Interventricular Septal Thickness and Left Ventricular Hypertrophy
An Echocardiographic Study

SANTOSH KANSAL, M.D., DAVID ROITMAN, M.D., AND L. THOMAS SHEFFIELD, M.D.

SUMMARY Septal and left ventricular posterior wall (LVPW) thicknesses and their ratios were studied at the left ventricular outflow tract and left ventricular cavity in 66 patients with echocardiographically diagnosed left ventricular concentric hypertrophy, 20 with idiopathic hypertrophic subaortic stenosis (IHSS), and 34 normal subjects. Concentric hypertrophy was due to hypertension in 41 subjects and to valvular disease in 15 subjects. Septal thickness in normal subjects was related to body surface area (p < 0.02). In 12% of normal subjects, 39% of patients with concentric hypertrophy and 95% with IHSS, the septal/LVPW ratio was ≥ 1.3. Thirty-two percent of patients with hypertension, 78% with aortic stenosis, and 60% with aortic insufficiency had septal/LVPW ratios ≥ 1.3 at left ventricular midcavity level.

In conclusion, a septal/LVPW thickness ratio of ≥ 1.3 is common in patients with concentric left ventricular hypertrophy and may also occur in normal subjects. A ratio ≥ 1.5 may be more specific for genetically determined asymmetric septal hypertrophy.

SINCE THE INTRODUCTION of the term asymmetric septal hypertrophy (ASH) in 1973, controversy has arisen regarding its clinical and echocardiographic significance. The diagnosis of ASH is based on echocardiographic analysis that shows disproportionate septal thickness and a ratio of septal-to-left ventricular free wall of 1.3 or more. This specific ratio has led to study of the septum and its relation to the left ventricular free wall in many congenital and acquired cardiac diseases, and has revealed that many diseases, such as pulmonary hypertension, pulmonary stenosis, congenital malformation of the mitral valve, coarctation of the aorta, and aortic valvular disease manifest disproportionate septal thickness.

In this study we assessed the interventricular septal and left ventricular free wall thicknesses and their ratio in patients with concentric hypertrophy. We also compared patients who had idiopathic hypertrophic subaortic stenosis (IHSS) with normal subjects.

Subjects and Methods
One hundred twenty patients were studied, including 66 patients with concentric left ventricular hypertrophy, 20 patients with IHSS, and 34 normal subjects.

In the first group, the diagnosis of concentric hypertrophy was based on echocardiographic measurement of left ventricular posterior wall and interventricular septum thickness of ≥ 11 mm in the absence of a small left ventricular outflow tract or abnormal systolic anterior motion of the mitral valve. This hypertrophy was due to various primary conditions (table 1). In several patients more than one disease was present,
but only the principal disease was used for classification in this study. In this group, 37 (55%) were males and 30 (45%) were females; ages ranged from 16–70 years (mean 50 years).

In the second group, all 20 patients had been clinically suspected of IHSS and all had echocardiographic evidence of septal hypertrophy, abnormal anterior systolic excursion of the anterior mitral cusp, with or without any provocation, and small left ventricular outflow tract. None of these patients had aortic valvular disease, hypertension or chronic renal disease. In 11 patients, the diagnosis was confirmed by catheterization, and another four had relatives with typical IHSS. There were 10 males and 10 females, and the age range was 19–63 years (mean 34 years).

The third group included 34 normal subjects who were hospital employees and medical staff with normal medical histories, symptom reviews, and cardiovascular examinations. The four subjects with a septal-to-free wall ratio ≥ 1.3 had no abnormal systolic excursion of the mitral valve even after provocation with amyl nitrite. This group consisted of 22 males (65%) and 12 females (35%); age range was 20–54 years (mean 30 years).

Echocardiograms were recorded on a strip chart using a 2.22-MHz transducer of 0.5-cm diameter and 7.5–10 cm focal length. Echocardiograms were performed via the third or fourth left intercostal space along the left sternal border with the patient in the supine position. The 30° left lateral position was used for patients with inadequate records in the supine position. To evaluate the level of maximum septal thickness, we measured septal and left ventricular posterior wall thicknesses at the left ventricular outflow tract and at the middle of the left ventricular cavity.

The level of left ventricular outflow tract was defined as the level of the anterior mitral leaflet where both the interventricular septum and left ventricular posterior wall were simultaneously recorded while scanning from aortic root toward the mitral valve (fig. 1). The left ventricular midcavity level was taken just below the mitral valve where only discontinuous reflections from the edges of the leaflets were evident, with the transducer directed posteriorly to pick up the maximum septal and posterior wall excursions.

Interventricular septal thickness was measured, as shown in figure 1, from the most distinct echoes including right and left endocardial surfaces at end-diastole, which was determined by the peak of the R wave of the simultaneously-recorded ECG. Right septal wall echoes were demarcated by proper damping. Echoes reflected from the tricuspid annulus were not included in the measurements. The left ventricular

![Figure 1](https://example.com/figure1.png)
posterior wall was measured at end-diastole from the endocardium, which was clearly demarcated from posterior chordae tendineae on the basis of their smaller posterior excursion compared with that of the left ventricular posterior wall. The measurement was continued to the strong reflection where epicardium meets pericardium or, if pericardial effusion was present, to the epicardial-fluid interface reflection. Pairs of septal and left ventricular posterior wall measurements were made during the same cardiac cycle, and the average of at least three cycles was taken. Each patient’s records were measured simultaneously by two investigators.

Body surface area was estimated by height and weight. The diagnosis of concentric hypertrophy was based on a left ventricular free wall and septal thickness \( \geq 11 \) mm.

**Results**

**Concentric Hypertrophy**

Records adequate for analysis were obtained from 40 patients at the left ventricular outflow tract, and in 66 at the level of the left ventricular midcavity. The ratio of the interventricular septum to the left ventricular posterior wall (IVS/LVPW) at the left ventricular outflow tract and the left ventricular midcavity were identical (1.3 ± 0.2 [mean ± SD]). Measurements from three clinical subgroups follow.

**Chronic Renal Failure with Hypertension**

In 28 patients measured at left ventricular outflow tract and 41 at left ventricular midcavity the mean IVS/LVPW ratio was the same at both levels (1.27 ± 0.2 [table 2]).

**Aortic Stenosis**

Five patients were measured at left ventricular outflow tract level and nine at left ventricular midcavity. Mean IVS/LVPW thickness ratios were 1.5 ± 0.2 and 1.4 ± 0.2, respectively.

**Aortic Insufficiency**

Four patients were measured at left ventricular outflow tract level and five at left ventricular midcavity. IVS/LVPW thickness ratios were 1.2 ± 0.2 and 1.3 ± 0.1, respectively.

**IHSS**

Six patients were measured at left ventricular outflow tract level and 20 at left ventricular midcavity. The mean IVS/LVPW ratios were the same at both levels, 1.9 ± 0.3. In one patient, IVS/LVPW at left ventricular midcavity was 1.18. This patient had typical clinical and echocardiographic features of IHSS and had concentric left ventricular hypertrophy secondary to IHSS.

**Normal Subjects**

Records at the left ventricular outflow tract level from 24 subjects were satisfactory for measurement, yielding a mean IVS/LVPW ratio of 1.24, while in 34 subjects at left ventricular midcavity level the ratio was 1.16 ± 0.2 (table 2, figs. 2 and 3).

The number of patients in each group who had a septal-to-left ventricular free wall ratio \( \geq 1.3 \) (figs. 4 and 5) at left ventricular midcavity level was determined. Table 3 shows that 12% of normal subjects, 39% of patients with concentric left ventricular hypertrophy, and 95% of patients with IHSS had a ratio of \( \geq 1.3 \). According to this criterion, 78% of patients with aortic stenosis, 60% with aortic insufficiency and 32% with chronic renal failure also had ASH.

Interventricular septum and left ventricular posterior wall thicknesses were considered in relation to body surface area. This correlation was studied by linear regression equation. Patients with edema, which

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**Table 2. Ventricular Wall Measurements and Ratios**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Measurements at left ventricular outflow tract (mm)</th>
<th>Measurements at left ventricular midcavity (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of patients studied</td>
<td>IVS/LVPW ratio (range and mean ± SD)</td>
</tr>
<tr>
<td>--------------------------------</td>
<td>------------------------</td>
<td>--------------------------------------</td>
</tr>
<tr>
<td>Concentric hypertrophy</td>
<td>40 R 8.6-22 R 10-18</td>
<td>M 16.8 ± 3.1 M 13 ± 2.2 M 0.7-1.8</td>
</tr>
<tr>
<td>Chronic renal failure with</td>
<td>28 R 8.6-22 R 10-18</td>
<td>M 16.7 ± 3.0 M 13 ± 2.2 M 0.7-1.8</td>
</tr>
<tr>
<td>hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>5 R 12-21 R 10-15</td>
<td>M 18 ± 3.6 M 12 ± 1.9 M 1.2-1.8</td>
</tr>
<tr>
<td>Aortic insufficiency</td>
<td>4 R 12-18 R 12-15</td>
<td>M 15.6 ± 2.9 M 12.8 ± 1.4 M 1.2 ± 0.2</td>
</tr>
<tr>
<td>IHSS</td>
<td>6 R 18-24 R 10-12</td>
<td>M 21 ± 2.3 M 11 ± 1 M 1.9 ± 0.29</td>
</tr>
<tr>
<td>Normal subjects</td>
<td>24 R 8.6-11 R 5-8.6</td>
<td>M 8.6 ± 1.6 M 7 ± 1.3 M 0.8-1.6</td>
</tr>
</tbody>
</table>

Abbreviations: R = range; M = mean; IVS = interventricular septum; LVPW = left ventricular posterior wall; IHSS = idiopathic hypertrophic subaortic stenosis.
would affect body weight, were not included in the correlation of measurements with body surface area.

In normal subjects interventricular septum and left ventricular posterior wall thicknesses measured at the left ventricular outflow tract (fig. 6) were more strongly related to body surface area (p < 0.02) than were the same measurements made at left ventricular midcavity (p < 0.05) (fig. 7). There was no significant relation of body surface area to the IVS/LVPW ratio (p < 0.22).

In patients with IHSS, left ventricular posterior wall thickness at left ventricular midcavity was significantly related to body surface (p < 0.01), but other measurements were not (p > 0.15).

Discussion

According to the previous reports, normal septal-to-left ventricular posterior wall ratio is 1:1, while recently higher ratios have been reported by Maron and Bahler. The maximum septal/posterior wall ratio at midcavity in one of our normal subjects was between 1.5 and 1.6, an unusually high value. Maron has reported the maximum ratio of 1.4. This minor difference may be due to use of different fiducial points for end-diastole. In the present study the maximum septal thickness in one normal subject was 14 mm; Bahler has reported 16 mm.
In normal subjects the evidence of disproportionate septal hypertrophy was more frequent (24%), and the ratio was higher (mean ratio 1.24) at the left ventricular outflow tract level than at left ventricular midcavity. This variation was due to the relatively thinner left ventricular posterior wall at left ventricular outflow tract. The interventricular septum and left ventricular posterior wall are relatively thicker at the level of left ventricular cavity. Disproportionate septal thickness has been reported in young athletes\(^{14}\) and weight lifters.\(^{15}\) According to Bulkley,\(^{16}\) the septum is disproportionately thick in the embryo and frequently in neonates as well.

There have been few echocardiographic reports on concentric left ventricular hypertrophy. In this study, 39% of patients had disproportionate septal hypertrophy by a conventional criterion. Abbasi\(^{7}\) reported a septal-to-free wall ratio of 1.2 ± 0.1 in such patients; according to Criley,\(^{17}\) 40.6% of patients with malig-
nant hypertension have disproportionate septal hypertrophy.

In this study 32% of patients with chronic renal failure with hypertension had ASH, while Abbasi and Schott reported 39% and 5% of patients, respectively. These authors used a ratio of 1.5 as criterion for ASH.

Maron reported ASH in 10% of aortic stenosis patients. The ratio was normal (1.03 ± 0.06) in 11 patients with fixed left ventricular outflow obstruction.

Figure 6. Relation of interventricular septum (IVS) and left ventricular posterior wall (LVPW) thickness at left ventricular outflow tract (LVOT) to body surface area (BSA) (m²) in normal subjects. Dense lines signify more than one subject with same body surface area.

Figure 7. Relation of interventricular septum (IVS) and left ventricular posterior wall (LVPW) thickness at left ventricular mid-cavity level (LVC) to body surface area (BSA) (m²) in normal subjects. Dense lines signify more than one subject with same body surface area.
reported by Henry. The present report shows a higher incidence of ASH in aortic valvular disease owing to patient selection; all the patients in the present study had increased left ventricular wall thickness. They represent a more severely diseased group than would result from the consecutive selection of patients with aortic valvular disease regardless of the presence of left ventricular hypertrophy.

ASH has been reported in diverse diseases such as pulmonary stenosis or hypertension, congenital mitral valve deformities, coarctation of the aorta, supravalvular ring of left atrium and ventricular septal defect, mitral valve prolapse, acromegaly, inferior myocardial infarction, and hyperthyroidism. It also appears in normal subjects and weight lifters. Considering this, there is doubt that ASH determined on the basis of an IVS/LVPW ratio of ≥1.3 has definite association with genetically determined hypertrophic cardiomyopathy. Even disorganized cardiac muscle cells are not specific for this entity. It seems preferable that an IVS/LVPW ratio of ≥1.3 not be used to designate ASH due to its occurrence in normal subjects and in patients with concentric left ventricular hypertrophy. Abbasi has reported evidence that a ratio of 1.5 has increased specificity for inherited ASH, and Maron and Shah have agreed with this position. A ratio of ≥1.5 can differentiate normal subjects from patients with IHSS, but this figure cannot satisfactorily differentiate patients with IHSS from patients with concentric hypertrophy, inasmuch as 18% of the latter group had ratios ≥1.5. Absolute septal thickness of <15 mm distinguishes normal subjects from IHSS but will not differentiate between IHSS and left ventricular hypertrophy because 50% of patients in latter group had septal thickness ≥15 mm.

The cause of ASH is not understood. In normal healthy subjects it may be a persistence of the neonatal pattern. According to Bahler, more physically active than sedentary people have thick interventricular septa. Of the four persons in the present study who have IVS/LVPW ≥1.3, none is athletic.

ASH may be an early marker of concentric left ventricular hypertrophy due to its occasional presence in aortic valvular disease and hypertensive patients in the absence of abnormally increased left ventricular posterior wall thickness, and one presumes that with the progression of the disease, concentric hypertrophy will develop. This view is consistent with that of Bahler.

ASH may also be an early marker of generalized cardiomyopathy due to various systemic diseases in which myopathy is a feature, such as thyrotoxicosis, Pompe’s disease and Friedreich’s ataxia.

Since normal cardiac size varies in proportion to body size, it was not surprising to find interventricular septal and left ventricular posterior wall thicknesses significantly related to body surface area. The wide fluctuation in weight of patients on chronic dialysis degraded this relationship. Abnormal increases in left ventricular wall thickness due to hypertrophy also reduce the relation of these measure-ments to body surface area. However, since in IHSS the posterior left ventricular wall is not primarily affected by hypertrophy as is the septum, it is expected that the relationship of this measurement to body surface area is preserved in this group of patients.

Any study involving measurements made by M-mode echocardiography must take account of the pitfalls and limitations of this method. Wall thicknesses are accurate only when the measuring beam is perpendicular to the structure measured, and internal dimensions vary according to the orientation of the measuring beam. Interface reflections can be insufficiently clear to yield less than 2 mm of imprecision. Different electrocardiographic landmarks for end-diastole in different studies may account for minor differences in septal and left ventricular posterior wall thicknesses and their ratio.

It was not generally appreciated at the inception of this study that most accurate echocardiographic measurements of thicknesses must be taken from first major impulse of the proximal interface to the first major impulse of the distal interface (‘leading edge to leading edge’). When optimum damping is used, as it was in this study, the imprecision contributed by aftervibrations from intracardiac interfaces is small. The measurement procedure used in this study is similar to that used in previous studies of septal thickness, and thus is directly comparable with them.

In conclusion, a septal/free wall thickness ratio of ≥1.3 is too common in concentric left ventricular hypertrophy and even in normal subjects to be a useful criterion of ASH. A ratio of ≥1.5 at midcavity level will differentiate normal subjects from IHSS, but this echo will frequently fail to distinguish concentric left ventricular hypertrophy from IHSS. An absolute septal thickness of 15 mm will differentiate normal subjects from IHSS, but will not differentiate between IHSS and concentric left ventricular hypertrophy.

Acknowledgment

The authors gratefully acknowledge Gladys Poe and Maria Clark for recording the echocardiograms; Faye Sprinkel for assistance in compiling clinical data; Katherine Kirk, Ph.D. for assistance in numerical analysis; and Myrnie Driskill, Juanita Brasher and Betty Doyle for assisting in the preparation of this manuscript.

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Interventricular septal thickness and left ventricular hypertrophy. An echocardiographic study.
S Kansal, D Roitman and L T Sheffield

Circulation. 1979;60:1058-1065
doi: 10.1161/01.CIR.60.5.1058

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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