Physical Signs in Differential Diagnosis of Left Ventricular Obstructive Cardiomyopathy

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The diagnosis of left ventricular obstructive cardiomyopathy can be made in most instances by simple clinical examination, as practised by Paul Wood as early as 1960 (personal communication). Occasionally, there is difficulty in differentiating other causes of a systolic murmur associated with left ventricular disease. We have, therefore, assessed the diagnostic value of palpation of the carotid pulse and indirect carotid pulse recording, palpation of the praecordium and displacement recording, and auscultation and phonocardiography, in left ventricular obstructive cardiomyopathy with and without additional mitral regurgitation, in aortic valve and fixed subvalve stenosis, and in pure mitral regurgitation.

SUBJECTS AND METHODS

Five groups of patients were included in this retrospective study, and in each patient cardiac catheterization and angiocardiology, phonocardiography, and apex displacement records had been made. In a few patients the quality of the angiographic study or recordings was inadequate for inclusion in the study. No patient was in heart failure at the time of investigation.

Group 1: Left Ventricular Obstructive Cardiomyopathy. There were 20 patients, 14 male and 6 female, whose ages ranged from 6 to 49 years (mean 32 years). The diagnosis was based on the typical appearance of the left ventricular angiogram (Braunwald *et al.*, 1960; Steiner, 1964; Simon, Ross, and Gault, 1967) together with haemodynamic evidence of left ventricular outflow obstruction either at rest or after provocation (Braunwald and Ebert, 1962). The resting left ventricular outflow gradient ranged from 5 to 90 mm. Hg, and most were over 50 mm. Hg, and in one patient provocative measures produced a gradient of 175 mm. Hg. Six patients had additional mitral regurgitation demonstrated angiographically, and they were analysed separately from the

14 patients without angiographic evidence of mitral regurgitation.

Group 2: Aortic Valve Stenosis. There were 40 patients, 32 male and 8 female, whose ages ranged from 10 to 64 years (mean 43 years). The diagnosis was established by left heart catheterization and angiography and confirmed by operation in 22 patients and at necropsy in 5 patients. The systolic gradient across the aortic valve ranged from 35 to 100 mm. Hg in 17 patients, and was over 100 mm. Hg in 12 patients. In 11 patients the gradient was unknown because of failure to cross the aortic valve by the retrograde arterial route, but severe aortic valve stenosis was confirmed in each at the time of operation. Patients with moderate or severe aortic regurgitation were excluded, but 27 patients had minimal aortic regurgitation demonstrated by aortic root angiogram. Trivial mitral valve disease was present in 5 patients and severe mitral regurgitation in 1 patient.

Group 3: Fixed Aortic Subvalve Stenosis. There were 7 patients, 6 male and 1 female, whose ages ranged from 8 to 29 years (mean 16 years). The diagnosis was established by left ventricular angiogram, and confirmed by operation in 6 patients. The systolic gradients (across the area of fixed subvalve stenosis) ranged from 25 to 150 mm. Hg (mean 55 mm. Hg).

Group 4: Mitral Regurgitation. There were 20 patients, 13 male and 7 female, whose ages ranged from 5 to 59 years (mean 39) with uncomplicated moderate or severe mitral regurgitation. The diagnosis was established by left heart catheterization and left ventricular cine-angiogram (Rees, Jefferson, and Harris, 1965), and confirmed by operation in 5 patients and at necropsy in 1 patient. Patients with additional mitral stenosis or aortic valve disease were excluded. The rhythm was atrial fibrillation in 3 patients.

A five-channel photographic recorder, operated at a paper speed of 100 mm. a second, was used to record the phonocardiograms simultaneously from 2 sites (including aortic area, pulmonary area, tricuspid area, and mitral area) together with lead II of the electrocardiogram, respiration and indirect carotid pulse or apex

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TABLE I
CAROTID PULSE

	Left ventricular obstructive cardiomyopathy		Aortic stenosis		Pure mitral regurgitation
	Without mitral regurgitation	With mitral regurgitation	Valve	Fixed subvalve	
Cases	14	6	40	7	20
<i>Upstroke time</i> (in sec., corrected for rate)					
0.04	1				
0.05	1				
0.06					1
0.07					1
0.08	2			1	
0.09	2	3			1
0.10	1	2			1
0.11	2				2
0.12	3				1
0.13			3	1	2
0.14	1	1	2		2
0.15					3
0.16					
0.17				1	3
0.18	1				2
0.19			3		
0.20			2		1
0.21				1	
0.22			1		
0.23			4		
0.24			4		
0.25			1	1	
0.26					
0.27			3	1	
0.28			3		
0.29			2	1	
0.30			4		
0.31			2		
0.32			3		
0.33			1		
0.34			1		
0.35			1		
Mean	0.10 sec.	0.10 sec.	0.26 sec.	0.20 sec.	0.14 sec.
<i>Ejection time</i> (in sec., corrected for rate)					
0.21					1
0.22					1
0.23					1
0.24	1				2
0.25	1				
0.26					3
0.27					
0.28	1				3
0.29	1				1
0.30	2			1	5
0.31	1	2	1		1
0.32	1	1	3	1	2
0.33	2		3		
0.34	1		3	1	
0.35	1	1	3		
0.36			6	2	
0.37	1	2	5	1	
0.38	1		4		
0.39			1		
0.40			4	1	
0.41					
0.42			4		
0.43			3		
Mean	0.31 sec.	0.34 sec.	0.38 sec.	0.35 sec.	0.28 sec.
Carotid shudder	0	0	35	4	0

displacement. The phonocardiograms were taken with attenuation of the low frequencies to approximate to clinical auscultation, and the galvanometers responded uniformly from 30 to 800 cycles a second (Leatham, 1952). The indirect carotid pulse was recorded by means of an air-filled cuff and a linear manometer and amplifying system (Robinson, 1963). Apex displacement was recorded with a hand-applied crystal transducer in the area of the apex manifesting maximal movement. All recordings were made with the patient reclining at 30°–40° and during continuous respiration.

The ejection time was measured from the onset of the indirect carotid pulse to the dicrotic notch, and was divided by the square root of the preceding R–R interval of the electrocardiogram in order to correct for variations in heart rate; from 0.26 to 0.34 sec. was taken as the normal range. The upstroke time of the carotid pulse was measured from the onset of the indirect carotid pulse to the summit and was similarly corrected for heart rate; from 0.09 to 0.26 sec. was taken as "normal". The "normal" range was obtained from 75 healthy children and young adults and 164 patients with a wide variety of cardiovascular disorders other than aortic stenosis, including congenital, rheumatic, ischaemic, and hypertensive heart disease (Robinson, 1963).

The apical displacement recordings were analysed for the height of the atrial and rapid filling waves in relation to the ventricular, and the presence or absence of a bifid ventricular impulse.

The phonocardiograms were analysed for splitting of the second heart sound in the pulmonary area and the relative intensity of A_2 and P_2 (Harris and Sutton, 1968), the presence or absence of ejection, mid or late systolic sounds, and the presence of opening snaps, rapid filling (3rd), and atrial sounds. The intensity of the first heart sound could only be assessed subjectively by auscultation since accurate calibrations of phonocardiograms are impracticable.

RESULTS

Carotid Pulse (Table I)

Group 1: Left Ventricular Obstructive Cardiomyopathy (20 patients). The carotid pulse felt sharp and the upstroke time of the indirect carotid pulse recording was abnormally short in 4 patients and in the lower range of normal (0.14 sec. or less) in all but 1 of the remainder (Fig. 1). The ejection time was abnormally short in 2 patients, normal in 12 patients, and abnormally long in 6 patients, but correlation with resting gradients was poor. There were no differences when additional mitral regurgitation was present.

Group 2: Aortic Valve Disease (40 patients). The carotid pulse felt slow rising and sustained, and the upstroke time of the indirect carotid pulse was abnormally long in 20 patients and in the upper range of normal (0.15 sec. or more) in the remaining 20 patients. The ejection time was abnormally long

FIG. 1.—Left ventricular obstructive cardiomyopathy, showing the systolic murmur maximal after midsystole and finishing before the second heart sound. The external carotid pulse shows a very sharp upstroke to the peak ejection point. The time intervals in this and subsequent records are 0.20 and 0.04 sec. PA, pulmonary area; HF, high frequency; 1, first heart sound; 2, second heart sound; Car., external carotid pulse; Resp., respiratory trace; II, lead II of electrocardiogram.

in 30 patients and in the upper range of normal (0.31 sec. or more) in the remaining 10 patients. A carotid shudder was felt and recorded in 35 patients.

Group 3: Fixed Aortic Subvalve Stenosis (7 patients). In fixed aortic subvalve stenosis, the carotid pulse felt slow rising and sustained in all but 1 young patient in whom it felt sharp though sustained. The upstroke time of the indirect carotid pulse was abnormally short in 1 patient, normal in 4 patients, and abnormally long in 2 patients. The ejection time was abnormally long in 4 patients and in the upper range of normal (0.30 sec. or more) in the remaining 3 patients. A carotid shudder was felt and recorded in 4 patients.

Group 4: Mitral Regurgitation (20 patients). In pure mitral regurgitation, the carotid pulse felt sharp, but its small volume made it less obvious than in left ventricular obstructive cardiomyopathy. The upstroke time of the indirect carotid pulse was abnormally short in 2 patients and in the lower range of normal (0.20 sec. or less) in the remaining 18 patients (Fig. 2). The ejection time was abnormally short in 5 patients and normal in 15 patients.

Apex Displacement (Table II)

Group 1: Left Ventricular Obstructive Cardiomyopathy (20 patients). Technically satisfactory apex

FIG. 2.—Mitral regurgitation, showing a pansystolic murmur enclosing the aortic component of the second heart sound in the mitral area. The external carotid pulse shows a sharp upstroke to the peak ejection point. MA, mitral area; Insp., inspiration; 1, 2, 3, first, second, and third heart sounds.

recordings were obtained in 16 of the 19 patients in sinus rhythm with left ventricular obstructive cardiomyopathy. A double apex impulse due to an abnormal "a" wave (more than 10% of the systolic outward movement) was recorded and felt in 14 patients. A secondary systolic peak was recorded in 9 patients without mitral regurgitation (Fig. 3) and in 2 patients with mitral regurgitation. In addition, an abnormal systolic outward movement was felt and recorded in the left parasternal area in 3 patients.

Group 2: Aortic Valve Stenosis (40 patients). Technically satisfactory apex recordings were obtained in 24 patients in sinus rhythm with aortic valve stenosis. A double apex impulse due to an abnormal "a" wave was felt and recorded in 21 patients. A secondary systolic peak was recorded

in 10 patients (Fig. 4). A systolic outward movement in the left parasternal area was never felt.

Group 3: Fixed Aortic Subvalve Stenosis (7 patients). Technically satisfactory apex recordings were obtained in 4 patients in sinus rhythm with fixed aortic subvalve stenosis. A double apex impulse due to an abnormal "a" wave was felt and recorded in all 4 patients. A secondary systolic peak was recorded in 3 of the 4 patients. A systolic outward movement in the left parasternal area was never felt.

Group 4: Pure Mitral Regurgitation (20 patients). Technically satisfactory apex recordings were obtained in 13 patients (and the rhythm in 3 patients was atrial fibrillation). A double apex impulse, due to an abnormal "a" wave, was felt and recorded

TABLE II
APEX DISPLACEMENT

	Left ventricular obstructive cardiomyopathy		Aortic stenosis		Pure mitral regurgitation
	Without mitral regurgitation	With mitral regurgitation	Valve	Fixed subvalve	
Cases in sinus rhythm	11	5	24	4	10
A wave (height of "a" wave as % of systolic outward movement)					
0 to 10% (normal)	2	0	3	0	3
11 to 25%	4	2	9	3	3
26 to 50%	5	2	9	1	3
51 to 75%	0	0	2	0	0
76 to 100%	0	1	1	0	1
Mean (%)	30	38	30	23	32
Secondary systolic peak	9	2	10	3	7
Prominent rapid filling wave	8	2	17	4	11

in 7 of the 10 patients in sinus rhythm. A secondary systolic peak was recorded in 7 of the 13 patients. A systolic outward movement in the left parasternal area was felt in 9 patients.

Heart Sounds (Table III)

Group 1: Left Ventricular Obstructive Cardiomyopathy (20 patients). In left ventricular obstructive cardiomyopathy, the intensity of the first heart sound (S_1) was normal in 10 patients, increased in 9 patients, and reduced in only 1 patient (P-R interval 0.22 sec.).

The second heart sound (S_2) showed normal splitting in 10 patients, was single in all phases of respiration in 3 patients, and showed reversed splitting (pulmonary component P_2 before the aortic component A_2) in 4 of the 6 patients, with resting left ventricular outflow gradients of 50 to 90 mm. Hg. An abnormally widely split S_2 in expiration (A_2 - P_2 interval more than 0.03 sec.) was present in 3 patients, 1 of whom had a right ventricular outflow gradient of 7 mm. Hg, and 1 a prolonged Q-RV upstroke time. Of the 17 patients with both A_2 and P_2 recorded, the intensity of A_2 was always greater than, or equal to, the intensity of P_2 , and in 4 patients the intensity of A_2 was more than twice the intensity of P_2 . In 3 patients, all with pulmonary arterial pressures above 50 mm. Hg, P_2 was heard and recorded in the mitral area. An aortic ejection sound was never heard or recorded.

Group 2: Aortic Valve Stenosis (40 patients). In aortic valve stenosis the intensity of S_1 seemed normal in 19 patients and reduced in 21 patients.

S_2 was single and the intensity greatly reduced in

FIG. 3.—Left ventricular obstructive cardiomyopathy apical motion (A.C.G.), showing a small "A" wave and double outward systolic movement over the left ventricle. The termination of the systolic murmur just before the second heart sound is particularly well seen. S_1 , S_2 , first and second heart sounds; R.F.W., rapid filling wave.

21 patients (Fig. 5), split physiologically in 16 patients, and reversed in 2 patients. In 1 patient, S_2 was abnormally widely split in expiration, A_2 before P_2 , and this patient had in addition severe mitral regurgitation and pulmonary hypertension. Of the 19 patients with both A_2 and P_2 recorded, the intensity of A_2 was greater than or equal to the intensity of P_2 in 17, and in 4 of these patients, all young and with non-calcified aortic valves, the intensity of A_2 was more than twice the intensity of P_2 . The intensity of A_2 was less than P_2 in only 2 patients. P_2 was never heard or recorded in the mitral area.

An aortic ejection sound was heard and recorded

FIG. 4.—Aortic valve stenosis (non-calcific). The first heart sound (1) is soft and the second heart sound (2) is of normal intensity. The systolic murmur has an earlier crescendo than in many cases of left ventricular obstructive cardiomyopathy, and finishes well before the second heart sound and is preceded by an ejection sound (E). A recording of apical movement shows a small "A" wave (at) and a double outward systolic movement.

TABLE III
HEART SOUNDS

	Left ventricular obstructive cardiomyopathy		Aortic stenosis		Pure mitral regurgitation
	Without mitral regurgitation	With mitral regurgitation	Valve	Fixed subvalve	
Cases	14	6	40	7	20
<i>S₁</i> —Intensity					
Reduced	1	0	21	4	4
Normal	6	4	19	3	4
Increased	7	2	0	0	12
<i>S₂</i> —Splitting					
Physiological	5	5	16	5	15
Wide in expiration	3	0	1	0	3
Fixed	0	0	0	0	2
Reversed	3	1	2	1	0
Single	3	0	21	1	0
<i>P₂</i> —in mitral area	3	0	0	0	9
<i>S₂</i> Single	3	0	21	1	0
Split <i>A₂</i> > <i>P₂</i>	11	6	17	5	7
Split <i>A₂</i> < <i>P₂</i>	0	0	2	1	13
Aortic ejection sound	0	0	18	1	0
Mid- or late systolic click	0	0	0	0	1
Opening snap	0	0	0	0	3
Cases over 40 years	5	2	27	0	10
<i>S₃</i> present	2	0	13	0	8
Atrial sound	11	5	24	0	7

in 18 patients and was absent in the remaining 22 who, with only 1 exception, had calcification of the valve on fluoroscopy.

Group 3: Fixed Aortic Subvalve Stenosis (7 patients). In fixed aortic subvalve stenosis, the intensity of *S₁* was normal in 3 patients and reduced in 4 patients.

S₂ was single in 1 patient, split physiologically in

5 patients, and reversed in 1 patient. The intensity of *A₂* appeared to be normal or reduced. Among the 6 patients with both *A₂* and *P₂* recorded, the intensity of *A₂* was greater than or equal to the intensity of *P₂* in 5 patients and less than *P₂* in 2 patients.

P₂ was never heard or recorded in the mitral area.

An aortic ejection sound was heard and recorded in 1 patient with fixed aortic subvalve stenosis.

FIG. 5.—Aortic valve stenosis (calcified). The first and second heart sounds are greatly diminished. The external carotid pulse is extremely slow rising with a shudder.

TABLE IV
HEART MURMURS

	Left ventricular obstructive cardiomyopathy		Aortic stenosis		Pure mitral regurgitation
	Without mitral regurgitation	With mitral regurgitation	Valve	Fixed subvalve	
Cases	14	6	40	7	20
Systolic murmur					
Midsystolic	14	6	40	7	0
Pansystolic	0	0	0	0	18
Late systolic	0	0	0	0	2
Peak of ejection murmur (% of distance from S ₁ to S ₂)					
30%	0	0	0	1	x
35%	0	0	1	0	x
40%	0	0	0	2	x
45%	0	0	3	2	x
Midsystole 50%	1	0	13	1	x
55%	5	1	8	1	x
60%	3	3	10	0	x
65%	1	0	3	0	x
70%	2	1	1	0	x
75%	0	0	1	0	x
80%	1	1	0	0	x
Mean (%)	62	65	55	44	x
Diastolic murmur					
Early diastolic	0	0	20	5	1
Mid diastolic	0	0	0	0	7

Group 4: Pure Mitral Regurgitation (20 patients). In pure mitral regurgitation, the intensity of S₁ was increased in 12 patients, normal in 4 patients, and reduced in 4 patients.

Splitting of S₂ was physiological in 15 patients, abnormally widely split in expiration (more than 0.03 sec.) in 3 patients, and fixed in 2 patients. The intensity of A₂ was greater than or equal to the intensity of P₂ in 7 patients, and less than P₂ in 13 patients (all patients had pulmonary hypertension). P₂ was heard and recorded in the mitral area in 9 patients.

An aortic ejection sound was never heard or recorded.

Third Heart Sound (Table III)

An audible third heart sound could only be considered abnormal over the age of 40 years; it was heard and recorded, and frequently palpated, in 2 of 5 patients with left ventricular obstructive cardiomyopathy, 13 of 27 patients with aortic valve stenosis, and 8 of 10 patients with pure mitral regurgitation, all over the age of 40.

Atrial Sound (Table III)

Of the patients in sinus rhythm, atrial sounds were heard and recorded in 16 of 19 patients with left ventricular obstructive cardiomyopathy, in 24 of 40 patients with aortic valve stenosis, and in 7 of the 14 patients with pure mitral regurgitation.

Heart Murmurs (Table IV)

Group 1: Left Ventricular Obstructive Cardiomyopathy (20 patients). Frequently the systolic murmur in left ventricular obstructive cardiomyopathy was difficult to classify by auscultation as either ejection or regurgitant. Phonocardiography showed either a midsystolic murmur finishing before S₂, or a longer murmur, with reduction of intensity before S₂ and crescendo-decrescendo or diamond in shape, so that we have classified both as ejection murmurs (Fig. 1). There was no difference between the patients with additional mitral regurgitation and those without mitral regurgitation. In 1 patient, only a faint systolic murmur was heard and could not be recorded, and in the remaining 19 patients, the maximal intensity of the murmur occurred 50 to 80 per cent of the way from S₁ to S₂. In 1 patient, intracardiac phonocardiography localized the murmur to the outflow tract of the left ventricle, and in another patient, intracardiac phonocardiography localized 2 murmurs, 1 to the left ventricular outflow tract and 1 to the right ventricular outflow tract. Early diastolic murmurs were never heard or recorded.

Group 2: Aortic Valve Stenosis (40 patients). In aortic valve stenosis, a midsystolic murmur was heard and recorded in all 40 patients, and the maximal intensity occurred 35 to 50 per cent of the way from S₁ to S₂ in 17 patients, but was identical

in timing with the murmur of left ventricular obstructive cardiomyopathy in the remaining 23 patients. An early diastolic murmur due to aortic incompetence was heard and recorded in 20 patients.

Group 3: Fixed Aortic Subvalve Stenosis (7 patients). In fixed aortic subvalve stenosis, a mid-systolic murmur was heard and recorded in all 7 patients, and the maximal intensity occurred 30 to 55 per cent of the way from S_1 to S_2 in all patients. An early diastolic murmur was heard and recorded in 5 patients.

Group 4: Pure Mitral Regurgitation (20 patients). In pure mitral regurgitation, pansystolic murmurs were heard and recorded in 18 patients, and late systolic murmurs in the remaining 2 patients, and in 1 the murmur was preceded by a click. The murmur of mitral regurgitation invariably reached and absorbed A_2 at the mitral area. An early diastolic murmur was heard and recorded in 1 patient with pulmonary incompetence due to pulmonary hypertension, and a short mid-diastolic murmur was heard and recorded in 7 patients and was preceded by an opening snap in 3 patients.

DISCUSSION

The single most important physical sign in differentiating left ventricular obstructive cardiomyopathy from other causes of a systolic murmur with left ventricular disease is the sharp rising carotid pulse, as emphasized long ago by Paul Wood, and this can be confirmed objectively using external carotid pulse recordings made by an undistorting system (Duchosal *et al.*, 1956; Robinson, 1963). Thus the upstroke time was 0.14 sec. or less in all but 1 of the patients with left ventricular obstructive cardiomyopathy overlapping with the lower range of normality, but in only 5 of the 47 patients with fixed aortic stenosis. The cause of the sharp upstroke in left ventricular obstructive cardiomyopathy is the unobstructed transmission of the rapidly rising left ventricular pressure pulse (Brockenbrough, Braunwald, and Morrow, 1961), for the obstruction does not begin until mid or late systole; it is not caused by the addition of mitral regurgitation, for the pulse is just as sharp when mitral regurgitation is absent. The upstroke time of the carotid pulse is nearly as short in pure mitral regurgitation, but it is much less remarkable clinically because its amplitude is less; this difference cannot be quantitated by an indirect carotid recording system. The left ventricular ejection time in left ventricular obstructive cardiomyopathy, as measured by the indirect carotid pulse, is usually

within the range of normal, and is variable without relation to the degree of obstruction or the presence of additional mitral regurgitation; nevertheless, the measurement of ejection time is useful since it is prolonged in most patients with fixed aortic stenosis; and abnormally short in some of the patients with mitral regurgitation fitting in with the early A_2 causing wide splitting of the second sound (Brigden and Leatham, 1953). The carotid pulse is no real help in differentiating aortic valve from fixed subvalve stenosis, nor is a double systolic wave specific to left ventricular obstructive cardiomyopathy (Snellen, 1964; Wigle, 1964), since it also occurs in fixed stenosis and indeed in normal subjects. Systolic vibrations of sufficient intensity to be recorded on the carotid pulse tracing or to be seen as a shudder in the neck (Evans and Lewes, 1945) are only found in fixed aortic valve disease.

Abnormal apical systolic movement can be palpated in the majority of these patients with left ventricular disease. The displacement recordings cannot be quantitated but show a systolic wave of abnormal pattern with about equal frequency in the four conditions with left ventricular disease. When a double apical impulse is palpated it is usually caused by an abnormally large "a" wave which is found to exceed 10 per cent of the systolic wave on the recording (of apical displacement) in about 85 per cent of cases, and is just as common in fixed aortic valve disease and mitral regurgitation as in left ventricular obstructive cardiomyopathy. No recordings were made of parasternal movement, but abnormal movement to palpation in patients with obstruction to left ventricular outflow seems to indicate obstruction to right ventricular outflow. In patients with pure mitral regurgitation parasternal movement is frequent and due to systolic expansion of the left atrium or to right ventricular hypertrophy.

Observations on the intensity of S_1 can only be subjective, but a striking reduction in intensity is found in about one-half of patients with fixed aortic stenosis. There was no correlation with the P-R interval or rate of rise of the left ventricular pressure pulse. The reduction in intensity is more frequent in the older age-groups in whom calcification of the aortic valve is more common and might have spread on to the anterior cusp of the mitral valve, but this would not explain the soft first sound found in 4 of the 7 patients with fixed subvalve stenosis. Splitting of the second heart sound is usually physiological in left ventricular obstructive cardiomyopathy, whether there is additional mitral regurgitation or not, but with severe obstruction it tends to be reversed from prolongation of left ventricular systole. Splitting is reversed or absent in about

half the patients with aortic valve stenosis, reflecting the greater duration of systole (and reduction in intensity of A_2). In the patients studied, abnormally wide splitting (A_2 before P_2) was caused by shortening of left ventricular systole from pure mitral regurgitation or prolongation of right ventricular systole from septal obstruction, and there was never very wide splitting of S_2 , indicating that there was no great discrepancy between the duration of left and right ventricular systole as found with complete right or left bundle-branch block. A comparison of intensity of A_2 and P_2 is seldom of value in left ventricular obstructive cardiomyopathy or aortic stenosis, owing to variations in both components; in pure mitral regurgitation P_2 greater in intensity than A_2 in the pulmonary area (or transmitted to the mitral area) indicates pulmonary hypertension.

Evaluation of the other sounds is of little diagnostic value, except that an aortic ejection sound, when present in these circumstances, is almost diagnostic of aortic valve stenosis, and an opening snap indicates mitral regurgitation which is organic rather than secondary to left ventricular obstructive cardiomyopathy. Third heart sounds and atrial sounds are frequent in all four conditions.

The systolic murmur in left ventricular obstructive cardiomyopathy always shows a crescendo around mid-systole and finishes before A_2 , and is thought to be due to the rapid ejection of blood through the stenosing outflow tract; it is no different with additional mitral regurgitation. In nearly half the patients with left ventricular obstructive cardiomyopathy the crescendo of the systolic murmur is a little later than in aortic valve stenosis, reflecting the absence of obstruction from left ventricular obstructive cardiomyopathy in early systole. This "shift to the right" is more obvious clinically than on the phonocardiogram and sometimes causes the misdiagnosis of a pansystolic murmur. Indeed we wondered if the phonocardiograph was failing to record high frequency vibrations in left ventricular obstructive cardiomyopathy, but a repeat of the recordings using a single beam cathode ray tube with unlimited high frequency response again showed cessation of the murmur before A_2 , an invariable distinction from mitral valve regurgitation when the pansystolic murmur reaches and engulfs A_2 .

The finding of an aortic diastolic murmur indicates aortic valve or fixed infundibular stenosis rather than left ventricular obstructive cardiomyopathy. A diastolic murmur in the rapid ventricular filling phase (mitral diastolic murmur), however, is found with both left ventricular obstructive cardiomyopathy and isolated mitral regurgitation.

In conclusion, any difficulty in differentiating left ventricular obstructive cardiomyopathy and aortic valve or fixed infundibular stenosis can be resolved simply by palpating and recording the carotid pulse. There may be greater difficulty in differentiating obstructive cardiomyopathy from primary mitral regurgitation, and a sound recording may be required to distinguish the delayed ejection murmur of left ventricular obstructive cardiomyopathy from a pansystolic murmur.

SUMMARY

The physical signs, indirect carotid pulse tracings, and phonocardiograms have been analysed in 87 investigated patients with left ventricular disease and a systolic murmur, in order to clarify clinical diagnosis. There were 20 patients with left ventricular obstructive cardiomyopathy, 40 with aortic valve stenosis, 7 with fixed subvalve stenosis, and 20 with pure mitral regurgitation.

The sharply rising carotid pulse of left ventricular obstructive cardiomyopathy was confirmed by measurement of indirect carotid pulse recordings which showed a clear difference from 44 of the 47 cases with fixed aortic stenosis, but not from those with pure mitral regurgitation. Measurement of ejection time was of little value except for its prolongation in most patients with fixed aortic stenosis, and its normality or shortening in pure mitral regurgitation.

Abnormally large "a" waves on the apex displacement recording were found equally commonly in each of the 4 groups with left ventricular hypertrophy.

Reduction in intensity of the first heart sound was found in about half the patients with fixed aortic stenosis, but not in the other groups. The second heart sound was not of great diagnostic help since reversed splitting or fusion of A_2 and P_2 , indicating prolongation of left ventricular systole, was common in aortic valve stenosis and frequent in left ventricular obstructive cardiomyopathy. Abnormally wide splitting with a normal sequence of A_2 preceding P_2 suggested shortening of left ventricular systole from pure mitral regurgitation or lengthening of right from the septal obstruction of left ventricular obstructive cardiomyopathy.

While the systolic murmur of fixed aortic stenosis and of pure mitral regurgitation could always be clearly recognized as midsystolic and pansystolic respectively, the murmur of left ventricular obstructive cardiomyopathy was more difficult to classify clinically into one or other of these two patterns; but on the phonocardiogram it invariably finished before aortic closure, was not altered

by additional functional mitral regurgitation, and was thought to be always due to ejection through the hypertrophied outflow tract.

One of the objects of this paper is to emphasize the contribution made by Paul Wood to the clinical diagnosis of left ventricular obstructive cardiomyopathy, particularly in recognizing the sharp arterial pulse. We would also like to thank our technicians.

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