Surgical treatment of idiopathic hypertrophic subaortic stenosis with other cardiac pathology[1]

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Twenty-nine patients underwent surgery for idiopathic hypertrophic subaortic stenosis (IHSS) solely or in combination with other cardiac pathology. Fifteen men and 14 women, 26 to 75 years of age (mean 56 years), had peak subvalvular gradients, with or without provocation, ranging up to 220 mm Hg (mean 93 mm Hg). The diagnosis of IHSS was confirmed preoperatively by echocardiography in the 23 patients on whom it was performed. Twenty-seven patients underwent left ventricular septal myectomy (LVSM), one had a left ventricular myotomy, and another received mitral valve replacement (MVR) alone for palliation of IHSS. In addition, coronary artery bypass (CAB) was performed in 18 of these IHSS patients: 8 had single grafts, 5 had two, 4 had three, and one patient had five grafts. Other operations included MVR in 3 patients, aortic valve replacement, excision of a subaortic membrane, and tricuspid annuloplasty in one patient each. One operative death (3%) occurred 34 days postoperatively. There have been 2 late deaths: one at 22 months from bacterial endocarditis on a prosthetic mitral valve, and another suddenly at 29 months after septal myectomy. After a follow-up ranging from 2 to 93 months (mean 25 months), 22 of 26 patients are asymptomatic. Three asymptomatic patients have been recatheterized 12, 14, and 21 months, respectively, after operation. Marked amelioration of subaortic gradients was noted, and patent grafts were found. Left ventricular septal myectomy (LVSM) can be combined with other cardiac operations to provide effective palliation.

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At The Cleveland Clinic Foundation, left ventricular septal myectomy or myotomy was performed on 28 pa-

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tients, and one had mitral valve replacement alone for treatment of idiopathic hypertrophic subaortic stenosis (IHSS). Coronary artery bypass grafting, aortic valve replacement, excision of a subvalvular membrane, mitral valve replacement, and tricuspid valve annuloplasty, when indicated, were performed concomitantly with septal myectomy. This report reviews our experience with left ventricular septal myectomy for severe symptomatic IHSS and simultaneous correction of other cardiac pathology.

Materials and methods

Between January 1974 and October 1981, 29 patients underwent surgery for IHSS at The Cleveland Clinic Foundation; 26 of these had been operated on since 1978. There were 15 men and 14 women, whose age ranged from 26 to 75 years (mean 56 years). Twenty-seven of these were New York Heart Association functional class III or IV. One was class I, and another was class II (*Table 1*).

Nine patients presented with angina pectoris alone. In 2 patients, the predominant symptom was syncope and in one, congestive heart failure. The remaining 17 had varying combinations of symptoms (*Table 2*). Twenty-six of the 29 patients presented with angina alone or combined with other symptoms.

Peak subvalvular gradients with or without provocation ranged up to 220 mm Hg (mean 93 mm Hg). The gradient and symptoms on presentation or the type of associated pathology were not correlated. One patient with systolic anterior motion of the anterior leaflet of the mitral valve and disproportionate septal thickening had no gradient at rest and was unstable during catheterization, so no provocation test was done. He had severe triple vessel coronary artery disease. At surgery, localized hypertrophy of the muscular septum with endocardial thickening was found and resected, and coronary artery bypass grafting performed.

Echocardiography, done in 23 of the 29 patients, suggested the diagnosis in all in whom it was performed. A septal-free wall ratio of greater than 1.3 was present in 14 patients. Systolic anterior motion of the anterior mitral leaflet was noted in 19, and both conditions were present in 11 patients.

Twenty patients had significant coronary artery disease with luminal stenosis of at least 50%. Four had triple vessel disease; eight, double and eight, single. The patient with aortic valvular disease had aortic stenosis secondary to calcification of a congenitally bicuspid aortic valve. The gradient across the valve was 100 mm Hg. A subvalvular membrane was present in one patient with echocardiographic evidence of septal hypertrophy. He demonstrated the highest gradient in the series, 220 mm Hg with Isuprel provocation. Nine patients had angiographic evidence of mitral valve disease, 4 with severe and 5 with moderate mitral regurgitation. All 9 had systolic anterior motion on the echocardiogram. Four of the patients with moderate and one with severe mitral regurgitation on exploration had a normal valve. Twenty patients had mild or no mitral regurgitation (*Table 3*). The type and number of each operation performed are listed in *Table 4*. Intraoperatively, gradients with and without Isuprel stimulation were checked in 19 patients. In 17 patients, the gradients ranged from 0 to 20 mm Hg. In one patient who had a left ventricular septal myectomy, the gradient was 40 mm Hg with Isuprel. In another patient having septal myectomy, the gradient with Isuprel was 50 mm Hg. No further septal resection was thought technically possible.

All surgery was accomplished via standard cardiopulmonary bypass techniques, systemic hypothermia, and direct infusion of cold potassium cardioplegic solution into the coronary arteries

Table 1. NYHA functional class before and after surgery

	Preoperative	Postoperative (at most recent follow-up)
I	1	22
II	1	2
III	19	2
IV	8	

Table 2. Symptoms on presentation

Angina	9
Syncope	2
Congestive heart failure	1
Angina/syncope/congestive heart failure	3
Angina/syncope	7
Angina/congestive heart failure	7

Table 3. Associated diseases

Coronary artery disease	20
Aortic valve disease	1
Subvalvular membrane	1
Mitral valve disease	4
Tricuspid disease	1

Table 4. Operative procedures

CABG + myectomy	13
CABG + myotomy	1
CABG + myectomy + MVR + TVA	1
CABG + myectomy + MVR	1
CABG + MVR	1
Myectomy + MVR	1
CABG + myectomy + AVR	1
Myectomy + excision of subaortic membrane	1
Myectomy	9

AVR = aortic valve replacement; CABG = coronary artery bypass graft; MVR = mitral valve replacement; TVA = triscupid valve annuloplasty.

for myocardial preservation. If coronary artery bypass was performed, the distal anastomoses were done first. The subaortic area was then inspected and myectomy done. If intrinsic mitral valve disease was suspected, the valve was explored and replaced, if indicated. If necessary, the aortic valve was then replaced and the aortotomy closed. The tricuspid valve was then explored and repaired or replaced, as indicated. The patient was rewarmed and the aortic cross-clamp removed. If aortocoronary bypass grafts (CABG) had been done, the proximal anastomoses were constructed with the heart beating during rewarming.

Results

One patient died 34 days postoperatively (operative mortality 3%). She had undergone CABG, myectomy, and mitral valve replacement. The postoperative course was complicated by low cardiac output, tamponade requiring reoperation and subsequent acute renal failure, mediastinitis, and septic shock. There have been 2 late deaths, one at 22 months from bacterial endocarditis of a prosthetic mitral valve, and the other suddenly at 29 months after myectomy. The late mortality attributed to cardiomyopathy was 3.7%.

There were no surgically created ventricular septal defects. One patient developed intermittent complete heart block. His-bundle studies showed prolonged His-ventricle (HV) interval for which a pacemaker was implanted. At most recent follow-up, she was in a slow sinus rhythm with occasional paced beats. Four other patients had a bundle branch block, and 3 had left anterior hemiblock. In 10 patients, atrial fibrillation, not present preoperatively, developed postoperatively. This persisted in all patients despite medical therapy. In 4 patients, normal sinus rhythm was present on follow-up ECG. Two patients had atrial fibrillation, and in 4, no ECG was available. Three patients had frequent premature ventricular contractions, and one had premature atrial contractions. These responded to medical therapy, and follow-up ECGs were normal. One patient developed a short run of ventricular fibrillation, the etiology of which was not clarified. This converted spontaneously to sinus rhythm, and with medical treatment, no further problems developed. Other complications are listed in *Table 5*.

Follow-up is from 2 to 93 months (mean 25 months). Twenty-two patients are asymptomatic, 2 are New York Heart Association functional Class II, and 2 are Class III. One of the Class III patients who underwent CABG and myectomy was asymptomatic for 27 months, then developed recurrent angina and shortness of breath. He has refused recatheterization. The other Class III patient had initial CABG which failed to relieve his angina. Because of continued chest pain and evidence of subaortic stenosis and mitral regurgitation, the mitral valve was replaced 25 months later. He remains symptomatic.

In the one patient who died postoperatively and in the 2 with late death, intraoperative gradients measured with Isuprel ranged from 0 to 20 mm Hg. In the 2 patients who were functional Class III, intraoperative gradients were 0. One of the functional Class II patients underwent a left ventricular septal myectomy and had a 50 mm Hg gradient with Isuprel stimulation. The other functional Class II patient had left ventricular septal myectomy and CABG, but no intraoperative gradient was measured.

Nine patients had repeat echocardiography. Four had systolic anterior motion of the anterior mitral leaflet, and one had a septal-free wall ratio of greater than 1.3. Four of these were functional Class I (2 myectomy and 2 CABG plus myectomy). One patient who had CABG and myectomy was Class II.

Three asymptomatic patients have been re-

Table 5. Complications

Complete heart block (intermittent)	1
Bundle branch and left anterior hemiblock	7
Atrial fibrillation	10
Other arrhythmia	5
Myocardial infarction	1
Low cardiac output	5
Neurologic	1
Infection	1
Reopened for bleeding tamponade	4

catheterized at 12, 14, and 21 months postoperatively. Each had had CABG and myectomy, and grafts were patent. Subaortic gradients were essentially zero in 2 patients, whereas the third had a gradient of 30 mm Hg at rest and 70 mm Hg with amyl nitrate provocation. Repeat echocardiography suggested recurrent stenosis; on recent follow-up, however, he remains asymptomatic.

Discussion

Patients with severe symptomatic IHSS unresponsive to medical therapy have obtained safe and effective palliation from left ventricular septal myectomy (LVSM). Little has been reported about other cardiac pathology occurring in the patient with IHSS. The surgical series of IHSS patients at The Cleveland Clinic Foundation includes those with associated aortic valvular disease, subvalvular membrane, mitral and tricuspid valvular disease, and coronary artery disease (CAD). The combination of CAD and IHSS is of particular interest because both may present with similar symptoms. Stewart and Schreiner[1] recently described 3 patients with CAD and IHSS treated successfully by coronary artery revascularization and LVSM. A review of the literature produced only 7 other such cases,[2–6] a small number considering 25% of patients with IHSS over 45 years of age will have significant CAD.[7] Isolated reports in the literature have cited concomitant surgical treatment of IHSS and partial anomalous pulmonary venous drainage,[8] aortic,[1,3,4,9] and mitral valve disease.[3,8–11]

The surgical technique of LVSM has been amply described by Morrow and others.[12,13] The history leading to its development makes for an interesting review.[14–27] With the transaortic approach and septal resection, a low operative mortality and morbidity with good functional results have been obtained.[3]

Once the diagnosis of IHSS is made on a clinical and echocardiographic basis, medical treatment with beta blockers is usually begun. In a third of these patients, symptoms progress despite optimal medical management, and surgery becomes a consideration.[28] As noted here, other pathology may be present, and cardiac catheterization is necessary to confirm the presence of a significant subaortic gradient and to define associated cardiac pathology.

Operation for isolated IHSS is indicated when symptoms are not adequately controlled by medical therapy and a gradient of greater than 50 mm Hg at rest exists secondary to hypertrophy of the septum. Operation should also be done for patients with severe symptoms and obstruction only with provocation. Recently, Morrow et al[29] have advocated surgery for patients with documented IHSS and previous history of cardiac arrest without other symptoms. Each additional abnormality must be evaluated regarding its severity, and, if indicated, these should be corrected simultaneously, with a low operative mortality and morbidity and a high expectation of symptomatic improvement.

Review of the literature on the surgical treatment of IHSS shows that good results have been reported for isolated IHSS, but little has been said about associated disease. From 1970–1981, 360 cases of IHSS treated surgically were reported.[1,3,6,8,10,11,13,19,24,29,30] Overall operative mortality was 8%. Functional Class I or II status was attained in 72% of cases. In those patients with IHSS and other pathology, only 33 patients undergoing combined surgery were described during the same time interval.[1–6,8–11,30] The operative mortality was 15%, and 64% of the patients were functional Class I or II.

Of the 29 patients treated operatively for IHSS at our hospital, 20 had combined procedures, and 9 had septal myectomy alone. There were no deaths in the myectomy group; one patient died postoperatively in the combined group (operative mortality 5%). There were 2 late deaths. One occurred at 22 months from bacterial endocarditis of a prosthetic mitral valve; the other patient died suddenly at 29 months after myectomy. Persistent atrial fibrillation developed postoperatively, and his death was presumably from arrhythmia. Maron[3] has noted an increased late mortality for those in atrial fibrillation. No postmortem examination was done in either patient.

Mitral valve replacement has been recommended as the primary treatment for IHSS by Shumaker and King[23] in 1965 and by Cooley et al[24] in 1971. This recommendation was based on studies showing that asymmetric septal hypertrophy dislocates the apex and papillary muscles anterosuperiorly.[31] During contraction, this malalignment is accentuated and results in the anterior mitral leaflet being pulled toward the hypertrophied septum. This, in turn, obstructs the left ventricular outflow tract during systole and subsequently causes mitral regurgitation. The mitral leaflets may become thickened, presumably sec-

ondary to trauma. Usually, however, they are structurally normal unless diseased by rheumatic, degenerative, or other processes. Cooley et al[32] described 27 patients treated primarily with mitral valve replacement. There was one operative death and 5 late deaths. Thirteen of 21 patients in follow-up were functional Class I or II. In our series, moderate or severe mitral regurgitation was documented in 8 patients preoperatively. At operation, 5 of these had normal valves. The others had intrinsic valvular disease, and mitral valve replacement was done. As noted by Roberts,[33] several complications are inherent to prosthetic valves used in patients with IHSS. Placement of the valve in a small hypertrophied ventricular cavity can result in injury to the ventricle and a significant gradient across the valve. There is the potential of periprosthetic leak from technical or infectious causes, as seen in our patient who died late postoperatively. There is the problem of embolic phenomena and the need for anticoagulation. The question of long-term durability of the valve itself makes the replacement of a normal-appearing valve unjustified.

Follow-up of our remaining 26 patients ranged from 2 to 93 months (mean 25 months). Good results were obtained in 24 patients. Two patients from the combined surgery group were functional Class III. One of these who underwent CABG and myectomy was asymptomatic for 27 months, then developed recurrent angina and shortness of breath. He has refused recatheterization. The other had initial CABG without relief of angina. Because of continued chest pain and evidence of subaortic stenosis and mitral regurgitation, the mitral valve was replaced. He remains symptomatic.

The natural history of patients with IHSS remains imcompletely understood because of variable treatment forms and relatively short follow-up periods. In a large cooperative study, Shah et al[34] reported a late mortality secondary to cardiomyopathy of 7% in surgically treated patients (mean follow-up 5.2 years). This is in agreement with the suggestion of a 1.8% per year late mortality rate by Maron et al.[3] In our series, one patient died late secondary to cardiomyopathy, for a rate of 3.7%. Shah and colleagues[34] also determined a late mortality rate of 19% and 23%, respectively, for patients treated medically and those not receiving treatment (mean follow-up 4.7 and 6.7 years). Sudden death, presumably from arrhythmias, was the most common cause of late death in both series regardless of the form of treatment. Although suggested in their studies, it remains unclear whether myectomy decreases the risk of sudden death and improves long-term survival. A longer follow-up is needed to resolve this question. Surgery must be regarded as palliative therapy, since it does not alter the underlying disease process. Significant relief of symptoms can be expected with surgery, as is evident in the literature review and our own series (*Tables 6 and 7*). Patients with documented IHSS and a history of cardiac arrest should undergo cardiac catheterization. If a significant subaortic gradient is found, surgery is recommended.

Table 6. Literature review of surgical treatment of isolated IHSS—1971–1981

Date	Author	Number of patients	Operative mortality	Follow-up (mean)	Functional class (I or II)	Late cardiac deaths
1981	Stewart[1]	11	0	?	11	0
1981	Jeffery[10]	17	1	5.8 yrs.	11	4
1979	Morrow[29]	9	1	3.1 yrs.	7	1
1978	Maron[3]	114	10	5.2 yrs.	82	11
1977	Agnew[19]	49	2	7.4 yrs.	39	6
1976	Reis[13]	30	0	3.0 yrs.	29	1
1976	Senning[30]	26	1	?	22	2
1976	Jaumin[8]	16	2	6.3 yrs.	11	2
1974	Tajik[11]	37	3	7.0 yrs.	27	4
1972	Gulotta[6]	1	1	—	—	—
1971	Cooley[24]	50	8	?	?	3
	TOTAL	360	29	5.4 yrs.	239	34
	Percent		8%		72%	10%
1984	Duda	9	0	2.1 yrs.	8	1
	Percent		0%		89%	11%

Table 7. Literature review of surgical treatment of IHSS and other cardiac pathology—1972–1981

Date	Author	Number of patients	Operative mortality	Follow-up yrs.	Functional class (I or II)	Late cardiac deaths
1981	Stewart[1]	6	0	?	6	0
1981	Jeffery[10]	3	0	6.7	0	2
1979	Scully[9]	1	1	—	—	—
1979	Bensaid[2]	1	0	?	1	0
1978	Maron[3]	6	1	5.2	?	/
1977	Cohen[4]	1	0	0.3	1	0
1976	Senning[30]	4	0	?	2	1
1976	Jaumin[8]	5	1	6.3	4	0
1974	Tajik[11]	3	1	?	2	0
1974	Marcus[5]	1	0	1.1	1	0
1972	Gulotta[6]	2	1	?	1	0
	TOTAL	33	5	3.9	16	3
	Percent		15%		64%	11%
1984	Duda	20	1	2.1	17	1
	Percent		5%		89%	5%

In conclusion, IHSS may exist alone or in combination with other cardiac pathology. The operative correction of all abnormalities can be done with low mortality and good functional results. Long-term survival is not adversely affected and may be improved with surgical therapy.

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