Systolic Coronary Flow Reversal and Abnormal Diastolic Flow Patterns in Patients With Aortic Stenosis: Assessment With an Intracoronary Doppler Catheter

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Decreased left ventricular coronary flow reserve has been reported in patients with normal coronary arteries and left ventricular hypertrophy in association with aortic stenosis. However, phasic coronary flow characteristics have not been analyzed in detail in similar patients. The purpose of this study is to assess phasic coronary flow characteristics and their relation to hemodynamic parameters in patients with aortic stenosis. Coronary flow velocities were recorded in the left anterior descending artery with a 20 MHz Doppler catheter in nine patients with aortic stenosis and nine control subjects with normal coronary arteries. Patient aortic valve area ranged from 0.34 to 0.51 cm². Flow reversal was observed in systole in all patients with aortic stenosis, and time velocity integrals of systolic flow were significantly smaller in patients with aortic stenosis than in controls (-0.3 ± 2.3 vs 4.0 ± 1.1 cm, $p < 0.01$). The time to peak diastolic velocity corrected by $\sqrt{R\text{-}R}$ interval was prolonged and the velocity half-time from peak diastolic velocity corrected by $\sqrt{R\text{-}R}$ interval was shorter in patients with aortic stenosis than in controls (5.3 ± 1.1 vs 4.0 ± 0.5, $p < 0.01$, 8.0 ± 2.6 vs 13.0 ± 3.3, $p < 0.01$, respectively). Peak velocity and time velocity integral of flow reversal showed significant correlations with mean pressure gradient across the aortic valve ($y = -1.3x + 37.3$, $r = 0.72$, $p = 0.03$, $y = 11.3x + 41.2$, $r = 0.81$, $p < 0.01$, respectively). These abnormal coronary flow patterns were no longer present after aortic valve replacement in six of nine patients with aortic stenosis who were studied again within 1 month after surgery. In summary, flow reversal in systole and slow acceleration and rapid deceleration of coronary flow velocity in diastole are characteristic in patients with aortic stenosis. These abnormal flow velocity patterns may be related to the pressure difference across the aortic valve. (J Am Soc Echocardiogr 1993;516-24.)

In patients with aortic stenosis, retrograde coronary artery flow in systole has been reported angiographically[1] and has been confirmed by epicardial Doppler studies during open heart surgery.[2] Its clinical significance is not completely clear, although it is thought to be limiting with respect to myocardial blood supply. This may be particularly disadvantageous in patients with aortic stenosis because angina pectoris in these patients, in the absence of coronary artery disease, has been explained by the imbalance between the supply and demand of oxygen to the extremely hypertrophied myocardium.[3,4]

With the development of intracoronary Doppler catheters, coronary flow velocities can be measured with ease and safety during cardiac catheterization.[5-7] This technique has been successfully used to assess coronary flow reserve and coronary circulation in human subjects.[8-14] A decrease in left ventricular coronary flow reserve has been reported in patients with normal coronary arteries and left ventricular hypertrophy caused by aortic stenosis when studied at the time of open heart surgery, and this impairment in coronary reserve has been proposed to be an important mechanism of angina pectoris in patients with aortic stenosis.[8] However, comparative analyses of phasic coronary flow characteristics by use of the

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Doppler catheter technique in patients with aortic stenosis and normal subjects has not been reported in detail.

The purpose of this study is to assess the characteristics of coronary flow dynamics and their relation to hemodynamic parameters in patients with aortic stenosis and normal coronary arteries during cardiac catheterization.

METHODS

Patient Selection

The study population consisted of nine consecutive patients with aortic valve stenosis (three men and six women). Patients with other concomitant valvular or congenital heart disease were excluded from study. No patient had more than mild aortic regurgitation as assessed by aortography. All patients were in sinus rhythm and had angiographically normal coronary arteries. Patient age ranged from 49 to 69 (61 ± 7, mean ± SD) years. Seven out of the nine patients underwent aortic valve replacement, and six (two men and four women; 64 ± 6 years) of these seven were studied again within 1 month of operation (23 ± 5 days). Nine subjects (seven men and two women) undergoing coronary angiography for the evaluation of chest pain syndrome served as controls. All were in sinus rhythm and had normal coronary arteries by angiography. Control subject ages ranged from 34 to 76 (54 ± 13) years.

Cardiac Catheterization and Hemodynamic Measurements

All medications, including β-blockers and calcium-channel blockers, were terminated at least 24 hours before cardiac catheterization. After sedation with 5 mg of diazepam administered orally, patients were taken to the catheterization laboratory. Any drugs that affect coronary hemodynamics, including nitroglycerin, were not used during catheterization.

Catheterization of the right and left side of the heart was performed from the femoral approach. The left ventricle was approached in a retrograde manner, and aortic and left ventricular pressures were obtained simultaneously. Pressure data were recorded with a fluid-filled catheter-transducer system. Cardiac outputs were measured by the thermodilution method. Aortic valve areas were calculated from the Gorlin formula. Selective coronary arteriography was carried out by the Judkins technique.

During cardiac catheterization, two-dimensional and M-mode echocardiograms were obtained with a Toshiba SSH-140 system (Toshiba Corp., Tokyo, Japan). These were recorded on videotape, and M-mode echocardiograms were also recorded by a strip-chart recorder at a paper speed of 100 mm/sec, together with electrocardiograms, phonocardiograms, and the left ventricular pressure tracing. Left ventricular mass was calculated from two-dimensional echocardiograms by use of the algorithm previously described.[15,16] Wall thickness and left ventricular dimensions were measured from the M-mode echocardiograms.

Recording and Measurement of Coronary Flow Velocities

Phasic coronary blood flow velocity was recorded in the proximal portion of the left anterior descending artery by use of a 3F coronary Doppler catheter (Model DC-201, Millar Instruments, Houston, Texas) and a velocimeter (Model MDV-20, Millar Instruments).[6] The frequency of the pulsed Doppler flowmeter attached to the tip of the catheter was 20 MHz and the pulse repetition frequency was 62.5 kHz. As shown in Figure 1, the Doppler catheter was advanced into the left anterior descending artery through an 8F coronary guiding catheter (Bard Inc., USCI div., Billevica, Mass.) over a 0.014 inch flexible steerable guide wire (Bard, USCI) and placed in the proximal portion of the left anterior descending artery just distal to the first septal branch. An optimal Doppler signal was obtained by moving the catheter slightly within the arterial lumen, placing torque on the flexible guide wire extending through the tip of the Doppler catheter, and adjusting the range gate control. Frequency analysis of the Doppler signals was carried out in real time by fast-Fourier transform on the Toshiba SSH-140 according to the previous report.[7] Doppler signals were recorded on videotape and by a strip-chart recorder at a paper speed of 100 mm/sec along with electrocardiograms, with or without phonocardiograms or aortic pressure tracings.

From the phasic coronary flow velocity recordings several measurements were made to analyze the characteristics of the coronary flow (Figure 2). These included systolic and diastolic peak velocities, systolic and diastolic time velocity integrals, the time from the beginning of diastole to peak diastolic velocity, and velocity half-time (a measure of the deceleration of velocity in diastole).

Statistical Analysis

Statistical analysis was performed with unpaired *t* testing as appropriate. Linear regression analysis

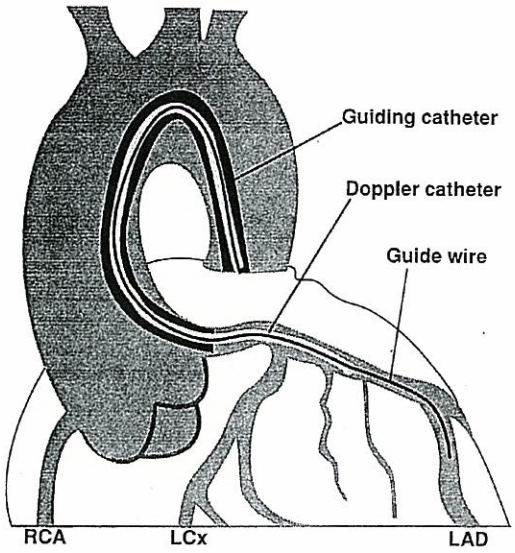

Figure 1 Schema of the placement of the Doppler catheter in the left anterior descending coronary artery. *LAD,* Left anterior descending coronary artery; *LCx,* left circumflex artery; *RCA,* right coronary artery.

was used for assessing the relationship between hemodynamic data and variables from the coronary flow velocity recordings. A probability value of <0.05 was considered significant.

RESULTS

Echocardiographic Data and Pressure Data

All patients with aortic stenosis had a significant mean pressure difference across the aortic valve (69 ± 18 mm Hg) and reduced calculated aortic valve area (0.45 ± 0.05 cm²). Left ventricular end-diastolic pressure was elevated (17 ± 7 mm Hg), although pulmonary capillary wedge pressure and left ventricular ejection fraction were within the normal range (Table 1). Left ventricular mass index and wall thickness were significantly greater in the aortic stenosis group compared with that in controls ($p < 0.01$) (Table 2), but in the six patients who were studied again within 1 month after operation there was no significant change (Table 3).

Coronary Flow Measurements

Coronary flow velocity parameters are shown in Table 2. Systolic flow reversal was seen in all patients with aortic stenosis (Figure 3), and peak systolic flow

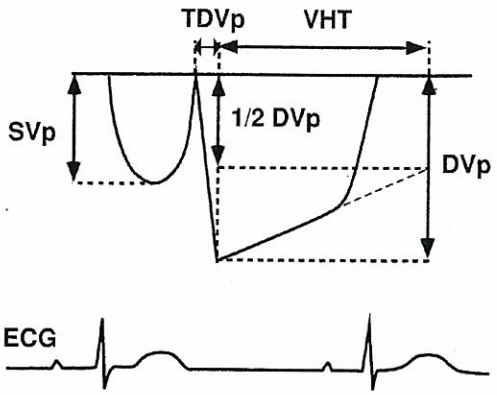

Figure 2 Measurements of variables of coronary flow velocity recording. *DVp,* Diastolic peak velocity; *TDVp,* time from the beginning of diastole to diastolic peak velocity; *SVp,* systolic peak velocity; *VHT,* velocity half time.

velocity and systolic time velocity integral in patients with aortic stenosis were significantly smaller than those in controls ($p < 0.001, p < 0.01$, respectively). Peak diastolic flow velocity was slightly higher, but not significantly so, in patients with aortic stenosis compared with that in controls. Diastolic time velocity integral was not significantly different between groups. The time from the beginning of diastole to diastolic peak flow velocity corrected by $\sqrt{\text{R-R}}$ interval ($\sqrt{\text{RR}}$) was prolonged and diastolic flow velocity half-time corrected by $\sqrt{\text{RR}}$ was shorter in patients with aortic stenosis compared with controls ($p < 0.01$, $p < 0.01$, respectively). These flow velocity patterns did not change as flow velocity mapping progressed from the left main coronary artery distally into the left anterior descending artery.

Systolic coronary flow revesal peak flow velocities and time velocity integrals in patients with aortic stenosis showed significant correlations with mean pressure differences across the aortic valve ($y = -1.3x + 37.3, r = 0.72, p = 0.03, y = 11.3x + 41.2, r = 0.81, p < 0.01$, respectively) (Figures 4 and 5). A lesser correlation was found between peak velocities of flow reversal and aortic valve areas ($y = -0.35x + 0.53, r = 0.67, p = 0.05$). No significant correlation was found between peak velocities or time velocity integrals of flow reversal and peak systolic left ventricular pressures or left ventricular mass indices.

Pattern After Aortic Valve Replacement

After aortic valve replacement the coronary flow velocity pattern changed with the development of a

Table 1 Echocardiographic and catheterization data of the subjects

	Patients with AS	Controls	
Age (years)	61 ± 7	54 ± 13	NS
Heart rate (beats/min)	73 ± 9	60 ± 7	$p < 0.01$
Echocardiographic data			
Left ventricular wall thickness (mm)	14 ± 2	8 ± 1	$p < 0.01$
Left ventricular end-diastolic dimension (mm)	43 ± 5	45 ± 2	NS
Left ventricular mass index (gm/m²)	211 ± 73	90 ± 11	$p < 0.01$
Cardiac catheterization data			
Cardiac index (liter/min/m²)	2.4 ± 0.4	2.5 ± 0.4	NS
Mean PCWP (mm Hg)	10 ± 6	8 ± 4	NS
Mean aortic pressure (mm Hg)	83 ± 15	103 ± 6	$p < 0.01$
Peak systolic LVP (mm Hg)	197 ± 43	130 ± 14	$p < 0.01$
LVEDP (mm Hg)	17 ± 7	14 ± 6	$p < 0.01$
LVEDV (ml)	112 ± 30	137 ± 23	NS
EF (%)	56 ± 7	55 ± 6	NS
Mean pressure gradient (mm Hg)	69 ± 18	0	
Aortic valve area (cm²)	0.45 ± 0.05	—	

AS, Aortic stenosis; *EF*, ejection fraction; *LVEDP*, left ventricular end-diastolic pressure *LVEDV*, left ventricular end-diastolic volume; *LVP*, left ventricular pressure; *PCWP*, pulmonary capillary wedge pressure.

flow velocity pattern similar to that seen in the control group (Figure 6 and Table 3).

DISCUSSION

In this study, an abnormal pattern of coronary artery flow was observed in patients with aortic stenosis characterized by systolic flow reversal in all patients, with associated diminished peak forward flow velocities and forward flow velocity integrals. Diastolic flow was characterized by a prolongation of the time interval from the beginning of diastole to peak diastolic flow velocity and a shortening of diastolic velocity half-time. The magnitude of systolic flow reversal peak velocity and velocity time integral correlated significantly with the magnitude of aortic valve pressure difference, suggesting a possible mechanism for this characteristic flow pattern.

Comparison With Previous Studies

Systolic coronary flow velocity reversals have previously been observed in patients with aortic stenosis. Although flow reversal was noted angiographically by Carroll and Falsetti[1] and with the epicardial Doppler technique by Fujiwara et al.,[2] the timing of flow reversal was different in each study. Retrograde flow was seen at end systole in the former, and in the first half of systole in the latter. In our study, flow reversal started from the beginning of systole and continued to midsystole or late systole. These differences might be due to the limitations of angiography with respect to temporal resolution of coronary blood flow. Doppler flow velocity measurement is thought to be more sensitive and reliable than angiographic analysis in this field.

Coronary blood flow velocity of the left anterior descending artery in patients with aortic stenosis has also been reported by Fusejima[17] with the transthoracic and epicardial Doppler technique. Only three cases were studied and the flow velocity pattern in systole was not described. Precise hemodynamic data were not presented. Furthermore, the transthoracic approach for recording of coronary flow may have a significant limitation in obtaining both an optimal echo window and angle correction between blood flow in the left anterior descending artery and the Doppler beam. These differences might affect the coronary flow velocity pattern observed. As previously noted Fujiwara et al.[2] first reported coronary artery systolic flow reversal at the time of open heart surgery in patients with aortic stenosis and normal coronary arteries by use of an epicardial approach with specially developed Doppler equipment. Their findings are confirmed by our Doppler study.

Possible Etiologic Mechanisms for Reversed Systolic Flow

Coronary driving pressure and impedance have been proposed as physiologic factors governing coronary flow.[1,18] Although inertia, capacitance, and resistance are thought to be included in the impedance component controlling coronary flow, resistance may be the most important of these factors. A marked accentuation in systolic compression of intramyocardial blood vessels due to an increased systolic pressure

Table 2 Parameters of coronary flow velocity recordings in patients with aortic stenosis and controls

	Case	Age (years)	Sex	HR (beats/min)	Peak velocity		Time velocity integral		
					Systole (cm/sec)	Diastole (cm/sec)	Flow reversal (cm)	Systole (cm)	Diastole (cm)
Patients with AS	1	49	M	63	−16	27	2.3	−0.9	13
	2	67	M	76	−36	67	4.8	−3.9	20
	3	68	F	61	−39	90	3.6	−2.8	34
	4	63	F	82	−10	55	0.8	−0.1	18
	5	52	M	63	−29	70	3.6	−1.5	33
	6	66	F	77	−20	97	1.6	−3.4	31
	7	61	F	81	−28	69	2.4	−0.1	23
	8	55	F	82	−17	89	1.3	−2.1	32
	9	69	F	72	−18	56	1.8	−1.3	19
	Mean ± SD	61 ± 7		73 ± 9*	−24 ± 10**	69 ± 22	2.5 ± 1.3	−0.3 ± 2.3*	25 ± 8
Controls	1	53	F	50	17	40	—	3.8	26
	2	64	F	62	18	50	—	3.8	27
	3	58	M	68	12	66	—	3.1	28
	4	38	M	56	21	73	—	5.0	33
	5	34	M	62	21	58	—	4.6	26
	6	76	M	55	14	61	—	3.1	24
	7	55	M	65	25	79	—	6.2	33
	8	50	M	50	10	27	—	2.6	14
	9	57	M	69	19	58	—	3.9	26
	Mean ± SD	54 ± 13		60 ± 7	17 ± 5	57 ± 16	—	4.0 ± 1.1	26 ± 6

AS, Aortic stenosis; *AVA,* aortic valve area; *HR,* heart rate; *LVM,* left ventricular mass index; *LVPW,* left ventricular posterior wall thickness; *PG,* pressure gradient; *TPV,* time to diastolic peak velocity; *VHT,* velocity half time.
*$p < 0.01$.
**$p < 0.001$ compared with controls.

Table 3 Parameters of coronary flow velocity recordings before and after aortic valve replacement in six out of nine patients with aortic stenosis

	Before AVR (n = 6)	After AVR (n = 6)	Controls (n = 9)
Heart rate (beats/min)	72 ± 8*	79 ± 7*	60 ± 7
Peak systolic velocity (cm/sec)	−28 ± 9*	21 ± 3	16 ± 6
Peak diastolic velocity (cm/sec)	75 ± 15	51 ± 18	53 ± 21
Time velocity integral (cm)			
Flow reversal	3.0 ± 1.2	0	0
Systole	−0.6 ± 2.6*	4.3 ± 1.7	3.8 ± 1.2
Diastole	27 ± 7	21 ± 8	24 ± 8
$TDVP/\sqrt{RR}$	5.4 ± 1.0	3.9 ± 13	4.1 ± 0.6
$VHT/\sqrt{RR}$	7.6 ± 2.1*	15.3 ± 1.9	13.0 ± 3.1
Left ventricular wall thickness (mm)	13 ± 2*	12 ± 1*	8 ± 1
Left ventricular mass index (gm/m^2)	211 ± 73*	208 ± 14*	95 ± 15

TDVP, Time to peak diastolic velocity; *VHT,* velocity half time.
*$p < 0.01$ compared with controls.

difference between the left ventricular cavity and the coronary artery may play an important role in the production of systolic flow reversal. This is suggested by the fact that the peak velocity of systolic flow reversal correlated significantly with the pressure difference across the aortic valve rather than peak systolic left ventricular pressure or aortic valve area. This pressure difference might increase the resistance of intramyocardial blood vessels and prevent blood from flowing forward normally. Furthermore, the pressure difference may increase left ventricular intramyocardial pressure and directly push blood back from the intramyocardial vessels into the epicardial coronary artery in systole. A lesser correlation was observed between peak velocity of systolic flow reversal and aortic valve area. As left ventricular func-

Table 2 (cont'd)

TPV (msec)	TPV/$\sqrt{RR}$	VHT (msec)	VHT/$\sqrt{RR}$	PG (mm Hg)	AVA (cm^2)	LVPW (mm)	LVMI (gm/m^2)
196	6.3	249	8.1	75	0.51	15	252
196	6.9	147	5.2	86	0.44	18	335
180	5.8	209	6.7	80	0.34	16	248
100	3.3	153	5.7	30	0.47	12	223
156	5.2	373	12.1	89	0.48	13	176
136	4.1	198	7.1	76	0.42	11	180
156	5.8	164	6.0	62	0.43	12	127
144	5.2	345	12.8	57	0.47	12	98
132	4.7	232	8.0	64	0.45	13	263
155 ± 32	5.3 ± 1.1*	230 ± 77*	8.0 ± 2.6*	69 ± 18	0.45 ± 0.05	14 ± 2*	211 ± 73**
136	3.9	588	17.0	—	—	7	83
132	4.0	520	15.9	—	—	8	84
120	4.0	419	14.1	—	—	9	106
108	3.3	339	10.4	—	—	9	93
104	3.3	486	15.6	—	—	8	73
136	4.1	305	9.2	—	—	8	93
132	4.3	436	14.4	—	—	8	83
152	4.4	260	7.5	—	—	7	105
146	4.9	379	12.8	—	—	10	95
130 ± 16	4.0 ± 0.5	415 ± 106	13.0 ± 3.1	—	—	8 ± 1	90 ± 11

tion was not markedly deteriorated in the patients studied, the correlation with aortic valve area most likely reflects the overwhelming influence of the pressure difference across the aortic valve in the calculation of valve area in this clinical setting.[19,20]

Another possible mechanism for the production of systolic flow reversal may be the Venturi effect. Bellhouse et al.[21,22] demonstrated that the turbulent jet produced during ejection with aortic stenosis can produce a low pressure region adjacent to the turbulent jet within the aortic sinuses. A pressure difference could possibly be generated across the orifice of the coronary artery, which might be able to reverse blood flow from the proximal coronary artery back into the aortic sinus. If this Venturi effect is a main cause of flow reversal during systole, flow reversal is likely to be observed not only in the left anterior descending coronary artery but also in the left circumflex and right coronary arteries. Coronary flow velocity recording in the right coronary artery was obtained in three out of nine patients with aortic stenosis during this study, and no systolic flow reversal was seen in systole. Further examination of coronary flow velocity recording in the right and left circumflex coronary arteries may be useful to prove or disprove this possible mechanism of flow reversal.

Diastolic Flow

Slow acceleration and rapid deceleration of diastolic flow velocity were constantly observed in patients with aortic stenosis compared with controls. Consequently, in those patients with aortic stenosis, a smaller time velocity integral was also expected. However, the diastolic time velocity integral was not significantly different in each group. The main factor affecting this may be heart rate. Coronary blood flow velocity is influenced by heart rate as demonstrated by the rate-related increase in resting coronary blood flow velocity observed during right atrial pacing.[12] In the present study, heart rate was significantly higher in patients with aortic stenosis than in the controls, and peak diastolic flow velocity in patients with aortic stenosis was slightly higher than that of controls. As a result of these opposing influences, diastolic time velocity integral in patients with aortic stenosis was not significantly less than that of controls.

The mechanism of slow acceleration and rapid deceleration of diastolic flow is not clear. Slow diastolic flow acceleration has been described in patients with hypertensive left ventricular hypertrophy[23] and in patients with aortic stenosis.[2] However, it may not be simply related to left ventricular hypertrophy, be-

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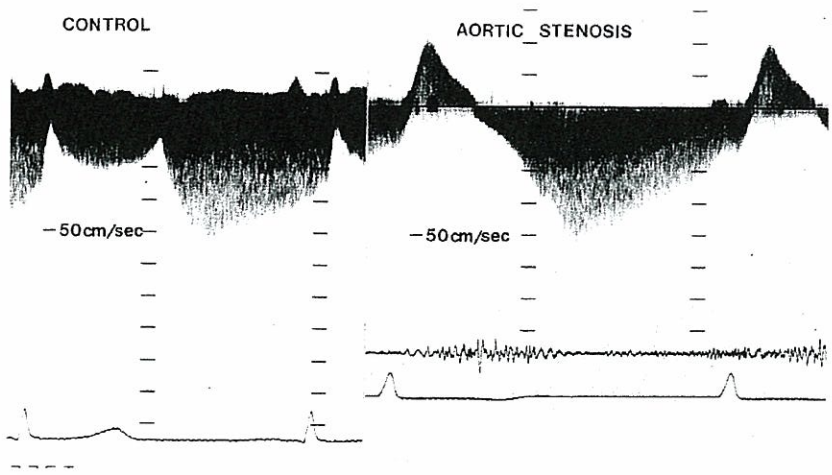

Figure 3 Examples of coronary flow velocity recordings in a control subject *(left)* and a patient with aortic stenosis *(right)*. Flow reversal in systole and slow acceleration and rapid deceleration of the flow in diastole are seen in a patient with aortic stenosis.

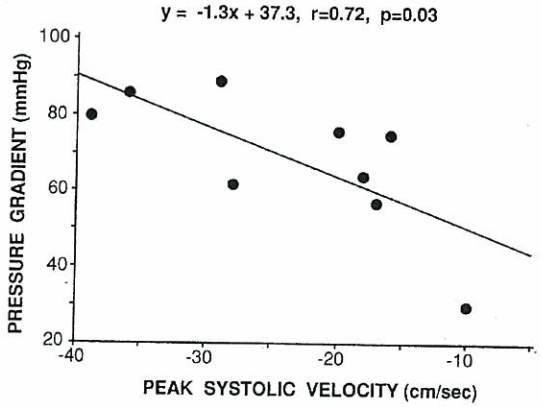

Figure 4 Plot of relation between peak velocity of coronary flow reversal and pressure gradient across the aortic valve in patients with aortic stenosis and normal coronary arteries. A significant linear correlation was noted between both parameters.

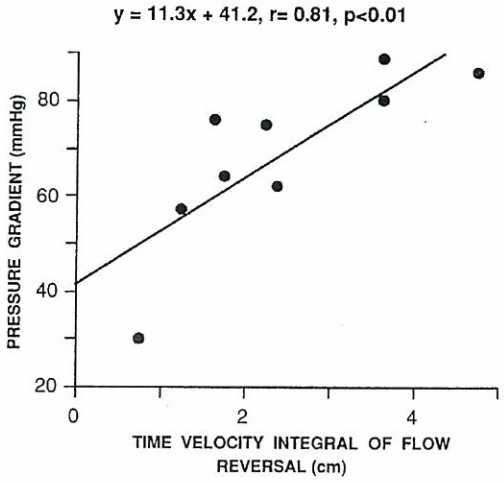

Figure 5 Plot of relation between time velocity integral of flow reversal and pressure gradient across the aortic valve in patients with aortic stenosis and normal coronary arteries. A significant linear correlation was also obtained between both parameters.

cause slow acceleration and rapid deceleration became normalized after aortic valve replacement despite the fact that left ventricular mass and wall thickness remained unchanged. The disappearance of slow diastolic flow acceleration has previously been observed soon after aortic valve replacement in patients with aortic stenosis.[2] Abnormal diastolic properties of the hypertrophied left ventricle due to "pressure overload" before valve replacement may be important. Comparison of left ventricular Doppler inflow velocity patterns before and after aortic valve replacement may be helpful to investigate this finding. No comparable data of left ventricular diastolic function before and after valve replacement were obtained in this study.

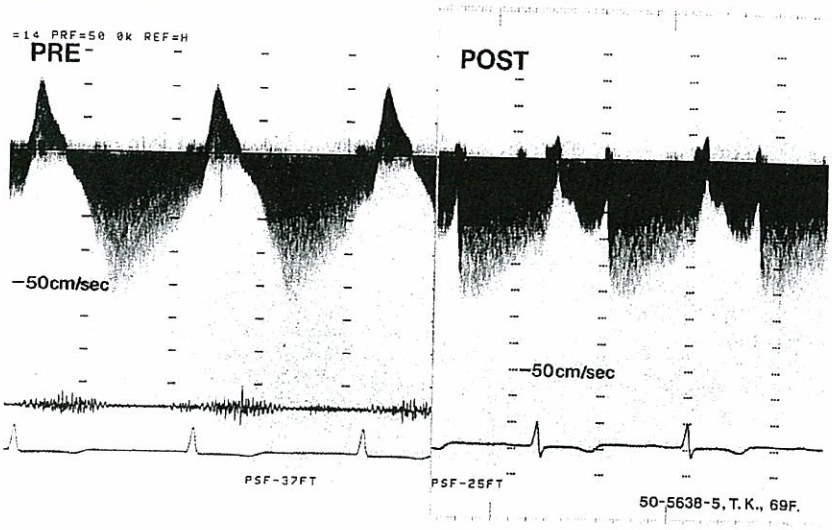

Figure 6 Examples of coronary flow velocity recording before *(PRE)* and after *(POST)* aortic valve replacement in a patient with aortic stenosis and normal coronary arteries. Characteristic pattern of flow reversal in systole and slow acceleration and rapid deceleration in diastole in patients with aortic stenosis is no longer present after aortic valve replacement.

High left ventricular end-diastolic pressure may also influence diastolic coronary blood flow. High intraventricular pressure may affect coronary flow as a result of increasing coronary impedance. Therefore a high end-diastolic left ventricular pressure might influence coronary flow by increasing diastolic coronary impedance. Similarly, coronary driving pressure in diastole is probably related to the difference between diastolic aortic pressure and coronary sinus pressure or left ventricular end-diastolic pressure.[1,18] In patients with aortic stenosis in this study, left ventricular end-diastolic pressure was significantly higher than that of controls and was also higher than mean right atrial pressure. The high end-diastolic pressure may therefore decrease acceleration of flow in diastole by a combination of increased coronary impedance and decreased driving pressure. Again if coronary driving pressure decreases rapidly because of high left ventricular end-diastolic pressure, diastolic flow velocity will reduce rapidly. Therefore, rapid deceleration might be observed. However, coronary flow regulation is obviously more complex,[24] and many other factors must be taken into consideration.

Limitations

Although only a relatively small number of subjects were studied, the Doppler flow characteristics observed were consistent in virtually all subjects with aortic stenosis, particularly with regard to systolic flow. However, subjects with marked deterioration in left ventricular function in cases with aortic stenosis were not included in this study. The peak flow velocity and time velocity integral of systolic flow reversal should be lower in patients with lower pressure gradients and smaller aortic valve area associated with impaired left ventricular function if the Doppler parameters are related to transvalvular pressure difference itself and not to aortic valve area or peak systolic left ventricular pressure. A study incorporating patients with impaired left ventricular function would clarify possible mechanisms of coronary flow in aortic stenotic patients.

As in all invasive human studies, truly normal controls are precluded for ethical reasons. Control subjects in this study were patients being investigated for chest pain. They were all normotensive and did not have left ventricular hypertrophy. However, they were not completely normal and may have small coronary artery disease or other abnormalities of myocardial perfusion that normal diagnostic coronary angiography cannot detect. Four of the controls had heart rates below 60 beats/min, and this appears to be somewhat low. Athletes who are not well conditioned might be included in the control subjects.

All pressure data were obtained with fluid-filled

transducers. More accurate pressure data could be obtained with high-fidelity micromanometer-tipped catheters, although differences in this study are likely to be only minor.

The Doppler catheter used in this study (3F) might cause some relative obstruction to coronary flow. The recently developed Doppler guidewire system might be more accurate in the measurement of the coronary artery flow velocity.[25]

Finally, measurements obtained during stress might be useful to resolve the mechanism of coronary flow dynamics observed.

CONCLUSIONS

We have observed a Doppler coronary blood flow velocity pattern in patients with aortic stenosis characterized by a flow reversal in systole and slow acceleration and rapid deceleration in diastole. We have shown a significant correlation between the magnitude of systolic flow reversal and the aortic valve pressure difference and postulate that a possible mechanism to explain these systolic flow reversals may be increased systolic coronary flow impedance due to the large difference between left ventricular intracavitary and proximal coronary artery pressure.

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