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Coexistence of asymmetric septal hypertrophy and aortic valve disease in adults

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ABSTRACT Echocardiography detected asymmetric septal hypertrophy (ASH) in five of 200 adults being assessed for aortic valve surgery. Four of these were among 119 patients with dominant aortic stenosis, which was severe in three. ASH was confirmed at the time of aortic valve replacement in two of these patients; the third declined operation.

The finding of ASH in only one of 81 patients with free aortic reflux is consistent with chance association. While the same explanation could apply to the higher prevalence in those with aortic stenosis, it may be that a long-standing pressure overload can trigger inappropriate septal hypertrophy in predisposed individuals.

Brock (1957) recorded subaortic hypertrophy in association with hypertension and postulated its development in response to chronic left ventricular pressure load from aortic valvar stenosis. Hurst and Logue (1966) suggested, on the basis of operative and necropsy data, that it might be present in about 10% of those with aortic valve disease. Another necropsy study (Parker *et al*, 1969) yielded ten instances of subaortic hypertrophy in association with aortic valve stenosis.

In echocardiography we now have a sensitive method for detecting excessive (asymmetric) septal hypertrophy (ASH) during life (Henry *et al*, 1973). We used it to screen a series of patients undergoing investigation with a view to aortic valve replacement and report our findings with respect to dominance of pressure or volume load on the left ventricle.

Patients and methods

Echocardiography was carried out in 220 adults undergoing assessment for aortic valve replacement. Technically satisfactory records could not be obtained from 20 of them. The remaining 200 patients (74 women, 126 men) form the basis of this study. Their ages ranged from 18 to 67 years (mean 47). None gave a family history suggestive of cardiomyopathy.

One hundred and nineteen patients had dominant aortic stenosis. Their symptoms and some aetiological details are shown in table 1. Their peak systolic—left ventricular-systemic arterial pressure gradients, estimated echocardiographically (Bennett *et al*, 1975) in all, and measured at cardiac catheterisation in 55, ranged from 20 to 100 mmHg. Of the 118 with gradients higher than 40 mmHg 117 had pressures of a similar order recorded at operation. One, with a valvar gradient of 100 mmHg at rest, refused the offer of surgical help.

Table 1 *119 patients with dominant aortic stenosis*

Age	18–67 yr (mean 47)
Sex	
Men	72
Women	47
Aetiology	
Congenital	3
Calcific	112
Unknown	4
Symptoms	
Dyspnoea	110
Angina	82
Syncope or faintness	40
Congestive heart failure	4

Of the 81 patients who had free aortic reflux, of varied aetiology, which was thought to be the basis of their symptoms (table 2), 80 underwent aortic valve replacement. In one the investigations showed only moderately severe reflux, with coexisting ASH, and operation was not advised.

The echocardiograms were recorded with the patients supine, using a C11 transducer with an Ekoline 20 ultrasonoscope and a Cambridge photographic recorder. Echocardiograms showed both

Table 2 *81 patients with aortic reflux*

Age	28–66 yr (mean 46)
Sex	
Men	54
Women	27
Aetiology	
Rheumatic	67
Bacterial	6
Syphilitic	2
Aortic dissection	1
Rickettsial carditis	1
Unknown	4
Symptoms	
Dyspnoea	81
Angina	20
Syncope or faintness	12
Congestive heart failure	10

surfaces of the interventricular septum (IVS), the posterior basal left ventricular wall (PLVW), and the peak excursion of the mitral leaflets (fig 1). Diastolic IVS and PLVW thickness was measured at a point in the cardiac cycle immediately before atrial systole. The left ventricular internal diamensions in systole (Ds) and diastole (Dd) were also measured. Mean circumferential fibre shortening velocity (Vcf) was calculated by the method of Cooper *et al* (1972).

The chief echocardiographic criterion used for diagnosing ASH in this study was an IVS : PLVW thickness ratio in excess of 1·3 : 1 (Henry *et al*, 1973). Systolic anterior movement of the mitral

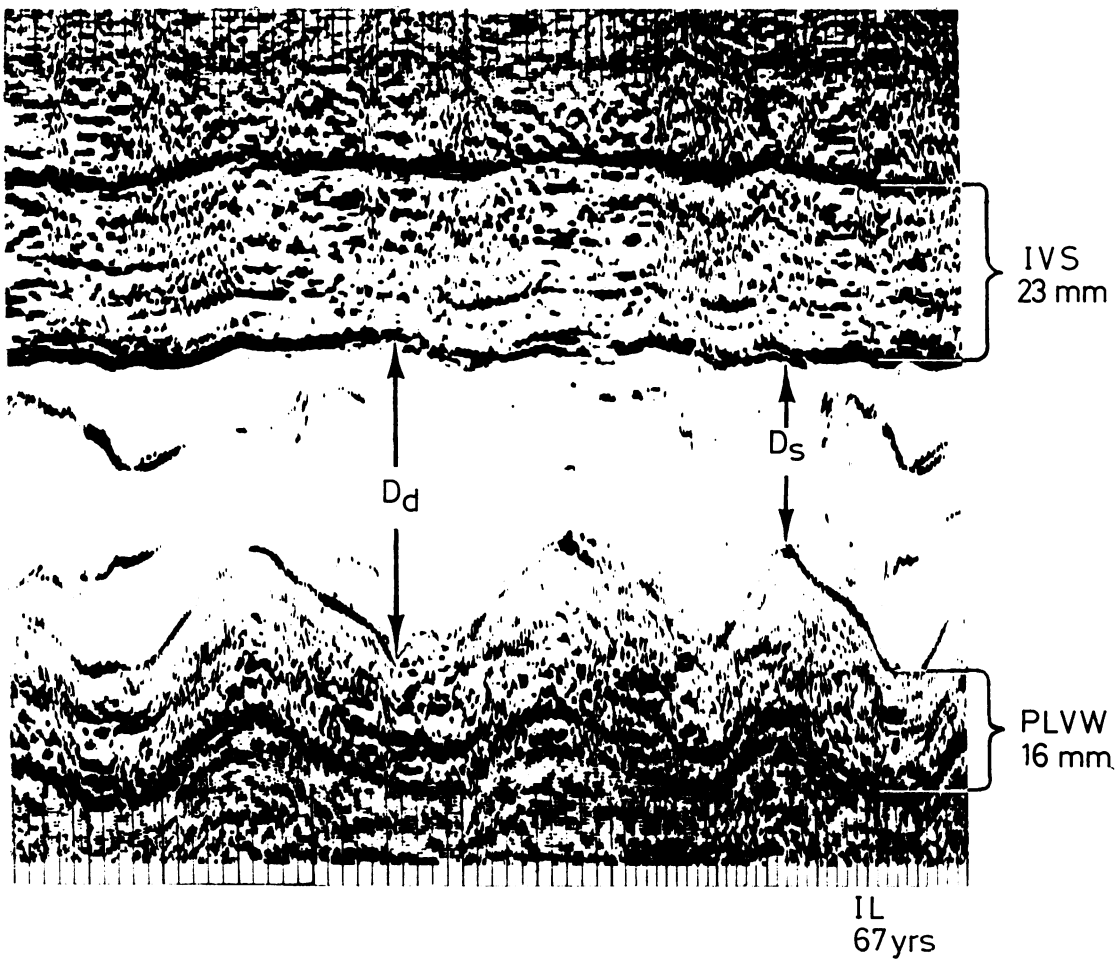

Fig 1 *Echocardiogram showing IVS and PLVW thickness and left ventricular internal dimensions at peak systole and end diastole. ASH is present in this case.*

valve (SAM), high Vcf, and small left ventricular cavity (Shah *et al*, 1969) were also sought.

Results

Table 3 shows the numbers, sex distribution, and echocardiographic findings in those with dominant aortic stenosis and table 4 in those with aortic reflux. Four patients with dominant stenosis (three women aged 63, 64, and 67 and one man aged 53) were found to have IVS : PLVW ratios higher than 1·3 : 1. All four exhibited typical SAM of the anterior mitral leaflet (fig 2), though in one only after inhalation of amyl nitrite. Retrograde left ventricular catheterisation showed systolic aortic valvar gradients of 20, 65, 75, and 100 mmHg, without subvalvar gradients, and showed ventriculographic features of hypertrophic cardiomyopathy in all four. The patient with the gradient of 100 mmHg declined operation. Excessive septal hypertrophy was confirmed at the time of aortic valve replacement in the other two with critical stenosis, and a myotomy was carried out in one.

Table 3 *Age and sex distribution and echocardiographic findings in subjects with aortic stenosis*

	IVS/PLVW ≤1·3		*Wall ratio >1·3*	
No	115		4	
Men	71		1	
Women	44		3	
	Range	*Mean*	*Range*	*Mean*
Age (yr)	18–66	47	52–67	62
IVS (mm)	10–22	15	17–31	22
PLVW (mm)	10–20	15	12–16	14
IVS/PLVW	0·7–1·3	1·0	1·4–2·0	1·7
Ds (mm)	16–47	28	15–25	20
Dd (mm)	30–60	46	30–41	35
Vcf (sec^{-1})	1·0–2·0	1·5	1·2–2·0	1·7
SAM	Absent		Present	

Table 4 *Age and sex distribution and echocardiographic findings in subjects with aortic reflux*

	IVS/PLVW ≤1·3		*Wall ratio >1·3*
No	80		1
Men	54		0
Women	26		1
	Range	*Mean*	
Age	28–66	46	62
IVS (mm)	10–18	13	23
PLVW (mm)	10–16	13	15
IVS/PLVW	0·8–1·3	1·0	1·5
Ds (mm)	26–70	42	24
Dd (mm)	55–90	63	41
Vcf (sec^{-1})	0·6–1·8	1·4	1·6
SAM	Absent		Present

One woman (aged 63) with aortic reflux was found to have an IVS : PLVW ratio in excess of 1·3 : 1. Her echocardiogram also showed typical SAM and cardioangiography confirmed asymmetric hypertrophy while suggesting that her aortic reflux was not severe enough to require aortic valve replacement.

All 195 patients without echocardiographic evidence of ASH were operated on, and no evidence of unusual septal hypertrophy was noted.

Discussion

Nanda *et al* (1974) pioneered the use of echocardiography for detecting idiopathic subaortic stenosis coexisting with aortic valve disease. We used the technique in an attempt to establish the prevalence of excessive asymmetric septal hypertrophy in a series of patients undergoing assessment with a view to aortic valve replacement. Since Henry *et al* (1973) had suggested that in adults without right ventricular hypertension an IVS : PLVW ratio in excess of 1·3 offers a sufficient basis for the diagnosis, we used this criterion in our study. Popp (1976) subsequently suggested that a ratio in excess of 1·5 : 1 is necessary. Had we used that criterion, we would have failed to identify two examples of ASH (subsequently confirmed by ventriculography and at operation) in association with disease of the aortic valve. In a still more recent study (Chahine *et al*, 1977), which pointed the lack of any pathognomonic feature, three patients finally considered to have hypertrophic cardiomyopathy had echocardiographic IVS : PLVW ratios between 1·0 and 1·2.

Only three of the patients in this study had coexistent ASH and disease of the aortic valve severe enough to require operation. On this evidence, the prevalence of ASH in adults with critical aortic stenosis may be of the order of 2·5%. The figure rises to 3·4% if the patient with a peak systolic gradient of 20 mmHg (and resting left ventricular pressure circa 160 mmHg) is included. Others have noted coexistence of ASH with aortic stenosis, and with systemic hypertension and coarctation of the aorta (Moreyra *et al*, 1970; Block *et al*, 1973; Feizi and Emanuel, 1975). The possibility exists that frequent or sustained excessive pressure loads may trigger inappropriate (asymmetric) ventricular hypertrophy in predisposed individuals. Alternatively, the association may be coincidental; however, in our series none of the four patients with ASH gave a family history suggestive of cardiomyopathy.

Among the 81 patients with aortic reflux, only one was found to have coexisting ASH. This

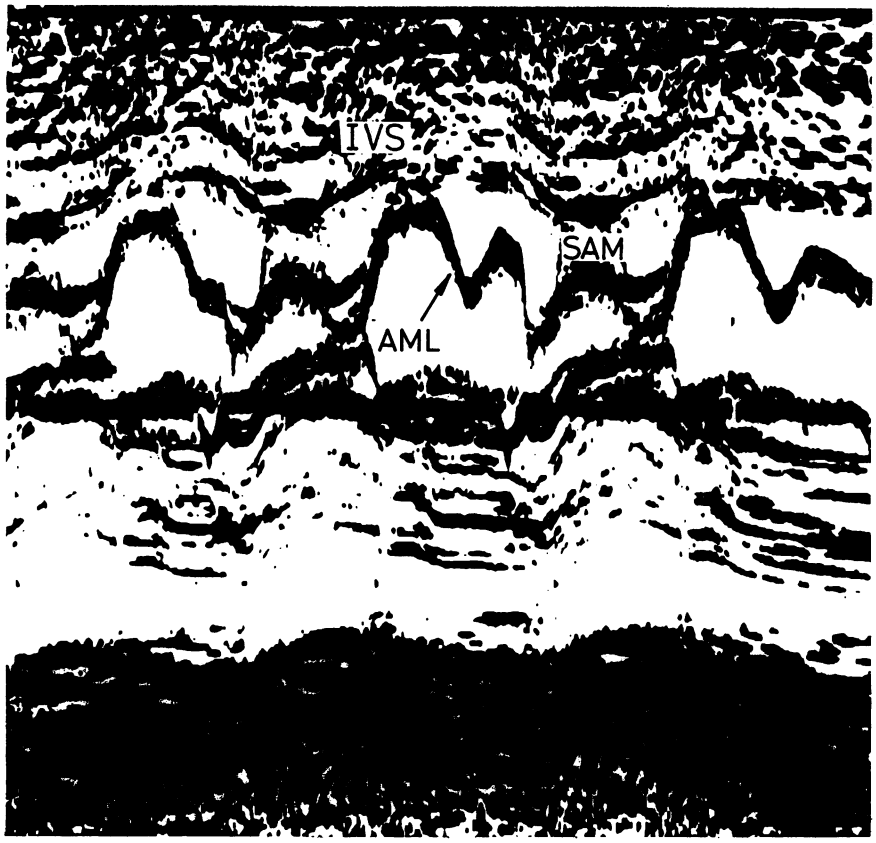

Fig. 2 *Echocardiogram of patient with moderate aortic reflux, illustrating SAM of the anterior mitral leaflet (AML).*

association was probably coincidental, although ASH has been detected in association with other volume-overload conditions such as thyrotoxic heart disease, persistent ductus arteriosus, and ventricular septal defect (Symons *et al*, 1974; Feizi and Emanuel, 1975; Larter *et al*, 1976).

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