Phasic Blood Flow Velocity Pattern in Epimyocardial Microvessels in the Beating Canine Left Ventricle

Kouichi Ashikawa, Hiroshi Kanatsuka, Toshimi Suzuki, and Tamotsu Takishima

We quantitated phasic epimyocardial microcirculatory coronary blood flow velocity patterns in the beating left ventricle. Using a newly developed floating objective and high-speed cinematography, red cell velocities in small arterioles, capillaries, and small venules and microvascular diameters in the superficial layer of the epimyocardium of beating left ventricle were determined throughout the entire cardiac cycle in open-chest anesthetized dogs. Heart rate was maintained at 140 beats/min by means of left atrial pacing. Peak red cell velocity was observed in midsystole in small arterioles and capillaries, and in late systole in small venules.Abrupt decline in red cell velocity and, in many cases, a momentary cessation or reverse of flow, was observed in these microvessels during the pre-ejection period. The internal diameter of small venule was increased in late systole, while that of small arteriole remained almost constant during the cardiac cycle. Furthermore, in these epimyocardial microvessels, a higher percentage of the total area under the velocity curve occurred during the ejection phase; 51% in small arterioles, 43% in capillaries, and 40% in small venules. These findings indicate that the phasic blood flow pattern is markedly different in the subepimyocardial microvessels from that in the large epicardial artery and the septal artery. During vasodilatation following dilazep (50 μg/kg, i.v.), an adenosine potentiator, red cell velocity increased throughout the entire cardiac cycle in epimyocardial microvessels with significant increases in the total area under the velocity curves accompanied by significant dilation of the arterioles. The present data will provide information useful in predicting or simulating transmural differences in the phasic blood flow pattern. (Circulation Research 1986;59:704–711)

It is well established that the main inflow in the epicardial large coronary artery occurs during diastole.1 However, it has been suggested that the phasic blood flow pattern in the epicardial coronary artery does not correctly reflect the intramyocardial flow pattern.2–4 Direct and continuous analysis of flow patterns in intramyocardial microcirculation would provide information useful in understanding this discrepancy. Bing and his colleagues5 first reported phasic blood flow velocity patterns in the capillary in the cat left atrium by means of an intravital microscope and a high speed camera. However, in the ventricle, continuous microscopic observation of coronary microcirculation has been almost impossible in situ beating mammalian heart because of technical difficulties, i.e., mainly focusing problems that result from the continual variation of the distance between the heart and the microscopic objective during cardiac contraction.

Recently, using a floating objective, we have developed a new microscope system that allows direct and continuous observation of coronary microcirculation in the beating canine left ventricle.6 In the present study, we measured internal diameters and phasic blood flow velocity patterns in arterioles, capillaries, and venules in the epimyocardium of the left ventricle in the control condition and after administration of dilazep, an adenosine potentiator.

Materials and Methods

Young mongrel dogs of both sexes, weighing 4–8 kg, were anesthetized with an intravenous injection of urethane (500 mg/kg) and chloralose (60 mg/kg). If necessary, additional doses were given to maintain anesthesia. The animals were ventilated through auffed endotracheal tube with a Harvard respirator. A positive end-expiratory pressure of 3 to 5 cm H₂O was introduced to prevent atelectasis of the lung. Arterial blood gases were kept within the normal range by adjusting ventilation volume and/or rate, as needed. Metabolic acidosis during anesthesia was prevented by an intravenous infusion of sodium bicarbonate, which maintained arterial pH at approximately 7.40. A polyvinyl catheter was introduced into the external jugular vein for drip infusion. A lead II ECG was monitored. A thoracotomy was performed in the fifth left intercostal space, and the heart was suspended in a pericardial cradle. A plastic wrapping was used to separate the lung from the anterior aspect of the heart. Aortic pressure was measured in the aortic root with a catheter (30-cm length) passed through the right carotid artery.
A 16-gauge teflon tube (15-cm length) was passed into the left ventricle through the apex for recording the left ventricular pressure. Pressures were measured with a Statham strain gauge (Model P 23). The time delay of our fluid-filled systems for measurement of pressure was less than 2 milliseconds compared with solid-state transducer (Millar Instruments, Inc., Houston, Tex.). Heart rate was kept constant at 140 beats/min by means of left atrial pacing after sinoatrial block, which was produced by injecting formaldehyde into the region of the sinus node. The preparation was kept moist during experiment by continuously dripping warm Krebs-Ringer solution (NaCl, 118.2 mM; KCl, 4.7 mM; CaCl₂, 2.5 mM; MgSO₄, 1.2 mM; KH₂PO₄, 1.2 mM; NaHCO₃, 25 mM; calcium disodium EDTA, 0.026 mM; and glucose, 5.5 mM; maintained at 37°C and pH 7.4) on the cardiac surface. Rectal temperature was maintained at about 37°C with a heat blanket. To reduce excessive vertical movement of the heart, two 24-gauge steel needles (5 to 7 mm distance) were horizontally inserted through the midmyocardium of the left ventricle (approximately 5 mm from the surface) beneath the area of interest. The end of each needle was fixed to a needle holder that was held with coil springs. This apparatus allowed the heart to move perpendicularly, but it limited excessive horizontal movements in order to hold the transilluminated area in the microscopic field of view. The ECG, aortic and left ventricular pressures were recorded on a Rectigraph (Type 8k 12-1S-ME, San Ei Sokki, Tokyo) at a paper speed of 100 mm/sec.

There are a number of technical difficulties involved in direct and continuous observation of coronary microcirculation. The greatest difficulty is the variation of the distance between the microscopic objective and the heart introduced by the cardiac contraction. This difficulty can be overcome by a floating objective system. The theoretical considerations and design of the microscope system using our floating objective have been previously reported. In brief, the floating objective system consists of a pair of convex lenses that transmit the real image to a standard microscope without any change in magnification. That is, the real image on the front focus of a convex lens facing the heart is transmitted to the back focus of another convex lens. This principle is not affected by variation in the distance between these two convex lenses. This real transmitted image is then observed with the objective of a standard microscope. The weight of the floating objective was reduced by mounting it in a thin aluminum tube (total weight of approximately 16 g, Figure 1). This minimal weight permits the floating objective to follow the cardiac motion because the inertial force in vertical movement is minimum. The floating objective was supported by a weight-adjusting coil spring and low-resistance ball bearings, which permitted the lens to move perpendicularly in unison with the cardiac motion. The distance between the floating lens and the heart was adjusted to the focal distance of this lens.

Direct visualization of coronary microcirculation was accomplished by transillumination of the epicardium of the left ventricle with a xenon arc lamp. A light-conducting glass fiber (0.6-mm diameter), which was introduced through the lumen of a 20-gauge stainless steel needle, was inserted into the subepicardial muscle layer of the left ventricle using a micromanipulator. The needle was fixed to a needle holder in a manner that allowed the tip of the needle to move up and down in unison with the cardiac motion. Thus, the illuminating fiber, the floating objective, and the myocardium all move as a unit in the system used. Moreover, in order to prevent compression of the tissue in the microscopic field of view, the floating objective was lifted by an arm connected to the needle holder and was lightly positioned just above the surface of the heart (Figure 1). To verify the depth of the microvessels below the epicardium, the microscope was focused on the surface of the area of interest and then focused on a visualized vessel within the epicardium. The number of scales of the fine focus adjustment knob required to focus on the vessel was then translated to the vertical displacement of the microscopic objective. In this way, the depth below the epicardium of the visualized vessels ranged from 30 to 200 μm in this study. The beam splitter view of the microscopic image was monitored by means of a rotary shutter camera (shutter speed 1/1800 sec; RSC 3000A, Sony) and a video motion analyzer (SVM-1110, 200/Am in this study. The beam splitter view of the microscopic image was monitored by means of a rotary shutter camera (shutter speed 1/1800 sec; RSC 3000A, Sony) and a video motion analyzer (SVM-1110,
Sony). The video motion analyzer employs a disc video-sheet recording system making it possible to obtain clear motion and still pictures and is useful for confirming good focus on the film. After focusing, motion pictures were taken at 500 frames per second with a 16-mm high-speed motion picture camera (Milliken DBM-5D). High-speed ektachrome film (Eastman Kodak 7251) was used with a 100-millisecond exposure time. Timing flashes at 10-millisecond intervals and signals that were synchronized with the R wave of the ECG were simultaneously recorded on each edge of the film to verify the film speed and to correlate with hemodynamic data. The optical magnification was 100 × or 150 × and was reduced to one-fourth on the film.

Red cell velocities in coronary microvessels were calculated by frame to frame analyses from the distance of cell progressions on a projection screen and the numbers of frames needed. In arterioles and venules, red cell velocities could be calculated only in precapillary or terminal arterioles (mean diameter 12.8 ± 4.1 μm) and in collecting venules (mean diameter 16.5 ± 6.5 μm). In this size microvessels, it was possible to track cell-to-cell or plasma pocket progression on the high speed film. The distance of cell or plasma pocket progression in serial frames was calculated as the difference in distances from a special marker on the vessel, usually the bifurcation, since the vessel was moving on the projection screen as in the heart. Arterioles could be easily differentiated from venules by the direction of flow at the bifurcation. The optical magnification on the projection screen and the film speed were confirmed by a reference scale and timing flashes, respectively. The red cell velocity curve was obtained from a plot of red cell velocity that was measured every 20–40 milliseconds during the entire cardiac cycle. The internal diameter of arterioles and venules were also measured during a cardiac cycle. Phasic diameter changes were estimated in small arterioles (mean diameter 24.9 ± 11.0 μm) and in small venules (mean diameter 27.7 ± 11.6 μm), the vessel walls of which were clearly recognizable on the projection screen. The diameter and the velocity curve were then correlated to ECG, aortic, and left ventricular pressure curves.

The area under the red cell velocity curve was planimetrically measured during the different segments of a cardiac cycle; isovolumic systole, ejection phase, and diastole.

The different segments of the cardiac cycle were determined from the aortic and left ventricular pressure recording. The areas under the red cell velocity curve during different segments of a cardiac cycle were expressed as percent of total area under the red cell velocity curve.

To verify the resolution of the internal diameter measurements, we have recently employed an incident light fluorescence microscope (Nikon XF-EFD, Tokyo), which is attached to our floating objective system. Briefly, the surface of the heart is illuminated by incident light from a mercury lamp. The maximal wave length of illumination is 495 nm using B2 and interference filters. The emitted light passes through a 510 nm filter. Contrast of the microvasculature is enhanced by left atrial injection of Fluorescein Isothiocyanate Dextran (molecular weight 40,500, Sigma). By means of a highly sensitive television camera (C 1000-12, Hamamatsu TV, Hamamatsu), continuous observation of the enhanced image is recorded on a videocassette recorder (VO-2710, Sony) and printed out on a video graphic printer (UP-701, Sony). In 5 arterioles, the internal diameter was estimated by both cineangiographic and fluorescence methods. There was no significant difference between these two measurements of internal diameter. In addition, we also estimated the response of coronary microvessels to a coronary vasodilator. We measured the change in arteriolar diameter and the area under the velocity curves from the control area during the different segments of the cardiac cycle following administration of dilazep (50 μg/kg, i.v.), an adenosine potentiator.

Results

Hemodynamic and arterial blood gas data are shown in Table 1. The parameters were kept in the normal range throughout the experiment. Heart rate was maintained constant at 140 beats/min by means of left atrial pacing.

Figure 2 shows mean red cell velocity curves in small arterioles, capillaries, and small venules in the epimyocardium of the beating canine left ventricle. In small arterioles (diameter range of 9 to 20 μm; 12.8 ± 4.1 μm, mean ± SD) and capillaries, peak red cell velocity occurred in midsystole, followed by a decrease during diastole. Abrupt decline in red cell velocity and a momentary (20–30 millisecond duration) reverse flow were observed in these microvessels during the pre-ejection period (Figure 2A). In small venules (diameter range 9 to 30 μm; 16.5 ± 6.5 μm, mean ± SD), red cell velocity reached its peak in late systole, followed by gradual decline during diastole. Momentary cessation or, in many cases, reverse flow was also noted in small venules (Figure 2B).

Figure 3 demonstrates an epimyocardial terminal arteriole that was obtained by the fluorescence microscopic method.

Table 2 shows phasic changes in the internal diameter of small arterioles and small venules. In small arterioles, no significant change in diameter was observed during a cardiac cycle under normal conditions. On the other hand, small venules were significantly dilated in late systole when red cell velocity was maximum (14.2 ± 8.2%; p < 0.05 compared with diastole).

Figure 4 illustrates the percent of total area under the red cell velocity curve during different phases of the cardiac cycle in small arterioles, capillaries, and small venules. During isovolumic systole, the percent of the total area under the velocity curve was minimum (mean ± SEM, 1.9 ± 1.9% in small arteriole, 4.4 ± 0.8% in capillary, and 6.4 ± 1.0% in venule). A considerably higher percent of the total area under the velocity curve occurred during the ejection phase in the
epimyocardial microvessels (mean ± SEM, 51.4 ±
3.6% in small arteriole, 43.6 ± 2.0% in capillary, and
40.0 ± 1.8% in small venule). During diastole,
approximately half of the total area under the velocity
curve occurred in epimyocardial microvessels
(mean ± SEM, 47.6 ± 5.5% in small arteriole,
52.5 ± 3.1% in capillary, and 55.2 ± 2.2% in small
venule).

Table 3 shows hemodynamic and arterial blood gas
data before and after dilazep administration. Heart rate
was maintained constant at 140 beats/min throughout
the experiments by means of the left atrial pacing.
Arterial blood gas and pH were maintained within their
physiological ranges. Administration of dilazep result-
ed in a decline in systemic blood pressure. Systolic
aortic pressure returned to control level within about
10 minutes, while diastolic aortic pressure remained
slightly but significantly lower following dilazep in-
jection. Measurements were performed 10–15 minutes
(mean 12 minutes) after dilazep injection.

After administration of dilazep, red cell velocity
curves in epimyocardial microvessels were shifted up-
ward throughout the entire cardiac cycle, with a sig-
nificant increase in the total area under the velocity
curves (137% from control in arterioles, 128% in cap-
illaries, and 130% in venules).

Figure 5 shows the effects of dilazep on the phasic
blood flow patterns in the small arterioles (14.0 ± 2.2
µm diameter) (Figure 5A), capillaries (Figure 5B),
and small venules (18.5 ± 2.2 µm in diameter) (Figure
5C). The percent area under the velocity curves signifi-
cantly increased in all different phases of the cardiac
cycle in these microvessels.

Figure 6 shows the typical response of a small arteri-
ole after administration of dilazep. The average response to dilazep is shown in Figure 7. The response of the internal diameter was estimated in the mean arteriolar size range of 27.5 ± 3.6 μm (mean ± SEM) in the control condition (range, 11.8–48.8 μm), in which the vessel walls were clearly recognizable. The internal diameters of small arterioles were significantly increased from 27.5 ± 3.6 μm to 35.8 ± 4.7 μm (130.6 ± 4.8%, p < 0.001), following dilazep administration.

Discussion

The major importance of this study is that it is the first continuous analysis of blood flow velocity patterns in the epimyocardial microvessels of the beating mammalian left ventricle throughout the entire cardiac cycle. The data demonstrate that 1) the phasic blood flow velocity pattern is apparently different between the epimyocardial microvessels and the epicardial coronary artery and the septal artery, which has been reported by others; 2) in the epimyocardial microvessels, blood flow velocity is accelerated during systole and reaches a peak in midsystole in small arterioles and capillaries and in late systole in small venules; and 3) abrupt decline of blood flow and momentary cessation or reverse flow is observed in the subepimyocardial microvessels.

Furthermore, the vasodilator effects of dilazep, an adenosine potentiator, on small coronary arterioles were directly visualized and estimated in the in situ beating left ventricle.

In our experimental preparation, mechanical factors that disturb the coronary microcirculation are eliminated as much as possible. In spite of our technical care taken, the insertion of the illumination fiber and the heart-stabilizing needles may have had some influence on the behavior of the microvessels. However, the effect should be minimal, since the red cell velocities in the microvessels did not significantly change for more than 2 hours in the control condition in our experimental preparation. Moreover, the minimal weight of the floating lens allows one to follow the cardiac motion throughout the entire cardiac cycle because of the minimal inertial force in vertical movement. By means of an atrium transillumination technique, Bing and colleagues have reported phasic red cell velocity patterns of capillaries in the left atrium of the cat heart. They have reported two peaks in the red cell velocity, which were considered to correlate with atrial contraction.

Using a pulsed Doppler flow meter, Marcus et al and Chilian and Marcus reported that peak blood velocity was observed during diastole in the epicardial coronary artery and the septal artery in the beating canine and human heart. They also showed that a large percent of the total coronary blood flow velocity oc-

Table 2. Phasic Change of Internal Diameter in Small Arterioles and Venules

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>25</th>
<th>75</th>
<th>125</th>
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<td>11.7</td>
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<tr>
<td>Venules</td>
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<td>Mean</td>
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<td>30.4</td>
<td>30.7</td>
<td>32.2*</td>
<td>31.1</td>
<td>29.3</td>
<td>28.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD</td>
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<td>13.2</td>
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<td>14.1</td>
<td>14.0</td>
<td>12.7</td>
<td>11.7</td>
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</table>

n = number of measurement; *p < 0.01 vs. 25 and 385 msec. Statistical significance was analyzed using the paired t test.
Table 3. Hemodynamics and Blood Gases Before and After Dilazep Administration

<table>
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<tr>
<th>Dilazep administration</th>
<th>Systolic aortic pressure (mm Hg)</th>
<th>Diastolic aortic pressure (mm Hg)</th>
<th>Heart rate (beats/min)</th>
<th>PaO₂ (mm Hg)</th>
<th>PaCO₂ (mm Hg)</th>
<th>pH</th>
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<td>94±14</td>
<td>140</td>
<td>95.2±13.4</td>
<td>35.9±2.6</td>
<td>7.41±0.06</td>
</tr>
<tr>
<td>After</td>
<td>137±11</td>
<td>85±12*</td>
<td>140</td>
<td>94.5±10.2</td>
<td>35.0±2.9</td>
<td>7.42±0.06</td>
</tr>
</tbody>
</table>

Data are means ± SD. n = 5.
*p < 0.05 vs. before dilazep administration.

Figure 5. Percent of the area under the red cell velocity curves during different phases of the cardiac cycle in epicardial arterioles (panel A, n = 5), capillaries (panel B, n = 6), and venules (panel C, n = 5) before and after dilazep injection. *p < 0.05 vs. before drug.

...curred predominantly during diastole in the left anterior descending coronary artery (75%) and the septal artery (92%). There may be no doubt that the phasic nature of blood flow velocity from the septal artery represents a composite of epicardial (right side of the septum), midmyocardial, and subendocardial instantaneous blood flow velocity.

In the present study, we have shown that the peak blood flow velocity was observed in the ejection phase and that a significantly higher percent of the total area under the velocity curve occurred during the ejection phase in the epimyocardial microvessels of the beating left ventricle.

It has been suggested that the blood flow pattern in the epicardial artery does not necessarily correctly represent the intramural flow mainly because of the capacitance of the epicardial arteries and intramyocardial capacitance effects. Chilian and Marcus have reported that epicardial capacitance is inversely related to perfusion pressure. Retrograde flow in the septal artery during systole has also been reported. Furthermore, Chilian and Marcus found that increasing extravascular forces (right ventricular hypertension) augmented retrograde flow in the septal artery and suggested that the phasic nature of intramyocardial blood velocity is intimately related to extravascular pressure. Since systolic retrograde flow has never been recognized in a large epicardial artery under normal perfusion, it has been supposed that negative systolic flow must be stored in the capacitance of the epicardial arteries. Furthermore, it has been reported that the epicardial capacitance function is large enough to store all of a normal systolic stroke flow. Systolic expansion of the epicardial coronary artery has also been reported by direct measurement of arterial diameter. These concepts of capacitance effects are convenient for explaining why the epicardial coronary blood flow pattern does not reflect correctly the intramyocardial flow pattern. Thus, it is clearly impossible to predict phasic blood flow patterns in coronary microvessels of each myocardial layer from measurements of phasic blood flow in the large coronary artery.

It is well known that cardiac contraction inhibits coronary inflow probably because of its extravascular compression effect. On the other hand, by means of radioactive microsphere methods, Hess and Bache demonstrated that subepicardial perfusion was normal...
even when coronary inflow was limited to either systole or diastole. From these results, they suggested that in the outer layers of the ventricular wall extravascular pressure is a relatively minor determinant of myocardial blood flow. Russell et al.\textsuperscript{17} have also indicated that virtually no extravascular effects are present at the subepicardium. However, they could not demonstrate the intramyocardial blood flow pattern because of methodological limitations. With a direct observation, we found no significant change in the internal diameter of subepimyocardial small arterioles or capillaries during the cardiac cycle. This lack of significant change in microvascular diameter in the subepimyocardium during the cardiac cycle seems to suggest that myocardial contraction does not greatly affect the vascular diameter in this region under normal conditions, probably because of lower intramyocardial pressure, compared with a relatively high perfusion pressure throughout the entire cardiac cycle.\textsuperscript{18-21} This also suggests that the pressure in these microvessels are relatively constant during the course of the entire cardiac cycle and implies that there may be substantial capacitance-smoothing pressure changes in these small vessels, keeping them relatively constant. This may be compatible with the assumption of Spaan et al.\textsuperscript{10} that coronary arterial resistance remains constant during the cardiac cycle under normal perfusion.

The lack of significant change in internal diameter indicates that red cell velocity patterns in small arterioles and capillaries reflect the patterns of volume flow in these microvessels, because volume flow is proportional to the product of blood flow velocity and cross-sectional area in small vasculature.\textsuperscript{22} Thus, from the direct and continuous analysis of red cell velocity patterns in the subepimyocardial microvessels, the present study shows not only that blood flow velocity is accelerated during the ejection phase, but also that a considerable amount of myocardial perfusion occurs during systole in the epimyocardium (approximately 50% of a stroke flow in small arterioles).

Tillmanns et al.\textsuperscript{23} have reported the phasic changes in red cell velocities in the epimyocardial microvessels of the turtle heart. However, because of technical difficulties, they could observe the coronary microcirculation only during very short and discontinuous periods of the cardiac cycle. Continuous observation of microcirculation is more advantageous for detecting small changes in the blood flow velocity patterns in microvessels. Although they were unable to observe retrograde flow during the cardiac cycle, they observed it during a very short portion (20-40 milliseconds) of the preejection phase. They suggested a 180° phase shift in blood flow from the arterioles to the adjoining capillaries in the epimyocardium of the turtle heart; i.e., arteriole red cell velocity diminished during systole, while capillary and venule red cell velocity increased during this period. However, we have never observed such a 180° phase shift in blood flow from the arterioles to the adjoining capillaries in \textit{in situ} beating canine left ventricle. Since no narrowing or collapsing of epimyocardial microvessels was observed, it is unlikely that such a phase shift in blood flow occurs in these microvessels under normal conditions. These differences may result from the difference in the experimental preparation and/or the species.

The present study provides suggestive information that may contribute to the understanding of the transmural distribution of phasic myocardial perfusion.

First, backward flow in the epimyocardial microvessels may suggest 1) the compression of downstream veins during preejection, or 2) the existence of...
an intramyocardial pumping action as suggested by Spaan et al. However, we were unable to find the site of volume change in our microscopic field of view, although we did not observe more distal, large veins. As pointed out by Spaan et al., a large change in volume may not be needed to explain this relatively small portion of reverse flow. Second, rapid acceleration of blood flow velocity following the backward flow may suggest that during early systole the epicardial layer of the left ventricle is perfused by both forward flow from the epicardial artery and probably a part of backward flow from the deeper myocardial layer. The backward flow in the septal artery has been reported during isovolumic and early systole by others.

After reaching its peak velocity, the blood flow velocity in the epicardium rapidly decreases, probably because blood flow pours into the deeper myocardial layer as soon as the extravascular compression is released in that region. As a result, cyclic blood flow shift may occur between the superficial and the deeper myocardial layers in the left ventricular wall. Hamlin et al. have reported during isovolumic and early systole by others. Since the extent of extravascular compression is supposed to be different in each transmyocardial layer, it is expected that the phasic blood flow pattern will be considerably different from the epicardium to the endocardium. Hamlin et al. have reported that there is a linear reverse relationship between the intramyocardial pressure and the regional myocardial blood flow across the left ventricular wall during systole.

In conclusion, the present study is the first to demonstrate the phasic blood flow pattern in the epicardial microvessels throughout the entire cardiac cycle in situ beating mammalian left ventricle. Although the present analyses are limited to the epicardium, the results suggest that the phasic blood flow pattern is considerably different across the left ventricular wall and that it is impossible to predict the phasic blood flow pattern in each myocardial layer from measurements of phasic blood flow pattern in the large coronary artery. It is thought that the present study will provide important information in understanding the transmural differences in phasic blood flow pattern in the beating left ventricle.

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References

Key Words: intravital microscope • floating objective • epicardial microvessels • phasic blood flow velocity • adenosine potentiator
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