured by inserting the optical fiber into the vessels with the aid of a light rubber cuff. Kilpatrick et al also have shown the high level of accuracy of the laser Doppler anemometer with an optical fiber catheter for measurements of coronary sinus flow in the dog. Hellenbrand et al successfully measured the anterior interventricular vein flow velocity by inserting a fiber probe into the vein. However, the measurement of blood flow velocities in superficial fine vessels is facilitated since the laser beam can be introduced into the vascular lumen from outside the walls without surgical isolation of the vessel. The blood volume flow rate calculated from measured velocity by our LDV method and vascular diameter showed an excellent correlation with the timed-collected volume flow rate with slight underestimation of the absolute blood flow. This underestimation may be mainly due to the assumption of a parabolic velocity configuration; the actual configuration may be blunter. Some errors in measurements of the vascular diameter and the angle between the fiber and blood stream axis may also result in underestimation of the absolute blood flow. To measure blood flow velocities, the fiber tip was fixed onto the outer wall of the vessel with a drop of cyanoacrylate. We succeeded in measuring the flow velocity in all cases when vessels of suitable size (about 150–500 μm o.d.) could be found on the left atrial surface. However, the amount of small arteries and veins on the left atrium is much smaller than the amount on the ventricle. In addition, flow velocities must be measured several millimeters from each other to avoid any effect by the above-mentioned cyanoacrylate treatment although arteries and veins of suitable size usually run close to each other. Eventually, we succeeded in making measurements of both artery and vein flow velocities for the same animal in only three of 14 dogs. Therefore, artery and vein velocity measurements were performed separately in the remaining dogs (artery, five dogs; vein, six dogs). In three additional dogs, measurements of both artery and vein velocities for the same animal were successfully carried out in one dog, and separate velocity measurements in an artery or a vein were performed in the other two dogs. The change in vascular diameter during the cardiac cycle, especially that in the vein, may cause the variation in velocity measurements. Hellenbrand et al measured the diameter of the anterior interventricular vein angiographically and found little change in the diameter during the cardiac cycle. A preliminary study we performed with a 25-MHz ultrasound echo system (spatial resolution, 0.2 mm) showed that the configuration of the anterior interventricular vein was changed slightly from round to ellipsoid during the cardiac cycle, but that the cross-sectional area remained almost unchanged (authors’ unpublished data). Therefore, the change in the vascular diameter of atrial veins may be of only minor importance for interpretation of the velocity waveform.

One of the major aims of our work was to study the previously unreported phase relation between the inflow and outflow of the atrial myocardium. This relation is extremely important to our understanding of the atrial coronary circulation in the beating heart. We found a phase opposition between arterial inflow and venous outflow, that is, a dip formation with reverse flow in the artery and a predominant forward peak flow velocity in the vein during atrial contraction (see Figure 7). The temporal coincidence between the dip formation on the atrial artery flow velocity and atrial contraction was confirmed during atrial flutter with an atrioventricular block. The phase opposition cannot be explained without considering the possibility of a relatively large intramyocardial capacitance in the left atrium. We have previously investigated the functional characteristics of the intramyocardial capacitance vessels of the canine left ventricle during prolonged diastole by analyzing coronary venous flow after stepwise increases in coronary artery pressure after coronary artery occlusion. Great cardiac vein flow resumed after a delay of 1–2 seconds after restoration of artery flow. Venous flow subsequently increased with a first-order time delay. We have explained our results using a model incorporating an “unstressed volume” in addition to vascular capacitance and resistance. During systole, blood is squeezed from the intramyocardial vessels, which partially or totally collapse. The diastolic refilling of these vessels requires a finite volume before any appreciable pressure develops. These results suggested to us the possibility of significant intramyocardial vascular capacitance in the atrial myocardium. To be specific, the greater part of the artery inflow into the atrial myocardium during ventricular systole may be stored in the atrial unstressed volume since the vein outflow from the atrium during this period was negligibly small. Therefore, we roughly estimated the blood volume stored in the left atrial unstressed volume from the amount of the artery inflow during ventricular systole. We approx-
imized the value as one fourth of that of the left ventricular myocardium stored during ventricular diastole. This is based on the following reports: 1) the systolic atrial artery flow was almost equal to the diastolic flow in the atrium,11 and 2) the left atrial blood flow was about one half of that in the ventricular myocardium.21-23

Since the tension that developed within the left atrial myocardium during atrial contraction is less than that within the left ventricle during ventricular contraction, can atrial contraction squeeze the blood in the capacitance vessels out into the vein? Although the intraluminal pressures in intramyocardial veins and venules may be small, the radial stresses developed during atrial contraction are unlikely to be sufficient to propel a large amount of blood into the venous outflow. Therefore, it is likely that active stresses at right angles to the radial direction generated by contracting muscle may play a major role in the displacement of the blood from the capacitance vessels to the venous outflow. This is inferred from the report of Baird et al24 that the coronary artery flow in the beating empty heart showed a similar velocity waveform (diastolic-predominant pattern) to that observed when the heart was generating pressure. This similarity indicates the importance of tangential stress in the impedance to coronary inflow and the promotion of coronary outflow. The myocardial fiber strain during atrial contraction may also contribute to the compressive force exerted on the capacitance vessels.

Little has been published on the phasic characteristics of blood flow velocities in the atrial circulation. To our knowledge, there is no available data on the atrial vein flow velocity. Bing et al12 and Hellberg et al13 first studied the coronary microcirculation of the cat left atrium by a transillumination method. They reported that two major peaks in red blood cell velocities were observed in the capillary under control conditions; the first is a large peak immediately preceding or at the onset of ventricular systole, and the second is a small peak immediately after aortic valve closure. As they stated, it is difficult to determine detailed phasic movement of red blood cells in the capillaries during the various phases of the cardiac cycle. However, the outline of the red blood cell velocity pattern in the capillaries under control conditions is similar to the velocity waveform that we observed in the small vein although the forward flow continues throughout the cardiac cycle in the capillaries. The similarity of the velocity waveform in the small veins and the capillaries may indicate that the capillaries can act as capacitance vessels in a similar manner to the veins. The presence of forward flows throughout the cardiac cycle may be one of the characteristics of the capillary blood flow. White and colleagues10,11 measured the blood flow velocity in the sinus node artery in the dog to study the effect of atrial fibrillation on the atrial blood flow and mechanics. They reported that the phasic blood flow in the sinus node artery was similar in systole and diastole. Although they did not discuss the velocity waveform itself in detail, the peak velocities of the sinus node artery can be seen during systole in their original tracings (Figure 3 in Reference 10 and Figure 1 in Reference 11). This is consistent with our observations and indicates that the outline of the blood flow velocity waveform between the left and right atria does not differ although the detailed configuration (phase and amplitude) of the velocity pattern may.

Coronary-luminal communications in the left atrium may modify the blood velocity waveforms of the atrial artery and vein flows. Part of the artery flow may enter directly into the left atrium through the arterioluminal channels, and some blood flow may exist between the atrial vein and the atrial lumen through the thebesian veins. However, the anatomic channels of the coronary-luminal communication are reported to be much more prominent in the right ventricle and atrium.25 Thus, their physiological importance in the left atrium may be small.

Isoproterenol administration accentuated the transient decrease in arterial flow velocity during atrial contraction and caused flow reversal in five cases out of eight, but it increased acceleration of forward flow velocity in the veins during atrial contraction (see Figures 3 and 4 and Table 1). Since the atrial pressure was not changed significantly by isoproterenol, the radial stresses may not differ greatly from those under control conditions. This again indicates the importance of active stresses at right angles to the radial direction generated by contracting muscle on the coronary inflow into and the outflow from the left atrial myocardium. Bellamy26 analyzed the origin of the atrial cove, that is, the transient decrease in the left anterior descending and the left circumflex coronary artery flows that occurs during atrial contraction. He indicated that the flow reduction is a direct function of the increase in the left ventricular intramyocardial pressure. This intramyocardial pressure rise is considered to be a passive phenomenon caused by the ejection of blood into the left ventricle by atrial contraction. However, it is unlikely that a passive rise in left atrial intramyocardial pressure in the left atrial myocardium can produce reverse flow in the atrial artery during atrial contraction although such an increase in pressure may contribute partly to a decrease in forward flow. Recently, McHale and Greenfield27 indicated significant importance of mechanical contraction of the atrial musculature for the cove in the left circumflex coronary artery. The "stop-cork effect" of Spaan28 may be helpful in understanding the arterial flow reduction during atrial contraction.

After nitroglycerin administration, the resumption of venous flow velocity during atrial relaxation occurred earlier than that under control conditions. This may have been due to earlier saturation of the unstressed volume by the increase in the arterial inflow. Although isoproterenol and nitroglycerin
modified the velocity waveforms in the arteries and veins on the left atrium, their general characteristics remained unchanged from control values. These drugs may change the vascular diameter from that under control conditions, and this change in diameter could result in the variation in velocity measurements. Therefore, we decided not to discuss changes in the absolute value of the blood velocity after drug administration in any detail.

After premature ventricular contraction, the left atrial pressure was higher than that of sinus rhythms. However, both the depth of the systolic artery dip and the peak forward velocity in the vein were smaller than those of sinus rhythms. These results may again indicate that the magnitude of the left atrial pressure does not correlate directly with the depth of the dip on the artery velocity waveform and the peak vein flow velocity. The higher instantaneous artery pressure during ventricular premature contraction may partly contribute to the smaller atrial artery dip. We speculate that the blood volume pooled in the atrial capacitance vessel before atrial contraction and contracting muscle force are the major factors contributing to the phase opposition between atrial inflow and venous outflow.

The present experiments were performed in an open-chest–open-pericardium canine model. This preparation may affect the arterial and vein blood velocities in the following ways. First, it has been reported by Maruyama et al.29 that the size of the heart chambers increased after pericardectomy under constant intracardiac pressure. This enlargement of the atrium may increase its developed active stresses and possibly result in an enhancement of the phase opposition between the artery and vein blood velocities. Second, the presence of pericardium may allow the atrial myocardium to be more effectively compressed by an increase in the left atrial pressure. In addition, the pericardium may augment the pressure interaction between the left atrium and other cardiac chambers.29 However, the effect of the passive radial stresses caused by these factors may be relatively small for the aforementioned reasons. Third, there may be a direct effect of intrapericardial pressure and/or pericardial surface pressure on the atrial arteries and veins, especially on the veins. That is, one might expect pericardectomy to lower venous resistance by removing the compressive force on the veins. If the waterfall mechanism acts in the epicardial vein30 on the left atrium with pericardium, the zero-flow pressure may be decreased by pericardectomy.31 However, the direct effect may be too small to cause a significant change in the general characteristics of the atrial artery and vein blood velocity waveforms although it might change the absolute values of the velocities slightly.

In summary, we measured the blood flow velocities in the small arteries and veins on the left atrium using our LDV with an optical fiber. The velocity waveform in the artery was similar to the pattern of aortic pressure during ventricular ejection but was characterized by a pronounced dip during atrial contraction. The vein flow velocity was characterized by a prominent atrial systolic flow. The phase difference between inflow and outflow indicates that a large portion of the coronary inflow to the atrial myocardium may be stored due to the presence of atrial intramyocardial vascular capacitance (probably unstressed volume and ordinary capacitance). We concluded that atrial myocardial contraction impedes the arterial inflow (the systolic dip) and promotes the venous outflow from atrial capacitance vessels.

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References


KEY WORDS • left atrial artery and vein • blood velocity waveform • Laser Doppler velocimeter • atrial contraction and relaxation
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