Diagnostic value of configuration of left ventricular outflow pressure gradient in differentiating hypertrophic obstructive cardiomyopathy from discrete types of aortic stenosis

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SUMMARY Since obstruction to left ventricular (LV) outflow in hypertrophic obstructive cardiomyopathy (HOCM) is dynamic, whereas that in discrete aortic stenosis is fixed, the shape of the transaortic pressure gradient should be characteristically altered in HOCM and thereby provide a means of differentiating these two disparate types of aortic stenosis. In all 22 HOCM patients, peak arterial pulse always occurred in the initial half of the systolic ejection period, while in 37 of 40 patients with discrete types of aortic stenosis it appeared in the final half. Peak left ventricular pressure was delayed significantly (P < 0.01) in HOCM compared with fixed aortic stenosis. Furthermore, the interval from onset of ejection to peak left ventricular pressure divided by ejection period was increased in HOCM, 0.59 compared with 0.49 (P < 0.01) in fixed aortic stenosis. The early arterial and late left ventricular pressure pulse peaks in HOCM are related to absence of obstruction to ejection early in systole. Moreover, the ratio of mean pressure gradient during the first half of ejection to that of the last half averaged 0.59 in HOCM. In contrast, this ratio averaged 1.24 in 23 patients with valvular aortic stenosis, 1.13 in 12 patients with discrete subvalvular stenosis, and 1.85 in 5 patients with supravalvular stenosis. This ratio allowed complete separation of HOCM from fixed aortic stenosis; < 0.80 identified HOCM while greater indicated fixed aortic stenosis. Whether brachial arterial pulse, appropriately adjusted for time, or central aortic pressure pulse was used did not alter these results.

The pathogenesis of obstruction to aortic outflow in hypertrophic obstructive cardiomyopathy (HOCM) is fundamentally different from that in patients with discrete fixed narrowing at the valvular, supra-valvular, or subvalvular level (Ross et al., 1966; Reis et al., 1974; Mason et al., 1975). In patients with discrete forms of aortic obstruction, the area of the stenotic orifice remains constant during haemodynamic interventions which alter the systolic volume or the contractile state of the left ventricle.

In contrast, in patients with HOCM, distinct variations in the magnitude of the intraventricular pressure gradient may be induced by a variety of physiological and pharmacological stimuli. Thus the dimensions of the obstructing outlet are not fixed in HOCM and the size of the left ventricular outflow tract varies, depending upon the contractile state of the myocardium, the venous return to the heart, and the distention of the ventricle during systole as governed by systemic arterial pressure (Braunwald and Ebert, 1962; Braunwald et al., 1964; Mason et al., 1965, 1966, 1967; DeMaria et al., 1974; Wampold et al., 1976). In the past, identification of HOCM by haemodynamics has required interventions to alter these determinants of left ventricular volume. The purpose of this report is to delineate

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a newly recognised haemodynamic characteristic of HOCM in the resting state: the configuration of the transaortic pressure gradient. Analysis of this gradient allows precise differentiation between HOCM and discrete aortic stenosis without the need for any provocative manoeuvre.

Patients

The left ventricular outflow pressure gradient was examined in 22 patients with HOCM (idiopathic hypertrophic subaortic stenosis) and in 40 patients with the discrete types of aortic stenosis. The patients with fixed aortic obstruction included 23 with valvular stenosis, 12 with discrete subvalvular membranous stenosis, and 5 with supravalvular stenosis. Left ventricular pressures were measured either by the transseptal method or by retrograde arterial catheterisation. In all 62 patients simultaneous intra-arterial pressure was obtained from the brachial artery and, in addition, sequential pressures were recorded by retrograde catheter pull-back across the obstructing area in 55 patients. The peak transaortic outflow systolic pressure gradient averaged 74 mmHg (range 32 to 119 mmHg) in HOCM, 96 mmHg (52 to 150 mmHg) in valvular aortic stenosis, 77 mmHg (55 to 115 mmHg) in membranous subvalvular stenosis and 105 mmHg (65 to 160 mmHg) in supravalvular aortic stenosis.

The configuration of the transaortic outflow pressure gradients was analysed in the following manner in each of the patients. The left ventricular pressure pulse upstream to the obstruction was compared with the simultaneously recorded arterial pressure pulse (Fig. 1). Five consecutive beats were examined and the results averaged in each patient. The upstroke of the brachial arterial pressure pulse was superimposed with the rise of left ventricular pressure to adjust for the brief temporal delay of brachial artery pressure transmission. Whether the brachial arterial pulse, properly adjusted for time, or the ascending aortic pressure pulse was used did not significantly affect the results. In the patients in whom both the left brachial artery and ascending aortic pressures were recorded, the peak systolic brachial artery pressure was only 4·8 ± 0·7 mmHg (SEM) greater than the simultaneously recorded peak systolic ascending aortic pressure. The duration of the ejection period was obtained from the onset of the upstroke of arterial pressure to the dicrotic notch of this pressure pulse. The ejection period was divided equally into its initial and final halves. The onset of ejection to peak left ventricular pressure was measured and the ratio of this interval to the duration of ejection calculated. Whether the peak of the arterial pressure pulse occurred in the first or second half of the systolic ejection period was also determined.

**Fig. 1** Representative recordings of simultaneous left ventricular (LV) and brachial arterial (BA) pressures in a patient with hypertrophic obstructive cardiomyopathy (IHSS) (A) and in a patient with fixed valvular aortic stenosis (AS) (B). The systolic ejection period is divided into its initial (A) and final (B) halves by the vertical dashed lines in the middle tracings in each panel. The trans-left ventricular outflow mean pressure gradient during the first half of ejection is indicated by the dotted area (A), while the mean gradient during the second half of ejection is depicted by the downward-dashed lines (B). The ratio of the time (seconds) from the onset to peak of the left ventricular pressure pulse related to the total duration (seconds) of the ejection period is indicated for the IHSS (HOCM) patient and the AS patient respectively. In addition, the characteristic ratio of the mean transaortic pressure gradient in the initial half of ejection (A) to that occurring in the second half of ejection (B) is also indicated for both representative patients.
Transaortic pressure gradient configuration in HOCM

The configuration of the transaortic pressure gradient was compared in HOCM and fixed aortic stenosis by measuring the mean pressure gradient during the first half of the ejection period relative to that during the second half (Fig. 1) by planimetry. The ratio of the mean pressure gradient of the initial one-half of the ejection period to the final one-half ejection period was then calculated.

Results

Representative recordings in HOCM and fixed valvular aortic stenosis are shown in Fig. 1 and the complete data from all of the patients are given in Figs. 2 and 3.

Peak arterial pulse

In each of the 22 patients with HOCM, the peak of the arterial pressure pulse occurred in the initial half of the systolic ejection period (Fig. 1A). In contrast, in 37 of the 40 patients with discrete aortic stenosis the arterial pressure pulse peak appeared in the final half of the ejection period (Fig. 1B). The 3 patients with discrete aortic stenosis in whom the arterial pressure pulse occurred in the first half of the ejection period had subvalvular membranous stenosis. In these 3 patients, the average peak systolic transaortic pressure gradient was 103 mmHg and there was considerable gross left ventricular hypertrophy; no evidence of muscular dynamic subaortic stenosis was detected by pre- or post-operative left heart catheterisation.

Onset of ejection to peak left ventricular pressure

Peak left ventricular pressure was delayed significantly (P < 0.01) in HOCM (Fig. 1A) compared with the total group of 40 patients with discrete aortic stenosis (Fig. 1B). Therefore, the relation between the interval from the onset of the peak of
the left ventricular pressure pulse divided by the ejection period was significantly greater (P < 0.01) in HOCM (0.59 ± 0.01, SEM) than this ratio in the 40 individuals with fixed aortic stenosis (0.49 ± 0.01) (Fig. 2). This ratio was also significantly greater in HOCM compared with the patients with valvular aortic stenosis (P < 0.01), the patients with discrete subvalvular membranous stenosis (P < 0.01), and the patients with supravalvular stenosis (P < 0.01).

RATIO OF MEAN PRESSURE GRADIENT DURING INITIAL TO FINAL HALF OF EJECTION
The ratio of the mean pressure gradient which occurred within the first half of systolic ejection to the mean pressure gradient during the second half of the ejection period was considerably less in HOCM (P < 0.01) (Fig. 1A) than in fixed aortic stenosis (Fig. 1B). Thus in HOCM the ratio of the initial to final mean pressure gradient was 0.59 ± 0.04; whereas this pressure gradient relation was 1.24 ± 0.06 in valvular aortic stenosis, 1.13 ± 0.06 in discrete subvalvular stenosis, and 1.85 ± 0.09 in supravalvular aortic stenosis (Fig. 3). Moreover, this ratio provided complete separation of all patients with HOCM from all individuals with the fixed types of aortic stenosis. Thus all patients with HOCM had a first to second half mean transaortic pressure gradient ratio of less than 0.80, whereas this ratio was always greater than 0.80 in all of those with discrete aortic stenosis (horizontal dashed line depicted on Fig. 3).

Discussion
Obstruction to left ventricular outflow in HOCM has recently been shown to be the result of paradoxical forward motion of the anterior mitral leaflet during cardiac contraction abutting against the relatively immobile hypertrophied interventricular septum (King et al., 1973a, b, 1974; Reis et al., 1974; Mason et al., 1975). The obstruction to left ventricular outflow in HOCM is thereby dynamic and forms with each systole. Furthermore, this dynamic obstruction in HOCM is incomplete in the initial phase of ejection, so that the earliest portion of ejection is not accompanied by obstruction while nearly complete impedance to left ventricular ejection takes place in the final one-half of systole (Pierce et al., 1964; Gault et al., 1966). In contrast, in the discrete types of aortic stenosis the obstruction to aortic outflow remains fixed throughout the entire period of systolic ejection and is thereby constant during the resting state and the haemodynamic interventions which alter contractility, venous return, and the systolic distending pressure of the left ventricular outflow tract.

The present study clearly documents that the configuration of the resting transaortic pressure gradient in patients with HOCM differs considerably from that of the fixed types of discrete obstruction to left ventricular outflow (Fig. 1). Thus, the peak of the brachial artery pressure pulse always occurred in the initial half of the ejection period in HOCM, while in the fixed types of aortic stenosis the peak arterial pressure pulse occurred in the second half of the ejection period. In addition, the onset of ejection to the peak of the left ventricular pressure pulse is related to the total duration and nature of obstruction to left ventricular outflow. Since peak left ventricular pressure is delayed in HOCM, the ratio of onset of ejection to peak left ventricular pressure relative to the duration of the systolic ejection period is greater in HOCM compared with the fixed, discrete types of aortic stenosis (Fig. 2). In addition, in contrast to HOCM, the left ventricular pressure pulse in the fixed forms of aortic stenosis was triangular in shape resembling an isovolumetric contraction, resulting in the peak left ventricular pressure pulse occurring in the middle or in the first half of the ejection period (Fig. 1B).

The aforementioned observations indicate that the early arterial and late ventricular pressure pulse peaks in HOCM are related to the absence of obstruction to ejection early in systolic ejection in HOCM (Fig. 1A). In contrast, in discrete aortic stenosis (valvular, subvalvular or supravalvular), as exemplified by the patient with valvular aortic stenosis shown in Fig. 1B, the constant obstruction to left ventricular outflow results in the peak arterial pressure pulse occurring in the final half of the ejection period and the peak left ventricular systolic pressure appearing in the mid-portion of the transaortic pressure gradient.

Most importantly, the ratio of the mean pressure gradient in the first half of ejection to that of the second half of ejection provided complete differentiation of HOCM from the fixed types of aortic stenosis at rest without provocative manoeuvres (Fig. 1 and 3). From these findings, it is clear that the examination of the configuration of the trans-left ventricular outflow pressure gradient at rest separates dynamic subaortic stenosis (HOCM) from the discrete forms of aortic stenosis.

References


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