

Significance of angina pectoris in aortic valve stenosis

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Of 60 patients aged 45 to 66 years with aortic valve stenosis, 28 (47 per cent) had angina pectoris. Significant coronary arterial obstruction was shown by selective coronary cineangiography in 14 of them. Systolic pressure gradients across the aortic valve were lower in patients with angina than in those without. In those with angina, systolic gradients were higher in those with normal coronary arteriograms than in those with demonstrable coronary arterial disease.

Aortic valve replacement relieved the angina in all patients who had normal coronary arteriograms. When valve replacement was combined with coronary bypass grafting in those with coronary arterial disease, surgical mortality was higher and symptomatic relief less predictable.

Incapacitating angina in patients with aortic stenosis was nearly always associated with significant coronary disease. In those with less severe angina it was impossible to predict the state of the coronary arteries. Two patients, who did not have angina and who did not undergo coronary arteriography, died after aortic valve replacement and were found at necropsy to have unsuspected severe coronary disease. We, therefore, suggest that coronary arteriography should be carried out in all patients over the age of 40 years in whom surgery is being considered for aortic stenosis.

Angina pectoris is a common symptom of aortic valve stenosis (Lewes, 1951; Mitchell *et al.*, 1954; Wood, 1958; Baker and Somerville, 1959; Basta *et al.*, 1975) and may occur in the absence of coronary artery disease (Linhart *et al.*, 1968; Hurst and Logue, 1970; Heulin *et al.*, 1973; Basta *et al.*, 1975). It is probably caused by ischaemia of a hypertrophic left ventricle which is subject to an excessive work load (Bonchek, Anderson, and Rösch, 1973). Coronary arteriography shows, however, that many such patients have significant coronary arterial obstruction (Loop *et al.*, 1972; Bonchek *et al.*, 1973; Berndt *et al.*, 1974; Basta *et al.*, 1973, 1975). This has a new importance now that coronary artery bypass grafting can be undertaken in addition to aortic valve replacement (Flemma *et al.*, 1971; Loop *et al.*, 1972; Anderson *et al.*, 1973; Berndt *et al.*, 1974). We, therefore, studied a series of patients with aortic valve stenosis who had angina pectoris, in order to assess the role of coronary artery disease in causing this symptom.

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Patients and methods

We investigated 73 consecutive adult patients with dominant aortic stenosis during the 25 months ending in January 1975. There were 53 men and 20 women and their ages ranged from 20 to 71 years. They were questioned regarding any history of chest pain. Angina pectoris was deemed to be present if they suffered from episodic retrosternal or praecordial pain with or without radiation to the neck, shoulders, or arms, which was brought on by effort, emotional stress, or exposure to cold, and relieved by rest or nitroglycerin. The severity of angina was graded as follows: grade 1—angina only with severe exertion; grade 2—angina with ordinary activity, emotional stress, or exposure to cold; grade 3—angina at rest, as well as with exertion or stress.

Risk factors such as family history of ischaemic heart disease, hypertension, excessive smoking, diabetes mellitus, obesity, and hyperlipidaemia were assessed in all those who had angina pectoris.

We judged the severity of the aortic stenosis from the peak systolic pressure gradient across the aortic valve. All cases had a gradient of more than 10 mmHg (1.3 kPa). Patients with significant aortic

TABLE 1 *Comparison of 60 patients aged 45 to 66 years with and without angina pectoris*

	Male	Female	Total	Mean age (y)
No angina	24	8	32	54.2
Angina pectoris	Grade 1	6	5	11
	Grade 2	8	1	9
	Grade 3	6	2	8
All	20	8	28	58.0

regurgitation or mitral valve disease were excluded from the study.

Selective coronary cineangiography was accomplished with either Sones or Judkins catheters in all patients with angina pectoris. Significant coronary artery disease was judged to be present when there was a narrowing of more than 60 per cent of one or more of the main coronary vessels.

Results

Of the 73 patients, 28 had angina pectoris. Their ages ranged from 45 to 66 years. They were compared with the 32 patients in the same age range, who had aortic stenosis but not angina pectoris, the other 13 being excluded from further consideration. The two groups were very similar as regards age and sex (Table 1). Of the 28 patients with cardiac pain, 11 had grade 1 angina pectoris, 9 grade 2, and 8 grade 3.

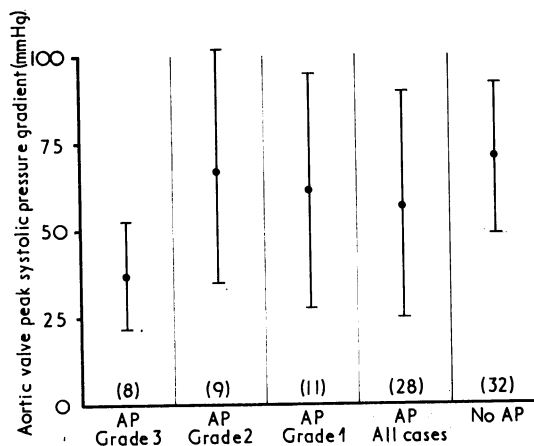
The mean of the peak systolic pressure gradients across the aortic valve was distinctly lower in those with angina pectoris than in those without (Table 2) (Fig. 1). Thus in the former group the range was 10 to 125 mmHg (1.3 to 16.6 kPa), with a mean of 56.9 mmHg (7.6 kPa), compared with 10 to 180 mmHg (1.3 to 23.9 kPa), with a mean of 70.3 mmHg (9.3 kPa) in the latter group of patients. In the 8

TABLE 2 *Average peak systolic pressure gradients in relation to degree of angina pectoris*

	No. of cases	Peak gradient (mmHg)*	
Angina pectoris	Grade 3	8	37.5 (± 15.6)*
	Grade 2	9	68.3 (± 33.9)
	Grade 1	11	61.8 (± 33.0)
	All	28	56.9 (± 32.1)†
No angina	32	70.3 (± 21.4)†	

*The difference between the pressure gradient in patients with grade 3 angina and in those with no angina is highly significant ($P < 0.001$).

†The difference between the pressure gradient in all those with angina and in those without is not statistically significant. Conversion from Traditional Units to SI Units: 1 mmHg \approx 0.133 kPa.

FIG. 1 *Aortic valve pressure gradient according to presence and severity of angina*

patients with severe (grade 3) angina the peak systolic pressure gradient was lowest of all (37.5 mmHg (5.0 kPa)) with a range of 10 to 55 mmHg (1.3 to 7.3 kPa). This was significantly lower than the gradient in those without angina ($P < 0.001$).

Coronary arteriography (Table 3) showed significant coronary artery disease in 14 of the 28 patients with angina, while in the other 14 the coronary arteries were considered normal. When the extent of the coronary disease was compared with the severity of angina pectoris we found that the more extensive the coronary artery disease, the more severe the angina pectoris tended to be. Thus 8 of the 11 patients with grade 1 angina had normal coronary arteriograms and the other 3 only single vessel disease; while of the 8 with grade 3 angina pectoris only 1 had a normal coronary arteriogram, whereas 5 of the other 7 had more than 1 coronary artery affected. These observations indicate that, while severe aortic stenosis often causes angina pectoris in the absence of demonstrable coronary artery disease, the angina tends to be mild or

TABLE 3 *Extent of coronary artery disease in patients with angina pectoris*

Severity of angina pectoris*	Coronary arteriogram				
	Normal	1-vessel disease	2-vessel disease	3-vessel disease	4-vessel disease
Grade 1 (11)	8	3	0	0	0
Grade 2 (9)	5	2	2	0	0
Grade 3 (8)	1	2	3	1	1
Total (28)	14	7	5	1	1

*Number of patients in parentheses.

TABLE 4 Coronary arteriograms in severe and less severe angina pectoris

Severity of angina pectoris	Normal coronary arteriogram	Abnormal coronary arteriogram
Grade 1 and 2	13	7*
Grade 3	1	7*

*The difference between patients with grade 3 angina and those with lesser degrees of angina is statistically significant ($P < 0.05$).

moderate and rarely incapacitating. Severe angina pectoris in our patients was associated with significant coronary disease in all but one of 8 patients (Table 4). The histogram (Fig. 2), which includes data on all 28 patients with angina pectoris, indicates that those with normal coronary arteries had gradients of widely ranging severity, but when coronary disease was present there was a predominance of milder aortic stenosis as judged from the peak systolic pressure gradient. In the patients with angina pectoris, the aortic valve gradient in those who had normal coronary arteries averaged 70.7 mmHg (9.4 kPa) (± 31.7) compared with 42.5 mmHg (5.7 kPa) (± 25) in those with coronary artery disease (Fig. 3). The difference was most pronounced in patients in whom two or more of the coronary vessels were affected.

Assessment of risk factors in the patients with angina pectoris revealed that 20 of the 28 patients did in fact have evidence of one or more such factors (family history of ischaemic heart disease, hyperlipidaemia, hypertension, diabetes mellitus, excessive cigarette smoking) (Table 5). Of those with 2 or more risk factors, 7 out of 10 had coronary artery disease (70%) while among the 18 with one or no risk factors 7 had coronary artery disease (39%) and 11 had normal coronary arteriograms. Eleven out of 14 patients with coronary disease (79%) had one or more risk factors as compared

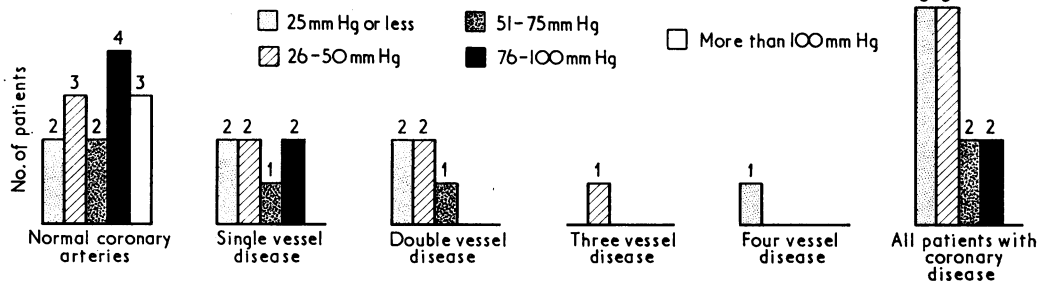


FIG. 2 Aortic valve gradient in patients with angina in relation to the extent of coronary artery disease

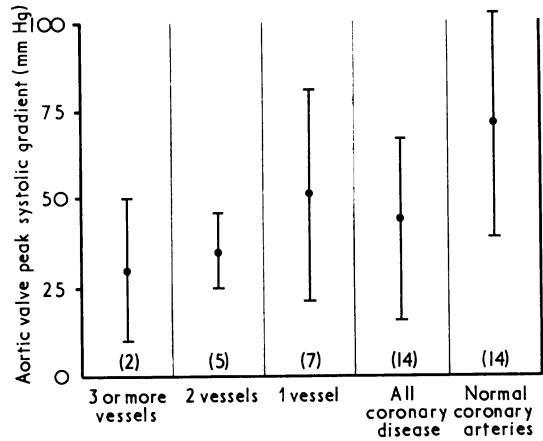


FIG. 3 Aortic valve gradient in patients with angina according to the extent of coronary disease

with 9 out of 14 of those with normal coronary arteries (64%) (Table 5).

Of the 28 patients, 20 subsequently underwent surgical treatment (Table 6). In 12 of the 14 with normal coronary arteriograms, aortic valve replacement alone was carried out with an operative mortality of 1 (8%). One patient required a second operation some months later because of a paraprothestic leak. In the remaining 10 patients angina pectoris was completely relieved. In 8 of 14 patients

TABLE 5 Risk factors in relation to coronary arteriographic findings in 28 patients with angina pectoris

Coronary arteriogram	Risk factors				
	None	One	Two	Three	Total with or more risk factors
Normal (14)	5	6	3	—	9 (64%)
Abnormal (14)	3	4	5	2	11 (79%)

TABLE 6 *Results of surgery in the 60 middle-aged patients (aged 45 to 66 years)*

	Total	No. operated	Surgical mortality	Angina		
				Abolished	Improved	Unchanged
Patients without angina	32	29*	2 (7%)	—	—	—
Patients with angina	14	12*	1 (8%)	11	—	—

*Aortic valve replacement only.

†Aortic valve replacement plus saphenous vein bypass grafting.

with abnormal coronary arteriograms, one or more saphenous vein bypass grafts were inserted at the same time as the aortic valve was replaced. Two of these patients died shortly after operation (25% mortality). At necropsy, both these patients were shown to have extensive coronary atheroma with healed myocardial infarction. A further patient died suddenly a few weeks after operation from a myocardial infarction; the saphenous vein graft was shown to be patent at necropsy. Of the 5 surviving patients, angina was abolished in 2, improved in another 2, and unchanged in 1. Of the 8 patients who were not operated upon, 2 died within 12 months of investigation, one suddenly, and the other from congestive heart failure. Of the 32 patients who did not have angina, 29 underwent aortic valve replacement, with an operative mortality of 2 (7%). Both these patients were found at necropsy to have severe coronary artery disease with healed myocardial infarction which had not been suspected clinically or by electrocardiography.

Discussion

Our findings confirm previous reports that angina pectoris is a common symptom of aortic stenosis. Thus, 47 per cent of the middle-aged patients in our series had angina pectoris. Heulin *et al.* (1973) found a similar incidence (44.4%) in a series of 52 cases of severe calcific aortic valve stenosis. The mean age of our patients with angina was 58 years, as compared with 54.2 years in patients without angina. Basta *et al.* (1975) reported similar mean ages of 57 and 54 years of their patients with and without angina, respectively.

An interesting finding was that the aortic valve gradients tended to be smaller in those patients with angina pectoris than in those without, particularly when the angina pectoris was severe. This seemed to be because of a greater prevalence of coronary artery disease in those with milder aortic stenosis. Berndt *et al.* (1974) had similar experience showing that the mean aortic systolic gradient was lower in patients with coronary artery disease than in those without. This implies that angina in those patients

with significant coronary artery disease is more the result of the coronary disease than of the aortic stenosis. It is also possible that in some cases myocardial ischaemia and infarction resulting from coronary artery disease itself leads to a fall in the aortic valve gradient.

The reported incidence of coronary arterial obstruction in patients with combined aortic stenosis and angina is from just over 20 per cent to above 60 per cent (Linhart *et al.*, 1968; Berndt *et al.*, 1974; Parker, Jacob, and Seabra-Gomes, 1974; Basta *et al.*, 1975). The incidence in our series was 50 per cent (14 of 28 patients).

Basta *et al.* (1975) reported that all of 19 patients with aortic stenosis who did not have angina had normal coronary arteriograms. Others, however, have found a significant incidence of coronary arterial disease in this category of patients. Loop *et al.* (1972), in their series of patients with aortic or mitral valve disease, found abnormal coronary arteriograms in 28 per cent of cases who had no history of angina. More recently Harris *et al.* (1975) have found an incidence of 10.3 per cent in their series of patients with dominant aortic stenosis. Two of our patients who had no angina were subsequently found at necropsy to have severe coronary artery disease. We, therefore, agree with Bonchek *et al.* (1973) that coronary arteriography should be undertaken in all patients over the age of 40 in whom aortic valve replacement is being considered. This should not only provide guidance to the surgeon as to the need for coronary artery grafting in addition to the valve replacement but also should help in assessing the risk of operation.

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