The prevalence of myocardial bridging and septal squeeze in patients with significant aortic stenosis[1]

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One hundred thirty-three cases of significant aortic valvular stenosis (mean systolic gradient = 93.3 ± 26.3 mm Hg) were studied to determine the prevalence of myocardial bridging and septal squeeze. Seven of 133 cases (5%) had myocardial bridging. Six of the 7 patients with myocardial bridging also had septal squeeze. Of the 126 patients without myocardial bridging, 92 (73%) had septal squeeze. The prevalence of myocardial bridging in patients with aortic stenosis (AS) is compared to that in patients with hypertrophic cardiomyopathy or normal left ventricles.

Index terms: Aortic valve stenosis • Myocardial bridging
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When 4 of the 5 patients with myocardial bridging reported by Ishimori et al[1] were noted to have left ventricular hypertrophy (LVH) secondary to hypertension, aortic stenosis (AS), and hypertrophic cardiomyopathy, it was suggested that LVH increases the prevalence of angiographically recognizable myocardial bridging because of an associated hypertrophy of the bridging fibers.

Although the prevalence of bridging is high in patients with LVH secondary to hypertrophic cardiomyopathy,[2,3] other investigators have been unable to demonstrate this relationship in patients with LVH secondary to AS.[3,4]

Methods

Review of the surgical log of the Department of Thoracic and Cardiovascular Surgery at The Cleveland Clinic Foundation identified 660 patients with significant aortic val-

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Table 1. Symptoms prior to cardiac catheterization

Predominant symptom		Aortic stenosis: no bridging No. patients		Aortic stenosis: bridging No. patients	Total
Angina pectoris	60		4		64 [48%]
Angina alone		16		1	
Angina and dyspnea		31		3	
Angina and syncope		5		—	
Angina, dyspnea and syncope		8		—	
Dyspnea on exertion	54		2		56 [42%]
Dyspnea alone		30		2	
Dyspnea and orthopnea, PND or edema		7		—	
Dyspnea and syncope		17		—	
Syncope	5		1		6 [5%]
Other	7		0		7 [5%]
Dizziness		2		—	
Palpitation		2		—	
Edema		2		—	
Fatigue		1		—	
Totals	126		7		133

PND = paroxysmal nocturnal dyspnea.

vular disease (AVD) who had had open heart surgery with aortic valve replacement (AVR) between 1975 and 1979. Patient records were reviewed retrospectively, and patients with aortic insufficiency greater than grade 2 (mild),[5] significant coronary atherosclerosis (greater than 50% lumen diameter reduction in any major coronary vessel), primary myocardial disease (PMD), or segmental impairment of myocardial contractility were excluded from further analysis.

The preoperative cineangiograms and hemodynamics of the remaining 133 patients were rereviewed to determine the degree of AS[5] and the prevalence of myocardial bridging[6] and septal squeeze.[4,7] Methods of film review and techniques for measurement of the bridged arterial segment have been reported previously.[6] Precatheterization clinical data in these 133 patients were obtained by retrospective chart review.

The chi-square test was used for statistical analysis, $P < 0.05$ being significant.

Results

Ninety-one men and 42 women, aged 18 to 80 years, (mean 59 ± 12) with significant aortic valve stenosis (AVS) were evaluated. All were symptomatic prior to catheterization (*Table 1*), the majority (90%) noting angina pectoris (AP) or dyspnea on exertion. The electrocardiogram (ECG) was abnormal[8] in 93 cases (70%) (*Table 2*), and the chest roentgenogram was abnormal in 65 cases (49%) (*Table 3*).

At catheterization, aortic valve calcification was apparent in all patients. In 121 of 133 cases (91%), retrograde transvalvular catheterization with pressure measurement and left ventricular angiography were accomplished. The aortic valve systolic gradient ranged from 45 to 160

Table 2. Electrocardiogram prior to cardiac catheterization

Electrocardiogram		Aortic stenosis: no bridging No. patients		Aortic stenosis: bridging No. patients	Total
Left ventricular hypertrophy	64		4		68 [51%]
LVH alone		46		2	
LVH and LAE		17		2	
LVH and LAHB		1		—	
Normal	39		1		40 [30%]
Bundle branch block	15		2		17 [13%]
CRBBB		7		—	
CLBBB		8		2	
Left atrial enlargement	8				8 [6%]
LAE alone		4		—	
LAE and BBB		4		—	

LVH = left ventricular hypertrophy; LAE = left atrial enlargement; LAHB = left anterior hemiblock; CRBBB = complete right bundle branch block; CLBBB = complete left bundle branch block.

Table 3. Chest roentgenogram prior to catheterization

Chest roentgenogram		Aortic stenosis: no bridging No. patients		Aortic stenosis: bridging No. patients	Total
Left ventricular enlargement	61		2		63 [47%]
LVE alone		51		2	
LVE and LAH		10		—	
Left atrial hypertrophy	2		0		2 [2%]
Normal	63		5		68 [51%]

LVE = left ventricular enlargement, LAH = left atrial hypertrophy.

mm Hg (mean 93.3 ± 26.3 mm Hg). Aortography was performed in all patients and showed aortic insufficiency in 94 patients (71%): grade 1 (trivial)—75 patients and grade 2 (mild)—19 patients. The left ventricle was visualized in 124 of 133 patients (left ventriculogram—121 patients, pulmonary angiogram—3 patients). Angiographic signs of LVH (increased trabecular pattern, increased papillary muscle size, and increased free wall thickness) were present in all cases. Overall contractility was normal in 74 patients, mildly impaired in 18, moderately impaired in 14, and severely impaired in 18. In the 9 patients without ventriculography, 8 showed electrocardiographic evidence of LVH, and all 9 had LVH at surgery.

Myocardial bridging of the left anterior descending artery was found in 7 patients (5%). No other vessel was involved. Reduction in lumen diameter during systole varied 14%–100% (mean 40.3 ± 28.5%) over an arterial length of 4 to 20 mm (mean 8.7 ± 5.5 mm). Six of these 7 patients also had septal squeeze. In the 126 patients without myocardial bridging, septal squeeze was seen in 92.

Discussion

Comparison of the prevalence of myocardial bridging and septal squeeze in patients with normal left ventricles and in those with abnormal left ventricles secondary to obstructive hypertrophic cardiomyopathy or AS is shown in *Table 4*. Myocardial bridging was seen in 81 of 658 patients (12%) with normal left ventricles,[6] in 7 of 133 (5%) with LVH secondary to AS, and in 20 of 66 (30%) with obstructive hypertrophic cardiomyopathy.[2] Prevalence of bridging was significantly greater in patients with cardiomyopathy compared to those with normal left ventricles or LVH secondary to AS ($x^2 = 20.34$, $P < 0.001$). These results support the observations of Bourmayan[3] and Pichard[4] and suggest that increased angiographic recognition of myocardial bridging in cardiomyopathy may not depend solely on the presence of LVH.

In contrast, septal squeeze does appear to be related to the presence of LVH or a systolic pressure gradient or both.[7] Septal squeeze was not seen in patients with normal left ventricles,[6] but occurred in 47 of 66 (71%) with obstructive hypertrophic cardiomyopathy and in 98 of 133 (74%) with LVH secondary to AS. The difference in the prevalence of septal squeeze between patients with normal ventricles and no pressure gradient as compared to patients with abnormal ventricles and a pressure gradient was significant ($x^2 = 577$, $P < 0.0001$).

The angiographic severity of myocardial bridging clearly varies in response to nitroglycerin,[2] isosorbide dinitrate, sodium nitroprusside, and noradrenalin.[9] Angiographic prevalence of myocardial bridging also appears to differ in relation to the underlying cardiac pathology. Myocardial

Table 4. Prevalence of myocardial bridging and septal squeezing in patients with normal left ventricles,[6] obstructive hypertrophic cardiomyopathy,[2] and aortic stenosis

Patient group	Total no. patients	Total with bridging	Total with squeeze	Bridging alone	Bridging and squeezing	Squeezing alone
Normal left ventricle	658	81 (12%)	0	81 (12%)	0	0
Obstructive hypertrophic cardiomyopathy	66	20 (30%)	47 (71%)	2 (3%)	18 (27%)	29 (44%)
Aortic stenosis	133	7 (5%)	98 (74%)	1 (1%)	6 (5%)	92 (69%)

bridging is seen most commonly in patients with hypertrophic cardiomyopathy. This frequent unexplained association is likely not due simply to the presence of hypertrophied bridging fibers.

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