Impaired Rate of Left Ventricular Filling in
Idiopathic Hypertrophic Subaortic Stenosis
and Valvular Aortic Stenosis

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SUMMARY
In order to determine whether there is any interference with left atrial emptying or left ventricular filling in idiopathic hypertrophic subaortic stenosis (IHSS) and aortic stenosis, the fall in pressure (y descent) of the left atrial y wave following the opening of the mitral valve was analyzed in 27 patients with IHSS and in 22 patients with valvular aortic stenosis, and the results were compared to those for 13 normal subjects and 24 patients with mitral stenosis. The y descent in 0.1 sec and the mean rate of the y descent, as well as the maximum rate of decline, were reduced in all three groups of patients, as compared to those of the normal group. The changes in IHSS were more marked than those occurring in patients with aortic stenosis or mitral stenosis. These findings in patients with IHSS and valvular aortic stenosis appear to result from reduced left ventricular compliance. It is concluded that there is an impairment of left ventricular filling in IHSS and that obstruction to ventricular inflow, as well as to outflow, contributes to the hemodynamic changes in this condition.

Additional Indexing Words:
Compliance Obstruction Mitral stenosis

Although extensive investigations have been carried out in order to elucidate the precise nature of the obstruction to ventricular outflow in idiopathic hypertrophic subaortic stenosis (IHSS), relatively little attention has been directed toward an analysis of ventricular inflow in this condition. It has been appreciated for some time that the circulatory changes and intensity of symptoms in IHSS are not necessarily related to the severity or even the presence of obstruction to left ventricular ejection. In attempts to explain the hemodynamic picture in this condition, it has been suggested that the distensibility of the hypertrophied left ventricle is diminished. This suggestion has been based on the finding of an elevated left ventricular end-diastolic pressure in the presence of a normal or even reduced left ventricular end-diastolic volume. Since the compliance of the left ventricle is diminished and ventricular volume tends to be reduced, the possibility was considered that this reduction of compliance might impair left ventricular filling and thus alter the hemodynamic and clinical state in patients with IHSS. Accordingly, in order to determine whether there is any interference with left atrial emptying and left ventricular filling, the fall in pressure of the left atrial y wave following opening of the mitral valve (y descent) was examined in patients with IHSS, and the results compared with those in normal subjects, in patients with valvular aortic stenosis, and finally in patients with mitral stenosis, in whom the major hemodynamic abnormality is an impairment of left ventricular filling.

Methods
A total of 86 patients was studied. Thirteen had normal cardiovascular systems and ranged in

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The initial 0.1-sec y descent in normal subjects (N) and patients with valvular aortic stenosis (AS), idiopathic hypertrophic subaortic stenosis (IHSS), and mitral stenosis (MS). Mean values ± 1 SEM are indicated.

Figure 1

Table 1

<table>
<thead>
<tr>
<th>y Descent indices</th>
<th>N-IHSS</th>
<th>N-AS</th>
<th>N-MS</th>
<th>AS-IHSS</th>
<th>IHSS-MS</th>
<th>AS-MS</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1-sec y descent</td>
<td>&lt; 0.01</td>
<td>&lt; 0.01</td>
<td>NS</td>
<td>&lt; 0.01</td>
<td>&lt; 0.01</td>
<td>NS</td>
</tr>
<tr>
<td>Ry index</td>
<td>&lt; 0.01</td>
<td>NS</td>
<td>&lt; 0.01</td>
<td>&lt; 0.05</td>
<td>NS</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>-dp/dt</td>
<td>&lt; 0.01</td>
<td>&lt; 0.01</td>
<td>&lt; 0.05</td>
<td>NS</td>
<td>&lt; 0.01</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviations: N = normal; AS = valvular aortic stenosis; IHSS = idiopathic hypertrophic subaortic stenosis; MS = mitral stenosis; -dp/dt = maximum rate of fall of left atrial pressure; NS = not significant.
3.4 ± 0.2 mm Hg (table 1). In patients with mitral stenosis the y descent in 0.1 sec averaged 6.3 ± 0.5 mm Hg, a value which was significantly greater (P < 0.01) than in the patients with IHSS, but not significantly different (P > 0.05) from that observed in patients with aortic stenosis and in normal subjects.

Figure 2 illustrates the Ry index in the patients studied. In the normal subjects this variable averaged 63.6 ± 6.1 mm Hg/sec, a value significantly greater (P < 0.01) than in patients with IHSS, in whom it averaged 34.4 ± 2.6 mm Hg/sec, and in patients with mitral stenosis in whom it averaged 35.7 ± 3.4 mm Hg/sec (table 1). The difference between Ry indices in normal subjects and in patients with aortic stenosis, in whom this value averaged 49.0 ± 5.8 mm Hg/sec, was not significant.

The most rapid rate of pressure decline (maximum negative dp/dt) averaged 128 ± 20 mm Hg/sec in the normal subjects, a value that was significantly greater (P < 0.01) than that observed in patients with aortic stenosis, in whom it averaged 77.0 ± 8.4 mm Hg/sec, and in patients with IHSS, in whom it averaged 66.6 ± 5.1 mm Hg/sec, as well as in those with mitral stenosis, in whom it averaged 91.0 ± 7.6 mm Hg/sec (P < 0.05) (fig. 3, table 1).

Discussion
Immediately following the opening of the mitral valve, left atrial pressure ordinarily falls rapidly, as blood flows from the left atrium into the left ventricle. However, when there is interference with left atrial emptying, as occurs in patients with mitral stenosis, the left atrium cannot empty rapidly in early diastole and the y descent of the left atrial pressure pulse is slow and prolonged. In addition, when there is restriction to diastolic filling of the left
ventricle as a result of reduced compliance of this chamber, the rate of decline of the y descent is also diminished. It has recently been proposed that the compliance of the hypertrophied left ventricle is reduced in patients with IHSS, since in these patients the left ventricular end-diastolic volume is reduced or normal, while the left ventricular end-diastolic pressure is elevated.¹⁻⁶

The major finding in this investigation is that slowing of the y descent also occurs in patients with IHSS. Thus, it was observed that three indices of the y descent, that is, the absolute decline in pressure during the initial 0.1-sec interval, the mean rate of pressure decline, and the maximum rate of fall of left atrial pressure decline were decreased in patients with IHSS when they were compared to normal subjects (figs. 1 to 4, table 1). These findings suggested that the ventricular inflow is impeded as a result of reduced ventricular compliance in IHSS, and it appears likely that limitation of left ventricular inflow contributes importantly to many of the hemodynamic changes in IHSS. It is appreciated that the left atrial pressure pulse is influenced by the pattern of blood flow entering the atrium throughout the cardiac cycle, as well as by

Figure 4
Representative left atrial (LA) pressure pulses in the four groups of persons studied. The values of the y descent that were calculated are shown at the bottom of each tracing.

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flow from the chamber during ventricular diastole. However, the characteristics of the fall of the y descent are related principally to the manner in which blood exits from the atrium. Further, the reduced compliance of the ventricle may result in elevations of left atrial pressure and often in reduced stroke volume and cardiac output. In attempting to produce more complete filling of the hypertrophied ventricle, a powerful atrial contraction occurs, reflected in prominent atrial contraction waves (a waves) in the left atrial pressure pulse (fig. 4). Despite a powerful atrial contraction, mean left atrial pressure remains elevated and leads to pulmonary venous and occasionally even to pulmonary arterial hypertension. Thus, the hemodynamic picture of left ventricular failure can be mimicked in the absence of true myocardial insufficiency.

It is important to note that the extent of elevation of the left ventricular end-diastolic pressure observed in patients with IHSS does not necessarily correlate with the severity of systolic obstruction nor with the presence of congestive heart failure. Since the major fraction of the stroke volume is expelled during the first half of systole in these patients and the ratio of the forward stroke volume to the end-diastolic volume (systolic ejection fraction) is normal, it appears that the reduction in ventricular end-diastolic volume may play a significant role in the small stroke volume which is noted in some patients with IHSS. In addition, diminished left ventricular compliance appears to play a significant role in the mechanism of one of the characteristic features of this condition, that is, the failure of the arterial pulse pressure to rise following a premature ventricular contraction. Thus, despite the prolonged diastolic pause following the premature contraction, the stiff, thickened left ventricle prevents the left ventricular end-diastolic pressure and volume from increasing normally. This factor combined with the post-extrasystolic potentiation of myocardial contractility prevents the stroke volume and therefore the arterial pulse pressure from increasing after the premature contraction.

It was observed in this study that the y descent is also slowed in valvular aortic stenosis; the decline during the initial 0.1-sec interval and the maximum negative dp/dt of the y descent were reduced, suggesting that the compliance of the left ventricle is also diminished in this condition. It appears, however, that the massively hypertrophied ventricle in patients with IHSS is even less compliant than that of patients with valvular aortic stenosis. Thus, both the left atrial pressure decline during the 0.1 sec following the opening of the mitral valve and the mean rate of y descent were significantly lower in patients with IHSS than in those with valvular stenosis (figs. 1 and 2, table 1). Further, in patients with IHSS the pressure decline during the initial 0.1 sec and the maximum negative dp/dt of the y descent were significantly lower than in patients with mitral stenosis (figs. 1 and 3, table 1).

The finding that ventricular compliance in IHSS appears to be more abnormal than in valvular aortic stenosis is consonant with other observations. Thus, left ventricular end-diastolic volumes in patients with valvular aortic stenosis tend to be larger than in patients with IHSS and, indeed, may be greater than in normal subjects. Further, mean left atrial and ventricular end-diastolic pressures tend to reach higher levels in IHSS than in patients with valvular aortic stenosis with a comparable severity of obstruction. These findings, taken together with the reduced systolic ejection fraction in patients with valvular aortic stenosis, suggest that the reduction of stroke volume which occurs in some patients with valvular stenosis results largely from inadequate ventricular emptying, while in patients with IHSS it is primarily consequent to inadequate filling of the left ventricle.

Although the mean left atrial pressure is elevated, the a wave dominant, and the y descent prolonged in each of the three conditions examined herein, only in mitral stenosis is there an absence of diastasis in the left
atrial pressure pulse during mid and late diastole (fig. 4). In IHSS and valvular aortic stenosis, ventricular filling, while slower than normal, does occur to an extent sufficient to fill the poorly distensible left ventricle and hence ventricular and atrial pressures usually rise during the latter half of diastole. The absence of diastasis with long diastolic intervals in the left atrial pressure pulse occurs characteristically in patients with mitral stenosis, as a consequence of the impaired filling of the normally compliant left ventricle.

It is appreciated that in patients with mitral stenosis, and perhaps in other conditions as well, the mitral valve may not open for a short interval after the peak of the y wave, that is, after the onset of the y descent. Thus, under these circumstances, the first portion of the y descent may be slightly less rapid than that which occurs after the valve opens, and the first portion might not be influenced by the size of the mitral orifice or the compliance of the left ventricle. However, this factor, which might result in a slight reduction of the pressure decline during the initial 0.1 sec and of the mean rate of the y descent, is generally of minor significance since this delay is generally of the order of only a few hundredths of a second. In addition, the maximum rate of pressure decline of the y descent, which occurred in the midportion of the y descent, well after the mitral valve had opened, was also examined in this study and the differences between the groups were similar in direction and degree to those obtained from the other characteristics of the y descent. The characteristics of the left atrial pressure pulse analyzed herein were not related to the mean left atrial pressure as is sometimes done, although such a relation would have resulted in an even greater separation of the measurements between the patients and normal subjects, since mean left atrial pressures were elevated in all three groups of patients.

Two possible mechanisms might be invoked to explain the reduced left ventricular compliance in patients with IHSS. It may reflect merely an increase in the total mass of the left ventricular myocardium, that is, an augmentation of the thickness of the left ventricular wall, without a change in the compliance of each individual myocardial fiber. Compliance may also be reduced by an increased stiffness of the myocardial fibers themselves. While no definite choice between these two mechanisms can be made, it is relevant that in studies of isolated myocardium obtained from rats and cats with experimentally induced ventricular hypertrophy, no abnormalities of resting length-tension relations were observed, when appropriate corrections were made for the thickness of the muscle. Since the left ventricular wall tends to be thicker in patients with IHSS than with other conditions, perhaps the reduced compliance can be best explained by a simple increase in thickness of the left ventricular wall. Finally, it is possible that there might be interference with complete opening of the mitral valve by massive ventricular hypertrophy in patients with IHSS. This postulation that there may actually be obstruction to left ventricular inflow in IHSS is consonant with the view of Goodwin and his collaborators that the enlarged septum may interfere with blood flow across the tricuspid and mitral valves.

In conclusion, the present investigation showed that the rate of left atrial pressure fall after the opening of the mitral valve and, presumably therefore, the rate of left ventricular filling are reduced in IHSS and valvular aortic stenosis in a manner similar to that observed in mitral stenosis. In contrast to mitral stenosis, inflow in the conditions associated with ventricular hypertrophy is attenuated by the decreased compliance of the thickened ventricular wall. Based on examination of the y descent, this diminution is most severe in IHSS and appears to contribute to certain hemodynamic abnormalities in this disease.

References

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