

Circulation Research

JOURNAL OF THE AMERICAN HEART ASSOCIATION



Phasic coronary blood flow velocity in intramural and epicardial coronary arteries. W M Chilian and M L Marcus

Circ Res. 1982;50:775-781

doi: 10.1161/01.RES.50.6.775

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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Phasic Coronary Blood Flow Velocity in Intramural and Epicardial Coronary Arteries

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SUMMARY. Knowledge concerning phasic coronary blood flow is based primarily on measurements obtained from epicardial coronary arteries, which, in part, function as capacitors. If present, epicardial capacitance effects could obscure the dynamic nature of phasic intramyocardial perfusion. To analyze this effect of epicardial capacitance, we simultaneously measured coronary blood flow velocity in an epicardial artery (left anterior descending) and an intramural artery (septal) in open-chest, anesthetized dogs. During control conditions, the percentage of total coronary blood flow velocity occurring during diastole per cardiac cycle was significantly greater ($P < 0.05$) in the septal artery (92%) than in the left anterior descending artery (75%). Furthermore, blood flow velocity during mid-systole in the septal artery was retrograde (-7.2%), whereas blood flow velocity at this time was antegrade in the left anterior descending artery (+3.5%). Blood flow velocity measurements from small epicardial arteries just before they penetrated into the myocardium revealed a phasic pattern similar to that of the septal artery. This suggests that the phasic blood velocity pattern in penetrating coronary arteries, in general, is different than that in large epicardial arteries. During vasodilation following nitroglycerin, dipyridamole, or a 20-second occlusion of the left main coronary artery, the retrograde component of mid-systolic blood velocity persisted in the septal artery, despite large increases (300–400%) in the mid-systolic antegrade component of blood flow velocity in the left anterior descending artery. These qualitative and quantitative differences in phasic blood flow velocity between intramural and large epicardial arteries are best reconciled by postulating the existence of a significant coronary capacitor. (*Circ Res* 50: 775–781, 1982)

IN 1963, Gregg and Fisher speculated about devices that measure phasic coronary blood flow in the following manner:

“Since the change in mean vessel tone during a cardiac cycle in the superficial vessels (in which the flow measurements are made) is presumably different from that of the deeper vessels, such a device measures a combination of ‘intramural’ and ‘extramural’ flow. It does not, therefore, necessarily indicate correctly the intramural flow at all times.”

From 1963 to the present, most knowledge concerning phasic coronary flow patterns has been based on measurements obtained from epicardial coronary arteries. Because the epicardial arteries function in part as capacitors (Douglas and Greenfield, 1970), the phasic flow pattern in an epicardial coronary artery may not accurately reflect the phasic pattern of myocardial perfusion. Eckstein and his colleagues, in 1962, theorized that capacitance of epicardial arteries may distort the actual myocardial perfusion in three ways: (1) the magnitude of retrograde flow during systole is concealed because blood expelled from the deep myocardial layers distends the vessels and is not measured; (2) the antegrade systolic flow that is measured is, in part, a capacitance flow that distends the epicardial arteries; and (3) the measured antegrade diastolic

flow is less than the actual perfusion since the epicardial capacitor would be discharging at this time.

In the canine, the septal artery courses almost entirely intramuscularly in the septum (Donald and Essex, 1954). Therefore, it should not be able to distend as readily as an epicardial artery during systole, and thus should have less capacitive properties. Consequently, measurements of phasic flow in the septal artery should more accurately represent the actual phasic perfusion of the myocardium than such measurements from epicardial arteries. Our hypothesis was that the epicardial arteries are serving as elastic reservoirs of flow; thus, measurements of phasic flow in the septal artery would more accurately represent the dynamic nature of phasic intramyocardial perfusion and reveal capacitance of the epicardial arteries.

Methods

General Preparation

Mongrel dogs of either sex (weight: 18–24 kg) were anesthetized with sodium pentobarbital (30–35 mg/kg iv). Animals were intubated and ventilated with positive end-expiratory pressure (2–3 cm H₂O) using oxygen enriched air. Blood gases and pH were determined frequently and, if needed, the respiratory volume or rate was changed to maintain these variables within normal ranges. The right femoral artery and vein were isolated and catheterized, and the catheters were advanced to the aortic arch and inferior vena cava, respectively. Arterial pressure was measured

This manuscript from the University of Iowa was sent to Robert M. Berne, Consulting Editor, for review by expert referees for the editorial decision and final disposition.

with a Statham strain gauge (model P23) and zeroed to the mid-chest level. A left thoracotomy was made through the 5th intercostal space. The pericardium was incised and a solid state transducer (3 mm in diameter) was inserted in the left ventricle through the apical dimple. The left atrium was catheterized via the left atrial appendage. The peak left ventricular pressure was calibrated against peak aortic pressure, and diastolic ventricular pressure was calibrated against left atrial pressure. The left main coronary artery was isolated, and a snare occluder was placed around it. A 2- to 3-mm segment of the septal artery was then isolated with only 1/3 to 1/2 of the circumference being visible. Thus, our preparation avoided major dissection of the septal artery.

In six animals, atrioventricular block was produced by injecting formaldehyde (0.1–0.2 ml) into the region of the atrioventricular node. After attachment of a unipolar electrode to the LV midwall, the heart was electrically paced.

Measurement of Phasic Velocity

Measurement of coronary blood flow velocity was performed with a specially designed probe consisting of a 20 MHz piezoelectric crystal (0.5–1.0 mm in diameter) housed in a Silastic cup. When the probe was applied to the vessel, the angle between the crystal and blood column was about 45°. The probe was placed on the artery and held securely in place by a vacuum (4–7 torr). The probe was connected to a pulsed Doppler flow meter, which was modified from the description by Hartley and Cole (1974). Probes were placed on both the proximal portions of the left anterior descending and septal arteries, which enabled simultaneous measurements of coronary blood flow velocity in epicardial and intramyocardial arteries. In eight animals, the Doppler probes were also placed on small epicardial arteries at a site before they penetrated into the myocardium. The criterion we established for measurements of phasic blood flow velocity in these small vessels was that the Doppler probe was placed at a site within 0.5 cm of the location of penetration of the artery into the myocardium. We hypothesized this would minimize any capacitance effect on the large "upstream" arteries, and blood flow velocity measurements would reflect that of the intramyocardial vasculature. These velocity data, along with the pressures, were recorded on an oscillographic recorder. Zero blood flow velocity was determined by switching the receiver of the Doppler off, i.e., zero kHz shift detected (electronic zero) and by occluding the main left coronary artery (occlusive zero). In our preparation, the occlusive zero and the electronic zero were identical (Fig. 1), and were determined frequently throughout the experiment.

The area under the blood velocity curve was measured from the coronary blood flow velocity recordings with a computerized planimeter during different segments of the cardiac cycle: isovolumic-systole, mid-systole, late-systole, and diastole (Fig. 2). The different segments of the cardiac cycle were determined from the left ventricular pressure trace, and the pressure at which the aortic valve opened (end of isovolumic systole) was determined from the aortic pressure recording. Mid- and late-systole were defined as equal portions of systole between the end of isovolumic systole and the onset of protodiastole. Diastole was defined as the remainder of the cardiac cycle. Phasic blood flow velocity measurements were obtained in at least six cardiac cycles for each experimental maneuver, and these values were then averaged. To describe the proportion of the area under the blood flow velocity curve which occurred during a given segment of the cardiac cycle, we expressed the area under the blood velocity curve during the particular seg-

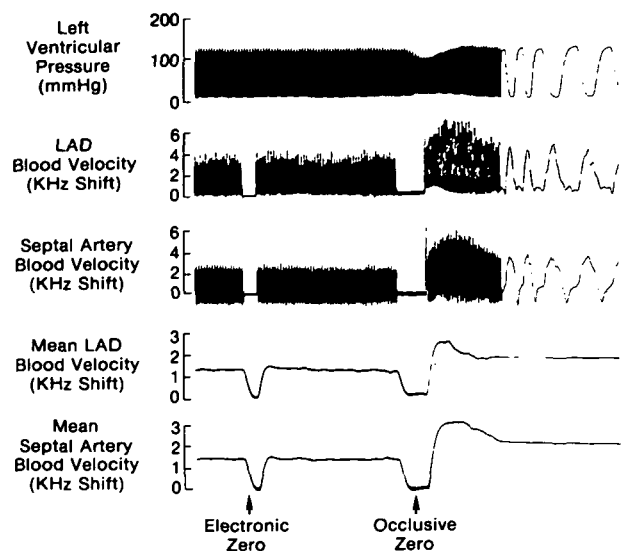


FIGURE 1. A representative recording of the identity of the electronic zero and occlusive zero in both the septal and left anterior descending arteries.

ment of the cycle as a percentage of total area under the blood velocity curve. We also measured the change in the area under the velocity curve from the control area during the different portions of the cardiac cycle following three experimental maneuvers: nitroglycerin (0.4 mg bolus, iv); dipyridamole (20 mg bolus, iv), and reactive hyperemia (post 20-second occlusion). The measurements were obtained during the maximal vasodilator response, as determined from the mean coronary blood flow velocity. Successive interventions were employed only after the coronary blood flow velocity measurements had returned to the control value for at least 5 minutes. Each intervention was duplicated in all experiments.

Research Plan

To examine the phasic character of coronary blood flow velocity in intramural and epicardial coronary vessels, we simultaneously measured coronary blood flow velocity in the septal and the left anterior descending arteries. Since contraction of the septum normally precedes the contraction of the left ventricular free wall, we hypothesized that the sequence of contraction in the left ventricle might affect our results. To exclude this possibility, measurements of phasic coronary blood flow velocity were obtained when the ventricle was paced from the left ventricular free wall. To determine whether the septal artery is an acceptable model of coronary arteries that penetrate the left ventricular free wall, we measured phasic coronary blood flow velocity in the left anterior descending artery, septal artery, and a small epicardial branch of the left anterior descending just before penetration into the myocardium. Because the mid-systolic component of septal artery blood flow velocity was retrograde under control conditions, we examined this retrograde component of systolic blood flow velocity during vasodilation. Under these conditions, mid-systolic antegrade flow velocity in epicardial vessels was markedly increased.

Data Analysis

Hemodynamic variables during the control condition and the three experimental maneuvers to produce vasodilation were analyzed by analysis of variance and the intergroup

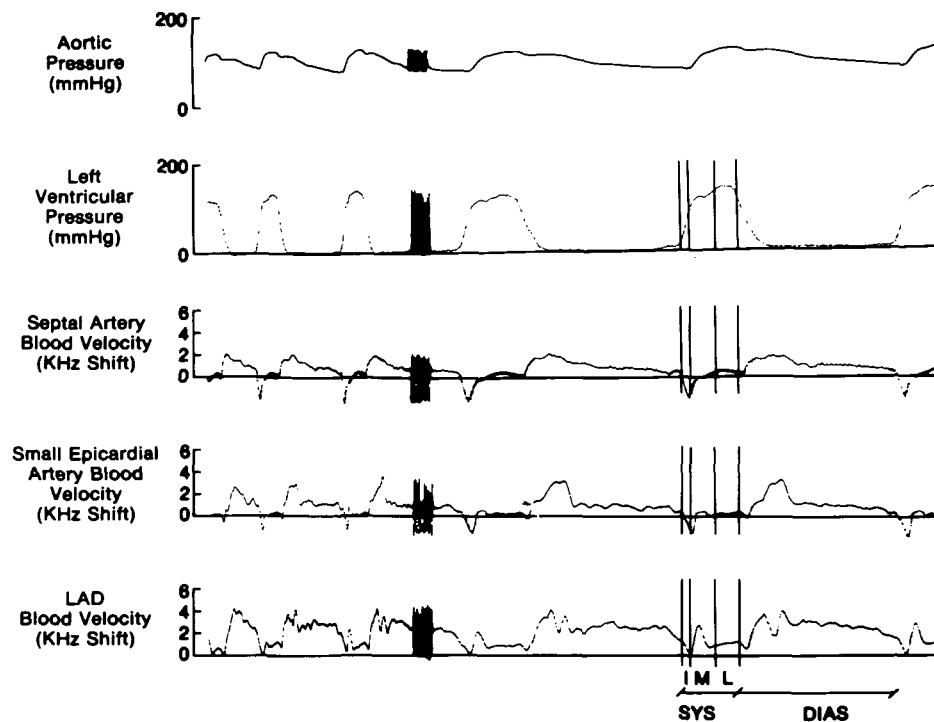


FIGURE 2. A tracing of hemodynamics, phasic septal artery blood flow velocity, phasic left anterior descending artery (LAD) blood flow velocity, and phasic small epicardial artery blood flow velocity. The method of data analysis is also illustrated, with the dissection of the cardiac cycle into systole (SYS), isovolumic-systole (I), mid-systole (M), late-systole (L), and diastole (DIAS). At least six cardiac cycles were analyzed and averaged for each datum, e.g., control isovolumic-systole in the septal artery. The coefficient of variation of the individual blood velocity measurements was usually around 8%.

differences were compared by Duncan's multiple range test. A *t*-test using the Bonferroni inequality was used to compare coronary blood flow velocity in the left anterior descending artery, septal artery, and small epicardial artery during segments of the cardiac cycle. To determine whether a blood flow velocity was antegrade or retrograde, a *t*-test against zero was performed. Coronary blood flow velocity during vasodilation was analyzed by paired *t*-tests using the Bonferroni inequality. A *P* value of less than 0.05 was accepted as statistically significant. Data are represented as mean \pm SEM.

Results

Hemodynamics

The hemodynamic data during the control state, electrical pacing, and during the interventions used to produce coronary vasodilation are shown in Table 1. The parameters were recorded at the time of the peak change in mean coronary blood flow velocity. Admin-

istration of nitroglycerin or dipyridamole produced a significant increase in heart rate and a decrease in arterial pressures. During reactive hyperemia following 20 seconds of left main coronary artery occlusion, heart rate and arterial pressure were not changed from control. Electrical pacing did not produce any significant changes in arterial pressures. Arterial blood gases and pH for the animals averaged: P_{O_2} : 122 ± 13 ; P_{CO_2} : 31 ± 2 ; and pH: 7.36 ± 0.02 .

Phasic Blood Flow Velocity in Septal, Left Anterior Descending, and Small Epicardial Arteries

Figure 3 illustrates the percent of the total area under the blood flow velocity curve that occurs during different segments of the cardiac cycle in the septal artery and the left anterior descending artery. During isovolumic-systole, the percent area under the velocity curve was less in the septal artery ($9.2 \pm 1.9\%$) than that in the left anterior descending artery (16.0

TABLE 1
Hemodynamic Parameters

	Systolic aortic pressure (mm Hg)	Diastolic aortic pressure (mm Hg)	Heart rate (beats/min)	<i>n</i>
Control	125 ± 4	96 ± 4	143 ± 6	13
Nitroglycerin	$110 \pm 4^*$	$81 \pm 4^*$	$160 \pm 5^*$	8
Dipyridamole	$110 \pm 4^*$	$83 \pm 6^*$	$156 \pm 8^*$	8
Reactive hyperemia	130 ± 5	98 ± 5	140 ± 6	8
Pacing	117 ± 7	90 ± 4	140	6

* *P* < 0.05 compared to control.

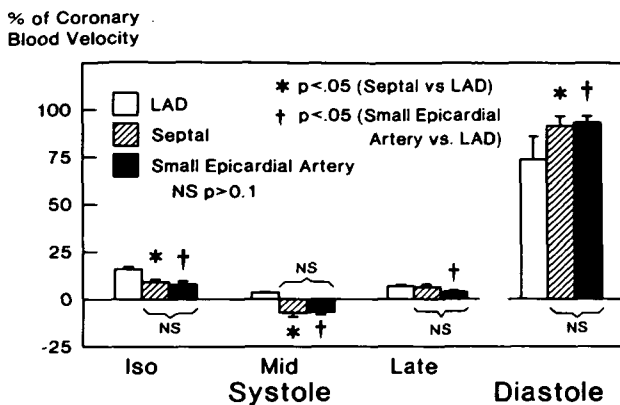


FIGURE 3. Percent of the area under the blood flow velocity curve during the particular segments of the cardiac cycle in the septal artery, left anterior descending artery (LAD), and small epicardial artery.

$\pm 1.8\%$). During mid-systole in the septal artery, the percent of the total area under the velocity curve was negative ($-7.2 \pm 2.6\%$) (significantly less than zero, $P < 0.05$), indicating retrograde blood flow velocity during this time. In contrast, in the left anterior descending artery, the mid-systolic area under the velocity curve was antegrade ($3.5 \pm 0.5\%$), i.e., significantly greater than zero ($P < 0.05$). Furthermore, a significantly higher percentage of the total area under the blood velocity curve occurred during diastole in the septal artery ($91.6 \pm 5.5\%$) than in the left anterior descending artery ($73.1 \pm 2.7\%$).

In small epicardial arteries (Fig. 3) the percent of the total area under the blood velocity curve occurring during isovolumic-systole ($9.8 \pm 2.2\%$) was similar to that in the septal artery, but significantly less than that in the left anterior descending. Correspondingly, during late-systole and diastole, the percent of the total area under the velocity curve occurring during these segments of the cardiac cycle were $3.8 \pm 1.2\%$ and $93.3 \pm 3.9\%$, respectively. These values were similar to those of the septal artery, but different from those in the left anterior descending. The percent of the total area under the velocity curve occurring during mid-systole was also retrograde ($-6.8 \pm 2.0\%$), and this value was significantly less than zero ($P < 0.05$).

Effect of Electrical Pacing

Following atrioventricular block, electrical pacing from the left ventricular free wall at 140 beats/min (Fig. 4) did not change the phasic nature of either left anterior descending artery or septal artery blood flow velocity. During isovolumic systole, the percent of the total area under the blood velocity curve in the septal artery ($7.4 \pm 1.3\%$) was less than that in the left anterior descending artery ($18.0 \pm 2.5\%$); in addition, these measurements from the paced hearts were not different from the control measurements. During mid-systole, the area under the velocity curve in the septal artery tended to be retrograde ($-0.7 \pm 0.5\%$) and in the left anterior descending artery was ante-

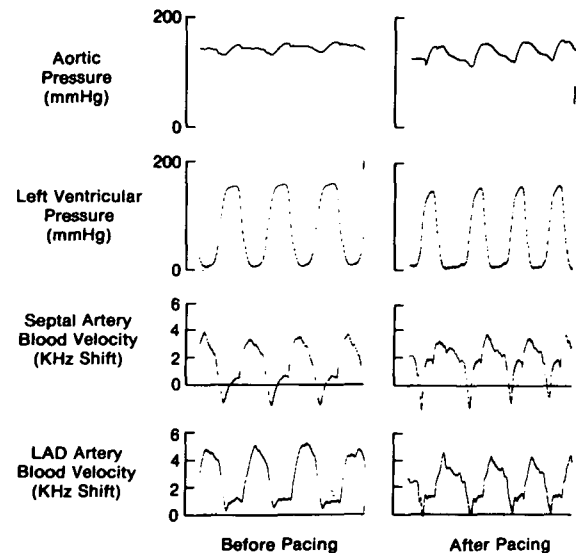


FIGURE 4. A record of hemodynamics, phasic septal artery blood flow velocity, and phasic left anterior descending artery blood flow velocity before and after electrical pacing.

grade $3.8 \pm 0.3\%$). No differences were observed during late systole between the septal artery ($4.8 \pm 0.2\%$) and the left anterior descending artery ($3.7 \pm 0.5\%$) or between these values and the control values. During diastole, the area under the blood velocity curve was greater in the septal artery ($88.4 \pm 2.5\%$) than in the left anterior descending ($74.5 \pm 1.8\%$). Moreover, these values were not different from their respective controls.

Effect of Vasodilation

Figure 5 shows a representative recording of the phasic coronary blood flow velocity in the septal and left anterior descending arteries during a control pe-

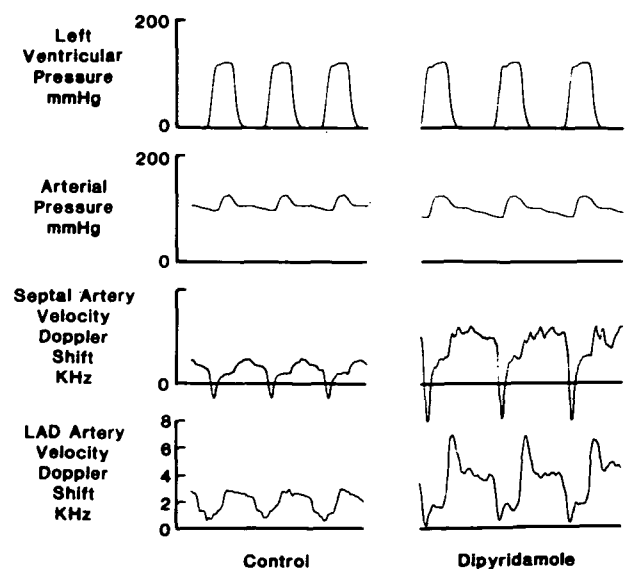


FIGURE 5. A record of hemodynamics, phasic septal artery blood flow velocity, and phasic left anterior descending artery blood flow velocity before and during dipyridamole administration.

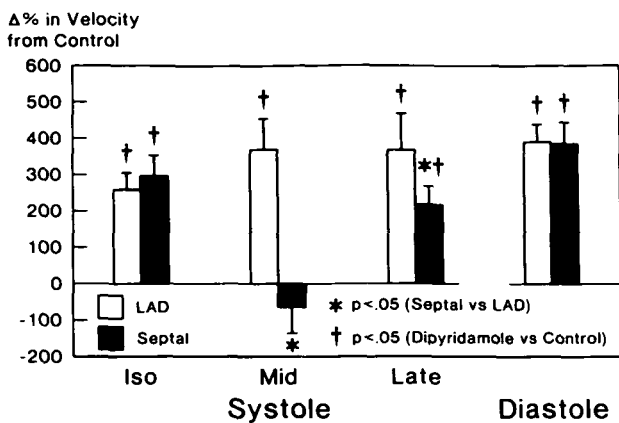


FIGURE 6. Percent change ($\Delta\%$) from the control are under the blood velocity curve during the different segments of the cardiac cycle following administration of dipyridamole.

riod and during the vasodilatory response with dipyridamole. Even though dipyridamole increased the area under the velocity curve in the left anterior descending coronary artery by 300% during mid-systole, in the septal artery retrograde systolic coronary blood flow velocity persisted. The percent increase in diastolic velocity curve was similar in both arteries. These average responses to dipyridamole are shown in Figure 6.

Figure 7 illustrates the effects of nitroglycerin on the percent increases in coronary blood flow velocity during the various segments of cardiac cycle from the respective control. Nitroglycerin increased the area under the coronary blood velocity curve in both the septal and left anterior descending arteries during isovolumic systole and diastole. During mid- and late-systole, however, nitroglycerin increased the area under the blood velocity curve ($P < 0.05$) only in the left anterior descending, $424 \pm 125\%$ and $260 \pm 75\%$, respectively. In contrast, the area under the velocity curve in the septal artery was not changed from control during mid- and late-systole, $-35 \pm 60\%$ and $59 \pm 24\%$, respectively.

During the peak reactive hyperemic period, (Fig. 8)

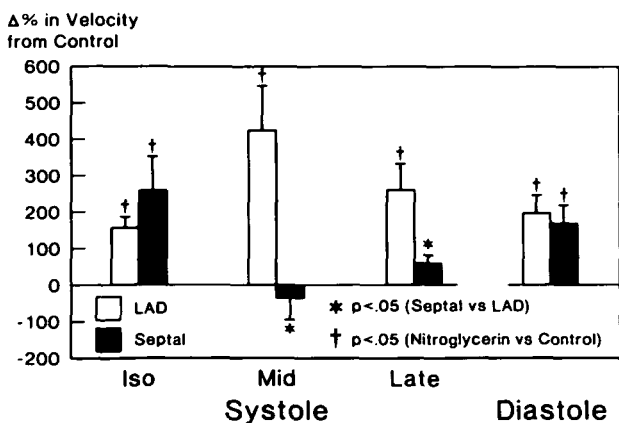


FIGURE 7. Percent change ($\Delta\%$) from the control area under the blood velocity curve during the different segments of the cardiac cycle following administration of nitroglycerin.

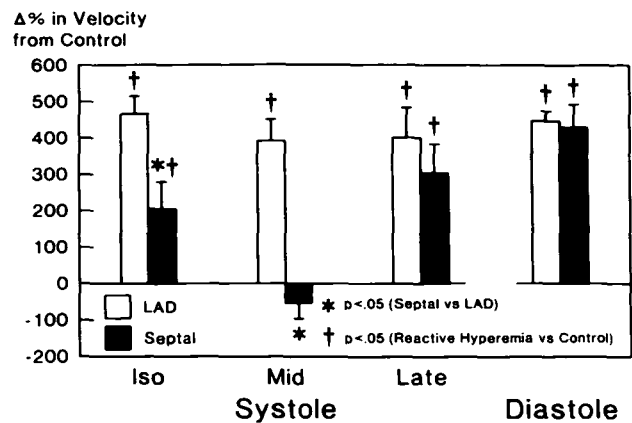


FIGURE 8. Percent change ($\Delta\%$) from the control area under the blood flow velocity curve during the different segments of the cardiac cycle during reactive hyperemia.

following a 20-second occlusion of the left main coronary artery, the area under the blood velocity curves during late-systole and diastole was increased significantly in the septal and left anterior descending arteries to a similar extent ($P < 0.05$). However, during reactive hyperemia, the percent change of the area under the blood velocity curve during isovolumic-systole in the left anterior descending artery ($466 \pm 51\%$) was greater ($P < 0.05$) than that in the septal artery ($205 \pm 77\%$). During reactive hyperemia, the mid-systolic area under the velocity curve in the septal artery was not significantly changed from control ($-53 \pm 45\%$) but was markedly increased in the left anterior descending artery ($391 \pm 61\%$).

Discussion

The major findings of this study are: (1) the phasic character of coronary blood flow velocity is markedly different in intramyocardial arteries from that of large epicardial arteries; (2) during mid-systole coronary blood flow velocity is retrograde in the intramyocardial arteries, whereas mid-systolic coronary blood flow velocity is antegrade in large epicardial arteries; and (3) during intense coronary vasodilation, the mid-systolic retrograde coronary blood flow velocity in the septal artery persists, even though mid-systolic coronary blood flow velocity in the left anterior descending increased by 300–400%.

The Doppler system we employed has certain advantages that enabled us to detect the differences in phasic coronary blood flow velocities between the septal and left anterior descending arteries. Our pulsed Doppler system is directional; thus, a negative coronary blood flow velocity is measurable. The zero of the system is stable; moreover, the electronic and occlusive zeros are identical. This stable zero is crucial for the measurements of blood flow velocity occurring during different segments of the cardiac cycle. In addition, because the piezoelectric crystal was placed in a small suction cup, only minimal exposure of the septal artery was needed to obtain adequate recordings of blood flow velocity.

A limitation of the Doppler system we employ is that blood flow velocity is measured rather than volume flow. Volume flow and blood flow velocity change in a parallel manner if major fluctuations in vessel diameter do not occur. Fortunately, in large coronary arteries, this is the case. Previous studies from our laboratory have shown that over a wide range, changes in mean coronary blood flow velocity measured with a Doppler probe correlate well ($r = 0.97$) with changes in flow measured by an electromagnetic flow meter and venous outflow collection (Marcus et al., 1981). Direct measurements of phasic coronary artery (proximal circumference) diameter varies by 4–5% during a single cardiac cycle (Vatner et al., 1980; Tomoike et al., 1981). This small change in diameter under normal circumstances should not significantly affect our results. Administration of dipyridamole decreased the circumflex diameter by 2.6% (Tomoike et al., 1981) and nitroglycerin increased the internal diameter by 0.8% (Vatner et al., 1980) at the time our blood flow velocity measurements were completed. Furthermore, the septal artery Doppler probe was placed on a segment of the vessel before it became encased by muscle; thus, contraction should not have greatly affected the vessel diameter at this site. In view of these considerations, it is likely that our measurements of coronary blood flow velocity reflect changes in coronary blood volume flow. Furthermore, we emphasize the error in estimating volume flow varies identically in the septal and left anterior descending artery (the cross-sectional areas should change similarly during the cardiac cycle); thus, our interpretations should be correct because the *relative* flows are correct.

A major assumption of the present study is that septal artery blood flow velocity reflects that in other intramural coronary arteries. Our results demonstrate that changing the temporal sequence of left ventricular contraction does not affect phasic blood flow velocity in the septal or left anterior descending arteries. Also, small epicardial blood vessels have phasic blood flow velocity patterns very similar to that of the septal artery. Since the phasic blood flow velocity pattern in the septal artery is similar to that in small epicardial arteries at a site immediately before they penetrate the myocardium, the septal artery is a suitable model for the analysis of phasic intramyocardial blood flow of large penetrating coronary arteries.

Phasic blood flow in the septal artery was studied previously (Eckstein et al., 1962; Carew and Covell, 1976). Eckstein and his colleagues measured septal artery blood flow with an orifice flow meter and found an early component of the systolic septal artery flow to be retrograde. These early studies lack precision because orifice flow meters produce stenosis, and phasic measurements are delayed because of the time lag that characterizes fluid-filled systems. Carew and Covell (1976) utilized acutely placed electromagnetic flow meters to measure septal artery blood flow. The encircling electromagnetic flow meter must have produced partial stenosis, because blood flow to the

septum, measured with microspheres, was abnormal (approximately 1/3 of that to the left ventricular free wall). Blood flow to the septum and left ventricular free wall are usually equivalent (Cobb et al., 1974; Marcus et al., 1975; and Bache et al., 1981). In addition, neither of these previous studies examined the effects of vasodilators on phasic septal artery blood flow.

Why is the phasic character of coronary blood flow velocity so different between the epicardial and intramural arteries? In our view, the most plausible explanation is that the epicardial arteries are functioning, in part, as capacitors, in addition to functioning as conduits. The capacitance of the epicardial arteries may theoretically distort the "true" flow (perfusion) measured at proximal sites on epicardial vessels because of the three capacitative effects predicted by Eckstein et al. (1962). Our data support these theoretical predictions concerning capacitative influences on the measured phasic coronary flow.

Where in the coronary vascular hierarchy is the "coronary capacitor" located? Douglas and Greenfield (1970) found the epicardial capacitance function to be large enough to account for normal systolic stroke flow, but not of sufficient magnitude to account for systolic stroke flow during vasodilation. Other investigators have found the epicardial vessels to be very compliant, two to three times that of similarly sized systemic arteries (Arts et al., 1979), suggesting potential capacitative effects. The epicardial coronary arteries are also reported to undergo 4–5% changes in diameter during a single cardiac cycle (Vatner et al., 1980; Tomoike et al., 1981), which also shows capacitative potential, i.e., capacitor "charging" during systole and "discharging" during diastole. Spaan et al. (1981) have suggested that the contracting myocardium functions as a pump capacitively coupled between the arterial and venous sides of the coronary circulation. Inherent in their model are two capacitors: one in the epicardial arteries and the other in the intramyocardial vasculature. They proposed that the intramyocardial capacitor has much greater capacitance effects than the epicardial capacitor and couples the intramyocardial pump to the coronary circulation. Although our results do not elucidate intramyocardial capacitance effects, they do support the hypothesis that the epicardial vasculature has capacitance function. Furthermore, our results support the concept of an intramyocardial pump generating systolic backflow. We speculate that with the three-dimensional changes of the left ventricle associated with contraction, i.e., a diminution of left ventricular size, the longitudinal stresses on the epicardial vessels may be lessened during systole. Correspondingly, this change in left ventricular chamber size during systole may increase the capacitative potential of epicardial coronary arteries.

Calculations based on our direct measurements support the concept that coronary capacitance is functionally located in both epicardial and intramyocardial vessels within the hierarchy of the coronary vasculature. We estimated the storage capacity of the left

anterior descending artery (epicardial portion) during control and vasodilated conditions to be 0.1 ml of blood (see Appendix). If stroke flow in the left anterior descending artery was 0.29 ml/beat under control conditions and 1.43 ml/beat during vasodilation, and the systolic stroke flow in the left anterior descending was 0.07 ml/beat under control conditions and 0.50 ml/beat during vasodilation (see Appendix), the total systolic stroke flow during vasodilation far exceeds the storage capacity of the epicardial vessels. Furthermore, during vasodilation, even mid-systolic stroke flow (0.14 ml/beat) exceeds the storage capacity of the epicardial segment (see Appendix); consequently, there could be additional capacitance function in small blood vessels downstream from large epicardial segments or subepicardial mid-systolic perfusion.

It has recently been suggested that the capacitance function of epicardial arteries may affect pressure-flow relationships in the coronary circulation (Eng et al., 1980). Some investigators have corrected for capacitance effects in the calculations of instantaneous coronary phenomena (Panerai et al., 1980), whereas others have discounted the importance of capacitance (Bellamy, 1980). Although the quantitative importance of coronary capacitance on pressure-flow relationships is controversial, it is reasonably certain that coronary capacitance contributes to the apparent critical closing pressure measurements when flow is measured in epicardial coronary arteries.

In summary, the present results indicate that it is difficult to predict phasic intramyocardial perfusion from measurements of phasic blood flow in epicardial coronary arteries. Discrepancies between phasic blood flow velocity patterns in large epicardial and intramyocardial arteries are best reconciled by postulating the existence of a significant coronary capacitor.

Appendix

Calculations and Assumptions

Assuming control left anterior descending artery blood flow is 40 ml/min and a heart rate of 140 beats/min (our experimental conditions), the stroke flow would be 0.29 ml/beat. The volume of the epicardial portion of the left anterior descending is assumed to be 1.0 ml [approximate value based on Douglas and Greenfield (1970)]. Since the epicardial coronary artery diameter increases by 5% during systole (Vatner et al., 1980; Tomoike et al., 1981), there is a 10% increase in vessel cross-sectional area. If the diameter change of epicardial vessels is uniform, the capacitance flow would be 0.1 ml during systole (10% increase in cross-sectional area from 1.0 ml). Under control conditions, approximately 25% of the stroke left anterior descending blood flow velocity occurred during systole, which computes to 0.07 ml/beat. During maximal vasodilation, if flow increased to 200 ml/min and the storage capacity of the epicardial vasculature is not changed, with the systolic proportion of the total stroke flow being 35% (results observed in present study), the systolic stroke flow is 0.50 ml, far in excess of the storage capacity. During mid-systole, accounting for both antegrade (3% of total stroke flow) and retrograde (-7% of total stroke flow) blood flow, the vessels would have to store 0.14 ml of blood during vasodilation,

which is slightly in excess of the storage capacity of the epicardial arteries.

We thank Charles Eastham for his assistance, Don Laughlin for construction of Doppler probes, Jane Beatty and Maureen Kent for typing the manuscript, and Drs. Allyn L. Mark and Francois M. Abboud for their critical review of this manuscript. We also wish to thank Dr. Ray Olsson for his suggestion that we could employ our Doppler system to study septal artery blood flow velocity.

Supported in part by Program Grant HL14388, National Institutes of Health Grant HL20827, a research grant from the Iowa Heart Association, and a research grant from the Veterans Administration. Dr. Marcus is a recipient of Research Career Development Award HL0032 from the National Heart, Lung, and Blood Institute.

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Received September 10, 1981; accepted for publication March 3, 1982.

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