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Effects of Intraaortic Balloon Pumping on Septal Arterial Blood Flow Velocity Waveform During Severe Left Main Coronary Artery Stenosis

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Objectives. **We sought to evaluate the effect of intraaortic balloon pumping on the phasic blood velocity waveform into myocardium with severe coronary artery stenosis.**

Background. **In the presence of severe coronary artery stenosis, it is not clear whether intraaortic balloon pumping augments intramyocardial inflow during diastole or changes systolic retrograde blood flow from the myocardium to the extramural coronary arteries.**

Methods. **Using anesthetized open chest dogs (n = 7), we introduced severe stenosis in the left main coronary artery to reduce the poststenotic pressure to approximately 60 mm Hg (>90% diameter stenosis). Septal arterial blood flow velocities were measured with a 20-MHz, 80-channel ultrasound pulsed Doppler velocimeter. Left anterior descending arterial flow, aortic pressure and poststenotic distal coronary pressure were measured simultaneously. The diastolic anterograde flow integral and systolic retrograde flow integral were compared in the presence and absence of intraaortic balloon pumping.**

Results. **Although intraaortic balloon pumping augmented diastolic aortic pressure, this pressure increase was not effectively transmitted through stenosis. Septal arterial diastolic flow velocity was not augmented, and left anterior descending arterial flow was unchanged during intraaortic balloon pumping.**

Conclusions. **In the presence of severe coronary artery stenosis, intraaortic balloon pumping failed to increase diastolic inflow to the myocardium and did not enhance systolic retrograde flow from the myocardium to the extramural coronary artery. Thus, the major effect of intraaortic balloon pumping on the ischemic heart with severe coronary artery stenosis may be achieved by reducing oxygen demand by systolic unloading.**

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Since the first clinical report in 1968 (1), intraaortic balloon pumping has been widely used in patients with coronary artery stenosis and poor cardiac pumping to relieve myocardial ischemia. Although the effect of intraaortic balloon pumping in reducing myocardial oxygen demand by systolic left ventricular unloading has consistently been recognized (2–6), the effects of intraaortic balloon pumping on the coronary artery blood flow remain uncertain. The coronary blood flow rate during intraaortic balloon pumping has been reported to increase (7–12), remain unchanged (13–16) or even decrease (6,17). One reason for the inconsistency of the coronary artery blood flow response during intraaortic balloon pumping may be the differences in the severity of the coronary artery stenoses in these studies. Moreover, the effects of intraaortic balloon pumping on the phasic pattern of the blood flow into the myocardium have not been evaluated in detail. It is now well known that the instantaneous blood flow patterns are different between proximal and distal coronary arteries, owing to the compliance of the coronary arteries (18). When the phasic pattern of the coronary inflow to the myocardium was evaluated at the peripheral portion of the artery or in the septal artery to avoid the influence of extramural coronary artery compliance, forward blood flow velocity waveform was almost exclusively limited to diastole (19,20) and was frequently accompanied by systolic retrograde flow. Systolic retrograde flow is augmented in the presence of coronary artery stenoses (21,22).

Thus, a careful examination of the phasic velocity waveform of a distal or an intramural coronary artery during coronary artery stenosis with well defined severity will be useful in understanding the effects of intraaortic balloon pumping on myocardial flow. The present study was designed to answer the following two questions when severe (>90% diameter narrowing) coronary artery stenosis is present: 1) Does intraaortic balloon pumping augment the diastolic coronary artery blood flow into the myocardium? 2) Does intraaortic balloon pumping enhance the systolic retrograde blood flow from deeper

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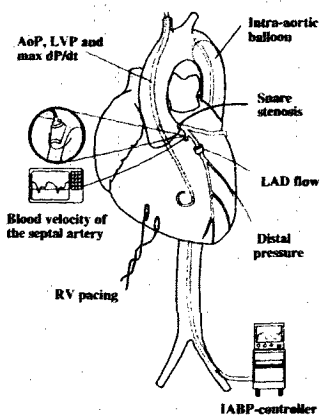

Figure 1. Animal preparations and measurements. Severe stenosis (>90% diameter stenosis) was introduced in the left main coronary artery with a snare occluder. AoP = aortic pressure; distal pressure = poststenotic distal coronary artery pressure; IABP = intraaortic balloon pumping; LAD = left anterior descending coronary artery; LVP = left ventricular pressure; max dP/dt = maximal increasing rate of left ventricular pressure; RV pacing = right ventricular pacing (with fixed heart rate, 100 beats/min).

myocardial layers toward the extramural stenotic coronary artery?

Methods

Animal preparations. Seven male or female mongrel dogs weighing 15 to 25 kg were premedicated with an intramuscular injection of ketamine (200 mg) and anesthetized with an intravenous injection of pentobarbital sodium (30 mg/kg body weight). Additional doses were given as needed throughout the experiment. After endotracheal intubation, the animals were ventilated by a respirator pump (3 to 5 liters/min oxygen) (VS600, Instrumental Development Corp.). Arterial pH and blood gases were measured frequently and kept in the physiologic range by adjusting the inspired oxygen concentration and minute ventilation. An arterial pressure catheter (models PC-470 and SPC-784A, Millar) was inserted into the right carotid artery to monitor and record the aortic pressure and left ventricular pressure. All dogs were anticoagulated with intravenous heparin (1,000 U hourly). Electrocardiograms were recorded from standard leads. After median sternotomy and left thoracotomy, the heart was exposed and supported in a pericardial cradle. The left main coronary artery was isolated, and a snare was placed around the proximal portion of the left main coronary artery to introduce stenosis (Fig. 1). The left anterior descending artery was also exposed to measure the left anterior descending artery flow with an electromagnetic flowmeter (EMF1200, FF-020, Nihon Kohden, Japan).

To measure phasic flow velocity in the first septal artery, the adipose tissue around the proximal part of the artery was carefully dissected away; dissection of only one-half of the circumference of the septal artery was sufficient for the blood velocity measurement. The heart rate was kept at 100 beats/min throughout the experiment by right ventricular pacing after formalin injection into the atrioventricular node. A pediatric intraaortic balloon pumping catheter (volume 12 ml; diameter 10 mm; length 17.8 cm) (Datascope Corp.) was inserted into the thoracic aorta through the left femoral artery. The end of the balloon was advanced into the descending aorta and was positioned just below the left subclavian artery, as confirmed by palpation. Intraaortic balloon pumping (model 90T, Datascope Corp.) was performed in the 2:1 mode so that the balloon was pulsated on every other heartbeat to compare the septal arterial flows between intraaortic balloon pumping turned on and off, with a minimal coronary vasomotor change between on and off.

Measurements of hemodynamic variables. We recorded aortic pressure, poststenotic distal coronary artery pressure, left ventricular pressure and maximal increasing rate of left ventricular pressure. To evaluate the transstenotic pressure gradient, we calculated the mean difference between the aortic pressure and the poststenotic distal coronary artery pressure using the time integral during systole and diastole, for example, for systole,

$$\overline{(AoP - DiP)}\,\text{systole} = \int_{\text{begin systole}}^{\text{end systole}} (AoP - DiP)\,dt/(t_{\text{end systole}} - t_{\text{begin systole}}),$$

where AoP and DiP are the aortic pressure and the poststenotic distal coronary pressure, respectively. *Systole* was defined as the period from the onset of isovolumetric contraction to the time of aortic valve closure. The onset of isovolumetric contraction was identified by the time corresponding to the peak R wave on the electrocardiogram, and the time of aortic valve closure was the dicrotic notch on the aortic pressure curve. Diastole was the remainder of the cardiac cycle. The resistance of the stenosis was calculated by $\overline{(AoP - DiP)}$/flow averaged over the entire cardiac cycle (23).

Measurements of septal arterial blood flow velocity. Septal arterial blood flow velocity was measured with a 20-MHz, 80-channel ultrasound pulsed Doppler velocimeter. The method has been described in detail previously (21,24). The system has 80 sampling gates and a depth resolution of ~0.2 mm. Doppler signals from the multicircuit are analyzed in real time by a zero-cross method, and the signal from an optional channel is analyzed by fast Fourier transform analysis. The unit data length of the Fourier analysis is 128 data points. The frequency resolution, temporal resolution and maximal detectable frequency of our system are 298 Hz, 2.56 ms and 25 kHz, respectively. The transducer of the Doppler system, which is discoid with a 1-mm diameter, was placed on the septal artery using a specially designed holder. The angle between the ultrasound beam and blood column was ~40°. The probe was placed carefully on the artery without compression of the vessel, and the holder was manipulated so that the maximal flow diameter was obtained by the 80-channel, zero-cross detector, indicating that the ultrasound beam passed

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onto or near the midpoint of the vessel. The envelope of the septal arterial blood flow velocities in the fast Fourier transform display, which is the velocity area, was integrated during systole, during diastole and throughout the cardiac cycle. The septal arterial blood flow velocity area was divided into three components—systolic retrograde velocity integral, systolic anterograde velocity integral and diastolic anterograde velocity integral. A "slosh ratio" was defined as the ratio of systolic retrograde velocity integral to diastolic anterograde velocity integral (Fig. 2).

All measurements were recorded simultaneously on the data recorder (model R-81, TEAC) and a multichannel pen recorder (model RIJ 5608, Nihon Kohden, Japan) at a paper speed of 25 mm/s. The data were analyzed during steady state after the onset of 2:1 mode pumping.

The experimental procedure and protocol were approved by the Animal Research Committee of the Kawasaki Medical School and were in compliance with the "Position of the American Heart Association on Research Animal Use," adopted by the Association in November 1984.

Study protocol. First, intraaortic 2:1 mode balloon pumping was started under control conditions without stenosis. The data were analyzed in steady state a few minutes after the onset of pumping. The flow velocity and the hemodynamic variables were compared with and without intraaortic balloon pumping. Then, 2:1 mode pumping was stopped. After the hemodynamic variables returned to the initial levels before pumping, severe coronary artery stenosis was introduced by the left main coronary artery ligature to decrease the poststenotic distal coronary artery pressure to ~60 mm Hg. The measurements of variables were repeated.

Statistical analysis. Results are expressed as mean value ± SE. Comparison was made by the nonparametric Wilcoxon signed-rank test (Abacus Concepts, Inc., StatView-4.02J) with and without intraaortic balloon pumping and also with and without stenosis when intraaortic balloon pumping was turned off. The effects of intraaortic balloon pumping on coronary artery blood flow, blood velocity and other hemodynamic variables were compared by two-way repeated-measures analysis of variance (ANOVA) (Abacus Concepts, Inc., StatView-4.02J) with stenosis and without stenosis. The criterion for statistical significance was $p < 0.05$.

Results

Systemic and coronary hemodynamic variables (Table 1). The flow resistance $(\overline{AoP} - \overline{DiP})/flow$ during control conditions without stenosis was negligible (0.0 ± 0.03 mm Hg/ml per min). Mean diastolic aortic pressure and mean diastolic poststenotic distal coronary artery pressure were augmented by 13% and 12%, respectively, during intraaortic balloon pumping (both $p < 0.05$). Mean systolic aortic pressure and mean systolic poststenotic distal coronary artery pressure were decreased during intraaortic balloon pumping by 2% and 3%, respectively (both $p < 0.05$).

Introduction of stenosis reduced mean systolic and mean diastolic poststenotic distal coronary artery pressures ($p < 0.05$ and $p < 0.005$, respectively, ANOVA). The flow resistance with stenosis was 2.0 ± 0.50 mm Hg/ml per min, much higher

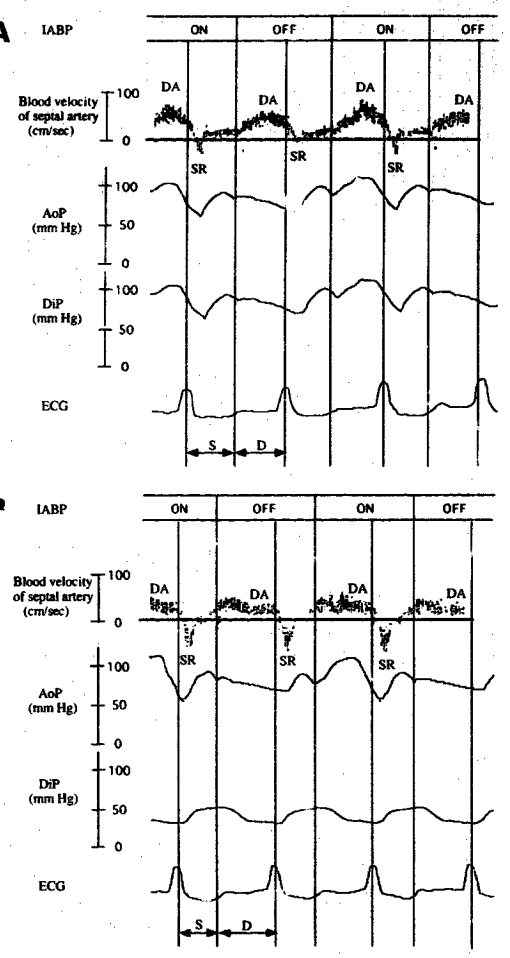

Figure 2. Representative tracings of septal arterial blood flow velocities, aortic pressures (AoP), distal coronary artery pressures (DiP) and electrocardiogram (ECG) in dogs with (+) and without (−) coronary artery stenosis. Without stenosis (**A**), the diastolic septal blood flow velocity increased during intraaortic balloon pumping (IABP) with simultaneous increases in aortic pressure and distal coronary artery pressure. Systolic retrograde flow integral (SR) was increased during intraaortic balloon pumping. With stenosis (**B**), diastolic septal arterial blood velocity failed to increase during intraaortic balloon pumping. Aortic pressure increased, whereas diastolic coronary artery pressure was almost unchanged. The magnitude of the systolic retrograde flow integral was unchanged by intraaortic balloon pumping. D = diastole; DA = diastolic anterograde flow integral; OFF = intraaortic balloon pumping turned off; ON = intraaortic balloon pumping turned on; S = systole.

Table 1. Systemic and Coronary Hemodynamic Variables During Intraaortic Balloon Pumping Turned On and Off

	Stenosis (−) (n = 5)		Stenosis (+) (n = 7)		p Value (ANOVA)	
	IABP Off (mean ± SE)	IABP On (mean ± SE)	IABP Off (mean ± SE)	IABP On (mean ± SE)	Stenosis (+) vs. Stenosis (−)	IABP On vs. IABP Off
Diastolic AoP (mm Hg)	106.6 ± 16.1	120.1 ± 15.7*	99.5 ± 9.5	115.4 ± 10*	NS	<0.0001
Diastolic DiP (mm Hg)	112.0 ± 18.7	125.5 ± 18.3*	49.6 ± 4.5†	52.1 ± 4.1*	<0.005	<0.0001
Systolic AoP (mm Hg)	111.6 ± 16.2	109.0 ± 15.8*	104.6 ± 9.6	101.5 ± 9.7	NS	<0.01
Systolic DiP (mm Hg)	116.0 ± 18.7	113.4 ± 17.9*	61.8 ± 6.9†	61.8 ± 6.3	<0.05	NS
Max dP/dt (mm Hg/s)	1782 ± 310	1740 ± 308	1322 ± 107	1262 ± 119	NS	NS

*p < 0.05, intraaortic balloon pumping turned off (IABP Off) versus intraaortic balloon pumping turned on (IABP On). †p < 0.05, presence of stenosis [Stenosis (+)] with intraaortic balloon pumping off versus absence of stenosis [Stenosis (−)] with intraaortic balloon pumping off. ANOVA = analysis of variance; AoP = aortic pressure; DiP = poststenotic distal coronary artery pressure; Max dP/dt = maximal increasing rate of left ventricular pressure.

than that without stenosis ($p < 0.05$), which corresponded to >90% diameter stenosis (23). Mean diastolic aortic pressure was augmented by 16% during intraaortic balloon pumping ($p < 0.05$), whereas mean diastolic poststenotic distal coronary artery pressure was increased by only 5%, although this was statistically significant ($p < 0.05$). Mean systolic aortic pressure and mean systolic poststenotic distal coronary artery pressure were almost unchanged by intraaortic balloon pumping (not significant).

Maximal increasing rate of left ventricular pressure tended to decrease during intraaortic balloon pumping in the presence and absence of stenosis, although the changes were not statistically significant.

Effects of intraaortic balloon pumping on coronary artery blood flow and septal arterial blood flow velocity (Table 2). The increase in the ratio of blood flow and velocity areas by intraaortic balloon pumping was different statistically with and without stenosis ($p < 0.05$, ANOVA). When blood flow and velocity areas with and without stenosis were compared when intraaortic balloon pumping was turned off, the left anterior descending arterial flow decreased, whereas the systolic retrograde flow integral and slosh ratio increased significantly ($p < 0.05$).

Figure 2 shows representative tracings of septal arterial blood flow velocities, aortic pressure and poststenotic distal coronary artery pressure with and without stenosis. Without stenosis (Fig. 3A), the forward diastolic septal blood flow velocity increased during intraaortic balloon pumping. However, with stenosis (Fig. 3B), the forward diastolic septal arterial blood flow velocity failed to increase during intraaortic balloon pumping.

The coronary artery blood flow (left anterior descending arterial flow) without stenosis was augmented by 12% during intraaortic balloon pumping ($p < 0.05$) (Fig. 3A, Table 2). The diastolic forward flow integral was increased by 12% and systolic retrograde flow integral by 44% during intraaortic balloon pumping (both $p < 0.05$). Accordingly, the slosh ratio was augmented by 33% during intraaortic balloon pumping ($p < 0.05$).

In the presence of left main coronary artery stenosis, intraaortic balloon pumping did not significantly enhance left anterior descending arterial flow, diastolic forward flow integral, systolic retrograde flow integral or the slosh ratio (Fig. 3B, Table 2). Thus, there was no apparent improvement in coronary hemodynamic variables by intraaortic balloon pumping in the presence of severe coronary artery stenosis.

Discussion

To evaluate the effect of intraaortic balloon pumping on the phasic arterial blood flow pattern in the intramyocardial artery in the presence and absence of severe coronary artery stenosis,

Table 2. Flow and Velocities During Intraaortic Balloon Pumping Turned On and Off

	Stenosis (−) (n = 5)			Stenosis (+) (n = 7)	
	IABP Off (mean ± SE)	IABP On (mean ± SE)		IABP Off (mean ± SE)	IABP On (mean ± SE)
LAD flow (ml/min)	16.9 ± 2.2	18.9 ± 2.6*	‡	4.0 ± 0.9†	4.0 ± 0.9
DA (cm)	9.7 ± 1.1	10.9 ± 1.3*	‡	11.3 ± 2.8	11.8 ± 2.7
SR (cm)	0.6 ± 0.1	0.8 ± 0.2*	‡	3.1 ± 0.7†	2.6 ± 0.6
Slosh ratio (SR/DA)	0.06 ± 0.02	0.08 ± 0.03*	‡	0.34 ± 0.08†	0.24 ± 0.04

*p < 0.05, intraaortic balloon pumping turned on (IABP On) versus IABP turned off (IABP Off). †p < 0.05, presence of stenosis [Stenosis (+)] with intraaortic balloon pumping off versus absence of stenosis [Stenosis (−)] with intraaortic balloon pumping off. ‡Analysis of variance ($p < 0.05$, stenosis present vs. stenosis absent) for percent differences of flow and velocity areas between intraaortic balloon pumping on and intraaortic balloon pumping off. DA = diastolic anterograde flow integral; LAD flow = left anterior descending coronary artery flow; SR = systolic retrograde flow integral.

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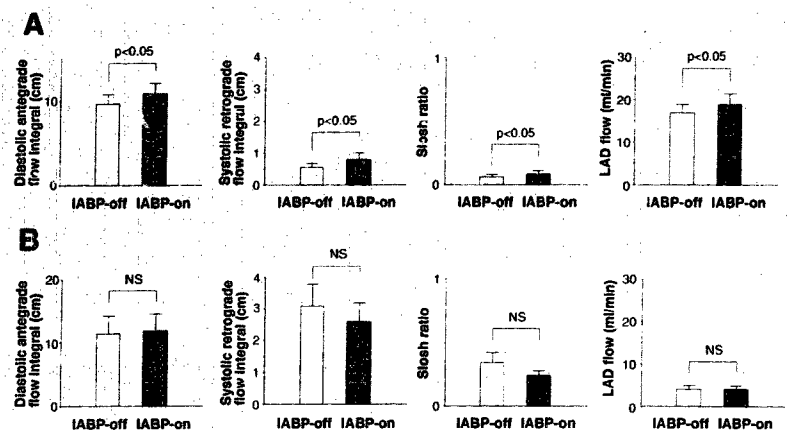

Figure 3. Effect of intraaortic balloon pumping (IABP) on septal arterial blood flow velocity waveform and coronary artery blood flow. **A,** Stenosis absent. Diastolic anterograde flow and systolic retrograde flow integrals were increased. Thus, the "slosh ratio" was augmented during intraaortic balloon pumping. The net left anterior descending coronary artery blood flow (LAD flow) was augmented during intraaortic balloon pumping. **B,** Stenosis present. Neither diastolic anterograde flow integral nor systolic retrograde flow integral were augmented by intraaortic balloon pumping. Left anterior descending coronary artery blood flow did not increase during intraaortic balloon pumping. Other abbreviations as in Figure 2.

we measured the blood flow velocity of the septal artery with and without intraaortic balloon pumping. In the absence of coronary artery stenosis, intraaortic balloon pumping enhanced the forward diastolic coronary artery blood flow velocity component by augmenting diastolic arterial pressure. At the same time, intraaortic balloon pumping increased systolic retrograde blood flow by reducing coronary artery systolic pressure. However, the time-averaged coronary artery blood flow rate was increased by the greater increment in diastolic flow.

In contrast, when severe coronary artery stenosis was present, intraaortic balloon pumping did not change the coronary poststenotic pressure pattern. Therefore, it failed to increase forward diastolic blood flow velocity or change systolic retrograde blood flow. Thus, systolic unloading by intraaortic balloon pumping may be the major beneficial effect on the heart in the patients with severe coronary artery stenosis with little effect on myocardial inflow.

Methodologic considerations. We used a 20-MHz, 80-channel pulsed Doppler ultrasound velocimeter by placing the transducer on the outside of the septal artery, which avoids disturbing the flow field, as with intravascular catheter or guide wire type Doppler velocimeters (9,10,25). The other advantage of our method is the ability to determine the maximal flow diameter with the 80-channel, zero-crossing detector, which enabled us to measure the velocity near the central axial region. It was also possible to avoid the blood flow velocity measurements in the disturbed flow area near the entrance of the septal artery by monitoring the broadening of the velocity spectrum by real-time fast Fourier transform analysis (24).

Intraaortic balloon pumping affects the coronary blood flow directly by changing coronary artery pressure and indirectly by altering vasomotor tone, owing to the change in myocardial oxygen demand. To evaluate the effects of intraaortic balloon pumping on the coronary blood flow with a minimal change in myocardial oxygen requirement, we chose the alternative beat 2:1 mode of intraaortic balloon pumping, although some changes in vascular tone might occur progressively even by this mode of pulsation. The observations of the present study were in normotensive animals without cardiogenic shock. The effect of intraaortic balloon pumping in these circumstances could be different from that in hypotensive animals.

Effect of intraaortic balloon pumping on myocardial inflow without coronary artery stenosis. Bregman et al. (11) reported an increase in coronary graft flow during intraaortic balloon pumping, as measured by an electromagnetic flowmeter. Yamagishi et al. (12) observed an increased peak flow velocity in the epicardial conduit coronary artery during intraaortic balloon pumping, as measured by transesophageal Doppler echocardiography. Kern et al. (9) also found an augmented proximal coronary blood flow velocity during intraaortic balloon pumping, as measured by a Doppler-tipped catheter. Our data are consistent with these reports of augmented coronary flow during intraaortic balloon pumping in the absence of coronary artery stenosis. On the other hand, using an electromagnetic flowmeter, Sugg et al. (17) reported decreased coronary flow in dogs 2 h after the introduction of intraaortic balloon pumping.

The effects of intraaortic balloon pumping on the coronary blood flow depends on both the direct mechanical effect on the coronary vessels and the indirect effect through myocardial oxygen consumption (5,26). The controversy of whether the coronary flow increases or decreases during intraaortic balloon

pumping may be explained by the relative weights of these two effects. When the coronary vasomotor tone was kept almost constant with intraaortic balloon pumping applied on alternative beats, we observed that diastolic pressure augmentation increased the diastolic myocardial inflow and that systolic unloading caused an increase in systolic retrograde flow (Fig. 2). The net myocardial inflow increased because the diastolic flow augmentation was greater than the increase in the systolic retrograde flow (Fig. 3A). With continuous intraaortic balloon pumping, systolic unloading reduces myocardial oxygen demand and mechanical augmentation of coronary blood flow by intraaortic balloon pumping may be overcome by metabolic vasoconstriction.

Effect of intraaortic balloon pumping on myocardial inflow with severe coronary artery stenosis. Williams et al. (6) measured the great cardiac vein flow in patients with severe left anterior descending coronary artery stenosis during intraaortic balloon pumping and observed a decreased time-averaged coronary artery blood flow. Kerber et al. (14) and Jett et al. (13) reported that intraaortic balloon pumping did not enhance the coronary blood flow in both ischemic and nonischemic regions using a radioactive microsphere technique. In contrast, Gill et al. (27) observed that the coronary flow was increased by intraaortic balloon pumping in the ischemic region but not in the nonischemic region. Saini et al. (7) and Swank et al. (8) found an augmentation of coronary flow in both nonischemic and ischemic regions. The divergent results may be due to differences in stenotic severity or vasomotor tone, or both.

In mild stenosis, where coronary blood flow is independent of the perfusion pressure (autoregulatory pressure range), an increase in coronary artery pressure by intraaortic balloon pumping may not increase the coronary blood flow significantly if myocardial oxygen demand is unchanged. However, when the severity of stenosis further increased and the flow reserve was lost, the coronary blood flow decreased and became pressure dependent. In this condition, if the poststenotic coronary artery pressure can be increased by intraaortic balloon pumping, an increase in the diastolic coronary blood flow would be expected. In severe coronary artery stenosis, when the stenotic pressure loss (friction and separation losses) becomes large and the poststenotic pressure is not elevated by balloon inflation, flow augmentation would not be expected. In the present study, the coronary artery stenosis was severe enough to decrease the poststenotic pressure to ~60 mm Hg, which is below the coronary autoregulatory pressure range (18,28,29). Thus, intraaortic balloon pumping would increase diastolic blood velocity only if the increase in aortic pressure was effectively transmitted to the poststenotic region. However, this was not the case in the present study (see Fig. 2). Our observation is consistent with the observations of Kern et al. (10) and Williams et al. (6) in that there was not a significant flow improvement beyond critical stenosis with intraaortic balloon pumping in the human coronary circulation.

The effect of intraaortic balloon pumping on the myocardial blood flow may differ between the deeper and superficial myocardial layers. Swank et al. (8) reported that intraaortic balloon pumping increased the epicardial blood flow, although it failed to increase the endocardial blood flow. Shaw et al. (15), Kerber et al. (14), Gewirtz et al. (5) and Smalling et al. (16) also showed by the radioactive microsphere technique that intraaortic balloon pumping failed to increase the endocardial blood flow.

Recently, it was pointed out that with increasing severity of coronary artery stenosis, the diastolic anterograde flow decreases whereas systolic retrograde flow from the deeper myocardial layers to the extramural coronary artery increases (21), resulting in a decrease in the endocardial blood flow (22). In the present study, although intraaortic balloon pumping failed to increase the diastolic inflow, the pumping did not worsen the systolic retrograde flow, probably because of the lack of a significant decrease in the poststenotic coronary artery systolic pressure. Thus, it is unlikely that intraaortic balloon pumping hampers the perfusion of the subendocardium by increasing the systolic retrograde flow or decreasing the diastolic anterograde flow in severe coronary artery stenosis. However, when coronary artery stenosis was absent, intraaortic balloon pumping enhanced the systolic retrograde blood flow from the myocardium to the epicardial coronary arteries (Fig. 2), when its increased diastolic flow enhancement was not sufficient. This may cause a deleterious effect of intraaortic balloon pumping on the perfusion of the subendocardium in some diseases without severe coronary artery stenosis, such as shock, aortic valvular disease and severe anemia. However, these problems are beyond the scope of the present study.

Conclusions. In the presence of severe coronary artery stenosis, intraaortic balloon pumping failed to increase the diastolic inflow to the myocardium and time-averaged blood flow rate because the augmented diastolic aortic pressure was inadequately transmitted through the stenosis. Intraaortic balloon pumping did not worsen the systolic retrograde flow from the deeper myocardial layers to the extramural coronary artery, which had a deleterious effect on the myocardial flow. Thus, the major beneficial effect of intraaortic balloon pumping on the ischemic heart with severe coronary stenosis may be achieved through systolic unloading, thus reducing myocardial demand.

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