Relation between Echocardiographically Determined Left Atrial Size and Atrial Fibrillation

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SUMMARY In an attempt to define quantitatively the relation between left atrial size and atrial fibrillation, echocardiography was used to study 85 patients with isolated mitral valve disease, 50 patients with isolated aortic valve disease, and 130 patients with asymmetric septal hypertrophy. In all three groups of patients, atrial fibrillation was rare when left atrial dimension was below 40 mm (3 of 117 or 3%) but common when this dimension exceeded 40 mm (80 of 148 or 54%). In addition, when left atrial dimension exceeds 45 mm, cardioversion, while initially successful, is unlikely to produce sinus rhythm that can be maintained at least six months. These data suggest that left atrial size is an important factor in the development of atrial fibrillation and in determining the long term result of cardioversion. The pathophysiologic mechanism most consistent with this is that a chronic hemodynamic burden initially produces left atrial enlargement which in turn predisposes to atrial fibrillation. Only prospective studies will determine definitively whether these observations will be useful in decisions concerning prophylactic anticoagulation and elective cardioversion.

ATRIAL FIBRILLATION occurs in association with a variety of disease states. A particularly strong association is found both in patients with mitral valve disease, and in patients with asymmetric septal hypertrophy (ASH), with the onset of this arrhythmia often precipitating or exacerbating the symptoms and signs of congestive heart failure.1-3 In addition, the development of atrial fibrillation markedly increases the risk of embolization, a complication that often is devastating or sometimes fatal.4-10

Because rheumatic fever is the etiology of the mitral valve abnormalities in many patients with mitral valve disease, some investigators have suggested that atrial fibrillation is related to rheumatic involvement of the left atrial wall.11 Others, however, have noted that patients with atrial fibrillation have a large left atrium and interpret this as evidence that atrial dilatation is in some way related to atrial fibrillation.3, 12, 13 Still others have suggested that age is an important factor influencing the development of atrial fibrillation.9

These associations are not only in understanding the pathophysiology of atrial fibrillation, but also have potentially important clinical and therapeutic implications. For instance, the embolic events that are a major complication of atrial fibrillation may occur at onset of the arrhythmia or shortly following conversion from atrial fibrillation to normal sinus rhythm.4-6, 14-16 Therefore, if a subgroup of patients in normal sinus rhythm could be identified who are at risk of developing atrial fibrillation, prophylactic anticoagulation and antiarrhythmic drugs might be used as a possible means of preventing fibrillation-induced emboli. In addition, if patients in atrial fibrillation could be identified who are unlikely to be maintained in sinus rhythm following cardioversion, attempts to convert the arrhythmia might be abandoned, thereby eliminating the risk of postcardioversion embolization.

Echocardiography has proven to be a valuable non-invasive tool for quantitatively assessing left atrial size.17-19 In the present study, we used this technique to evaluate the association of left atrial size and the development of atrial fibrillation as well as the relation of left atrial size to the success or failure of cardioversion in patients with isolated mitral valve disease, asymmetric septal hypertrophy (ASH), and isolated aortic valve disease. In addition, we examined the prevalence of embolic complications in these same patients.

Methods

Patient Population

The study population consisted of 85 patients with isolated mitral valve disease (55 women, 30 men), 50 patients with isolated aortic valve disease (11 women, 39 men), and 130 patients with asymmetric septal hypertrophy (44 women, 86 men), in whom adequate echocardiograms were obtained during evaluation either in the outpatient clinic or the inpatient service of the National Heart and Lung Institute. The degree of functional impairment of the patients ranged from minimal to severe, and many of the latter required cardiac catheterization and operative intervention.

Echocardiographic Evaluation

The echocardiographic system used consists of an Ekoline 20A ultrasound unit, Honeywell 1856 Line Scan Recorder, Hewlett-Packard X-Y display, and a custom-built video amplifier. Echocardiographic studies were performed in every patient supine and in a basal state. In each patient with aortic valve disease, review of the echocardiograms failed to reveal evidence of mitral stenosis. In patients with asymmetric septal hypertrophy, the septal free-wall ratio (by definition) equaled or exceeded 1.3 in every case.20

Left Atrial Size

Left atrial dimension in early diastole was obtained in every patient by angling the ultrasound beam medially and in a cephalad direction from the mitral valve tip until the aortic root was seen. Minor angulations of the transducer...
were made until the aortic valve signals were visualized.\textsuperscript{19} Signal intensity was then gradually reduced (using the damping control on the Ekoline 20A) in order to clarify the interface between the posterior left atrial wall and lung (fig. 1). Left atrial dimension in early diastole was measured from the damped portion of the record by recording the distance, in millimeters, from the posterior aortic wall to the posterior left atrial wall. In all cases, the left atrial dimension measured in early diastole also represented the maximum left atrial dimension. In those patients in whom a cardiac operation was performed, the left atrial dimension that was used in the present study was obtained prior to operation.

Clinical Evaluation

The clinical record of each patient was reviewed to obtain the patient's age, likely etiology of the valve lesion, age at onset of atrial fibrillation, and clinical data relating to results and efficacy of cardioversion, if attempted. In addition, the patient's past history of arrhythmias and of systemic or pulmonary emboli was obtained.

Patients were subdivided into one of three groups on the basis of their past history of arrhythmias (for patients subsequently undergoing operation, only the preoperative history was used): a) normal sinus rhythm — no history of documented atrial fibrillation, either transient or long-lasting; b) paroxysmal atrial fibrillation — in sinus rhythm at time of echocardiographic evaluation but with a previous history of atrial fibrillation (either documented by their referring physician or at the National Institutes of Health) lasting less than 24 hours and reverting to normal sinus rhythm spontaneously (in ten patients with paroxysmal atrial fibrillation, ECG documentation had not been obtained but the patient's history of a rapid, grossly irregular heart beat lasting at least 10 min was taken as indicative of atrial fibrillation); c) atrial fibrillation — in atrial fibrillation at time of echocardiographic evaluation, or if in sinus rhythm, having been previously in atrial fibrillation for longer than 24 hours and requiring DC cardioversion or medication for conversion to normal sinus rhythm.

Hemodynamic Evaluation

Cardiac catheterization data were available in 46 of 85 (54%) patients with isolated mitral valve disease, 43 of 50 (86%) patients with isolated aortic valve disease, and 81 of 130 (62%) patients with asymmetric septal hypertrophy. In each of the catheterized patients, mean left atrial pressure or mean pulmonary capillary wedge pressure data were available.

![Figure 1](http://circ.ahajournals.org/)

**Figure 1** Unretouched echocardiogram from a patient with mitral valve disease and atrial fibrillation. Left atrial dimension (LATD) is measured from the damped portion of the record. Ao = aorta; AoV = aortic valve.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2** Plot of age in years at time of echocardiographic study (horizontal axis) and left atrial dimension in millimeters (vertical axis) for patients with isolated mitral valve disease. Patients in normal sinus rhythm (NSR) are shown by open symbols, those with paroxysmal atrial fibrillation (PAF) by half closed symbols, and those with chronic atrial fibrillation (AF) by the completely closed symbols.


Results

Left Atrial Dimension and Age

The relation between age (at time of echocardiographic study), left atrial dimension, and cardiac rhythm is shown in figures 2-4 for each of the three groups of patients. In general, atrial fibrillation was common only in those patients whose age was greater than 40 years and in addition had left atrial dilatation.

Atrial Fibrillation and Specific Hemodynamic Abnormalities

When patients with mitral valve disease and a left atrial dimension of 40 mm or greater were subdivided according to the mitral valve lesion, no significant difference (by Fisher exact test) was found in the prevalence of atrial fibrillation (paroxysmal plus chronic): i.e., 18 of 23 (78%) patients with pure mitral stenosis were classified as atrial fibrillation versus eight of 13 (62%) patients with pure mitral regurgitation and 21 of 25 (84%) patients with mixed mitral stenosis and regurgitation. Similarly, in patients with asymmetric septal hypertrophy and a left atrial dimension of 40 mm or greater, no significant difference was found in the prevalence of atrial fibrillation (paroxysmal plus chronic) when patients were subdivided on the basis of the severity of left ventricular outflow obstruction:21 i.e., 19 of 46 (41%) patients with obstructive ASH were classified as having atrial fibrillation versus six of 14 (43%) with provokable ASH and six of 15 (40%) with nonobstructive ASH.

![AORTIC VALVE DISEASE](image1)

**Figure 3** Plot of age in years at time of echocardiographic study (horizontal axis) and left atrial dimension in millimeters (vertical axis) for patients with isolated aortic valve disease. Symbols are similar to those of figure 2.

![ASYMMETRIC SEPTAL HYPERTROPHY](image2)

**Figure 4** Plot of age in years at time of echocardiographic study (horizontal axis) and left atrial dimension in millimeters (vertical axis) for patients with asymmetric septal hypertrophy (ASH). Symbols are similar to those of figure 2.

Left Atrial Pressure and Age

No significant relation was found between age (at time of hemodynamic study), mean left atrial pressure (or mean pulmonary capillary wedge pressure) and cardiac rhythm in any of the three patient groups. Of particular pathophysiologic significance, the prevalence of atrial fibrillation was low (4%) in patients with aortic valve disease, despite the fact that 19 of 50 (38%) patients were at least 40 years of age and also had elevated mean left atrial pressure. Thus, in contrast to the left atrial dimension data, left atrial pressure does not provide a strong predictive index of atrial fibrillation.

Age at Onset of Atrial Fibrillation

The age at onset of atrial fibrillation is shown graphically in figure 5 for patients with isolated mitral valve disease and those with asymmetric septal hypertrophy. In eight patients, age at onset of atrial fibrillation could not be determined.

Left Atrial Dimension and Cardioversion

The relation between left atrial dimension, length of time in atrial fibrillation prior to cardioversion attempt, and the success or failure of DC cardioversion for nonoperated patients with either isolated mitral valve disease or asymmetric septal hypertrophy is shown in figure 6. In this analysis, cardioversion was considered unsuccessful either if normal sinus rhythm was not achieved or if achieved, it was not maintained for at least six months. In some individuals, more than one cardioversion attempt was made. If any one
of the multiple attempts produced normal sinus rhythm that was maintained for at least six months, the patient was considered to have been successfully cardioverted, even if previous or subsequent attempts were failures.

In every patient with asymmetric septal hypertrophy and 19 of 24 (79%) patients with mitral valve disease, cardioversion was attempted with DC electric shock. In five patients with mitral valve disease, cardioversion had been attempted with quinidine sulphate alone. Of note, the echocardiographic assessment of left atrial size was obtained within one month of the cardioversion attempt in 17 of 37 (46%) patients. If these patients are considered as a separate group, cardioversion was successful in producing and maintaining sinus rhythm for at least six months in six of 17 (35%) patients compared to nine of 37 (24%) of the total group of patients.

Occurrence of Systemic or Pulmonary Emboli

A history of at least one embolic event (either systemic or pulmonary) was noted in 22 of 85 (26%) patients with isolated mitral valve disease (17 systemic emboli and 5 pulmonary), one of 50 (2%) patients with isolated aortic valve disease (1 systemic), and 11 of 130 (8%) patients with asymmetric septal hypertrophy (10 systemic and 1 pulmonary). In this analysis, patients are not included in the embolization group if the only emboli that occurred were after operative treatment of their lesions.

Of the patients with mitral valve disease who had emboli, 21 of 22 (95%) patients at the time of echocardiographic study would have been classified by history as having either chronic (20 patients) or paroxysmal (one patient) atrial fibrillation. Moreover, of the 21 patients with mitral valve disease and atrial fibrillation who eventually embolized, eight (38%) had the initial embolus within one week of the onset of atrial fibrillation (table 1). When related to the number of patients who developed either chronic or paroxysmal atrial fibrillation, embolic episodes occurred at onset of the arrhythmia in eight of 49 (16%) patients with isolated mitral valve disease.

Of the patients with asymmetric septal hypertrophy who embolized, nine of 11 (82%) at the time of echocardiographic study would have been classified by history as having either chronic (7) or paroxysmal (2) atrial fibrillation. In these patients with asymmetric septal hypertrophy, three of nine (33%) had embolic episodes within one week of the onset of atrial fibrillation (table 1). When related to the number of patients with asymmetric septal hypertrophy who developed atrial fibrillation (either chronic or paroxysmal), embolic episodes occurred at onset of atrial fibrillation in

![Figure 5](image-url)  
**Figure 5** Age at onset of atrial fibrillation for patients with isolated mitral valve disease (upper panel) and those with asymmetric septal hypertrophy (lower panel). The horizontal axis in each panel is subdivided into two-year increments with the vertical axis showing the number of patients in whom atrial fibrillation first occurred during each of the two-year increments.

![Figure 6](image-url)  
**Figure 6** Plot of left atrial dimension in millimeters (vertical axis) and length of time in atrial fibrillation prior to the DC cardioversion attempt (horizontal axis) in nonoperated patients with isolated mitral valve disease (upper panel — circles) and asymmetric septal hypertrophy (lower panel — triangles). The closed symbols represent patients in whom cardioversion was unsuccessful (i.e., did not produce and maintain sinus rhythm for at least 6 months), while the open symbols represent patients who were successfully cardioverted and maintained sinus rhythm for 6 months or longer. The diagonal line identifies patients in whom the echocardiographic measurement of left atrial dimension was obtained within one month of the cardioversion attempt.
TABLE 1. Risk of Embolic Complications in Relation to Time from Onset of Atrial Fibrillation

<table>
<thead>
<tr>
<th>Interval from onset AF to initial embolic event</th>
<th>Number of pts at risk (in AF)</th>
<th>Number of pts in AF who embolized</th>
<th>% of total pts who embolized</th>
<th>Risk of initial embolus per patient year</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mitral Valve Disease</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset (&lt;1 week)</td>
<td>43</td>
<td>8</td>
<td>38%</td>
<td>—</td>
</tr>
<tr>
<td>1 week – 6 months</td>
<td>32</td>
<td>4</td>
<td>19%</td>
<td>26%</td>
</tr>
<tr>
<td>6 months – 1 year</td>
<td>25</td>
<td>0</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>1–5 years</td>
<td>25</td>
<td>5</td>
<td>24%</td>
<td>7%</td>
</tr>
<tr>
<td>5–10 years</td>
<td>12</td>
<td>2</td>
<td>10%</td>
<td>4%</td>
</tr>
<tr>
<td>&gt;10 years</td>
<td>7</td>
<td>2</td>
<td>10%</td>
<td>10%</td>
</tr>
<tr>
<td><strong>Asymmetric Septal Hypertrophy</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset (&lt;1 week)</td>
<td>30</td>
<td>3</td>
<td>33%</td>
<td>—</td>
</tr>
<tr>
<td>1 week – 6 months</td>
<td>13</td>
<td>2</td>
<td>22%</td>
<td>36%</td>
</tr>
<tr>
<td>6 months – 1 year</td>
<td>10</td>
<td>1</td>
<td>11%</td>
<td>10%</td>
</tr>
<tr>
<td>1–5 years</td>
<td>9</td>
<td>3</td>
<td>33%</td>
<td>11%</td>
</tr>
<tr>
<td>5–10 years</td>
<td>3</td>
<td>0</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>&gt;10 years</td>
<td>0</td>
<td>0</td>
<td>0%</td>
<td>—</td>
</tr>
</tbody>
</table>

*This column does not include six patients with isolated mitral valve disease and two patients with asymmetric septal hypertrophy who had a history of atrial fibrillation but in whom the time of onset of this arrhythmia could not be determined.
†Number of patients at risk in each time period is defined as the number of patients in atrial fibrillation who had reached the beginning of the particular time period without having embolized.
‡Computed using the method described in reference 25.

three of 32 (9%) patients. Although the number of patients in atrial fibrillation with asymmetric septal hypertrophy who embolized is relatively small, a reasonable estimate of the embolic risk probably can be determined for those patients who were in atrial fibrillation for up to five years. Our data do not permit an accurate estimation of the risk of embolization in patients with asymmetric septal hypertrophy after five years, however, because only three patients have been followed for longer than five years who have not as yet embolized. Whether this is related to the poor prognosis of patients with ASH who develop atrial fibrillation is unclear.

The relation between the occurrence of a systemic embolus and either type of mitral valve lesion (for patients with mitral valve disease) or severity of left ventricular outflow obstruction (for patients with asymmetric septal hypertrophy) is shown in Table 2. Although the percentages in Table 2 suggest that patients with either mitral stenosis or nonobstructive ASH have an increased prevalence of systemic emboli, the number of patients in each group is relatively small and no statistically significant difference was found among the various groups of patients.

Cardioversion and Embolic Events

Cardioversion was attempted 31 times preoperatively in 24 patients with isolated mitral valve disease; in these patients, no embolic events occurred within 48 hours of cardioversion. In 13 patients with asymmetric septal hypertrophy, cardioversion was attempted 23 times preoperatively: embolic events occurred within 24 hours of cardioversion in two of 13 (15%) patients (and following two of 23 [9%] attempts). In addition, another of the 13 patients with asymmetric septal hypertrophy had an embolus when DC cardioversion was attempted nine months postoperatively. This patient twice had been unsuccessfully cardioverted preoperatively and atrial fibrillation persisted after a successful myotomy. Thus, a total of three patients (23%) with ASH had embolic events in association with DC cardioversion.

Discussion

The results of the present study support previous suggestions that the presence or absence of atrial fibrillation is closely related to both the degree of left atrial dilatation and the age of patients with isolated mitral valve disease. Our results also demonstrate a similar relation in patients with aortic valve disease and asymmetric septal hypertrophy (ASH), the latter observation indicating that rheumatic involvement of the atrial wall is not the primary etiologic factor in the development of atrial fibrillation. In addition, the data indicate that a quantitative estimation of left atrial size by echocardiography provides a relatively sharp numerical cutoff in left atrial dimension that separates patients in normal sinus rhythm from those with chronic or paroxysmal atrial fibrillation. Thus, atrial fibrillation was rare in patients with a left atrial dimension below 40 mm (3 of 117 or 3%) but common when this dimension exceeded 40 mm (80 of 148 or 54%, P < 0.01). Furthermore, in the subgroup of patients who were 40 years of age or older and who had left atrial dimension 45 mm or greater, atrial fibrillation occurred in 47 of 53 (89%) of those with isolated mitral valve disease, two of five (40%) of those with isolated aortic valve disease, and 21 of 24 (87%) of those with ASH. When patients falling outside this subgroup are considered, atrial fibrillation occurred in only two of 32 (6%) patients with isolated mitral valve disease, 0 of 45 patients with isolated aortic valve disease, and 11 of 106 (10%) patients with ASH.

TABLE 2. Prevalence of Systemic Emboli in Relation to the Specific Type of Hemodynamic Abnormality

<table>
<thead>
<tr>
<th></th>
<th>Pts with AF</th>
<th>Systemic emboli</th>
<th>% of pts with systemic emboli</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mitral Valve Disease</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mitral regurgitation</td>
<td>8</td>
<td>1</td>
<td>12%</td>
</tr>
<tr>
<td>Mixed mitral stenosis &amp; regurgitation</td>
<td>21</td>
<td>9</td>
<td>43%</td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>20</td>
<td>7</td>
<td>35%</td>
</tr>
<tr>
<td><strong>Asymmetric Septal Hypertrophy</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obstructive</td>
<td>19</td>
<td>4</td>
<td>21%</td>
</tr>
<tr>
<td>Provocable</td>
<td>7</td>
<td>1</td>
<td>14%</td>
</tr>
<tr>
<td>Nonobstructive</td>
<td>6</td>
<td>3</td>
<td>50%</td>
</tr>
</tbody>
</table>
The relation between left atrial size and atrial fibrillation was not influenced by the specific type of mitral valve lesion in patients with isolated mitral valve disease or by the severity of left ventricular outflow obstruction for patients with asymmetric septal hypertrophy.

We believe these results have therapeutic application in that it may be possible with echocardiography to identify patients in sinus rhythm who are at high risk of developing atrial fibrillation. Since ten of 81 (12%) patients in the present series who had atrial fibrillation due to either isolated mitral valve disease or asymmetric septal hypertrophy had a history of an embolic event at the onset of the arrhythmia, either prophylactic anticoagulation, antiarrhythmic drugs, or both, might be considered in the management of a patient in normal sinus rhythm who appears to be in a subgroup at high risk of developing atrial fibrillation. The information obtained in the present study suggests that measurement of left atrial size by echocardiography may allow such a high risk group to be identified. Obviously, only a prospective trial can determine whether the potential benefit of a reduction in embolic events will be realized, and if it is, whether it will outweigh the hazards of drug therapy. Until such data are available, we presently recommend prophylactic anticoagulation to patients in sinus rhythm with either mitral valve disease or asymmetric septal hypertrophy if their left atrial dimension exceeds 40 mm and they are older than age 35 (fig. 5).

Moreover, although the embolic risk per patient year is greatest during the first six months after onset of atrial fibrillation (as indicated by previous studies on patients with mitral valve disease6, 5, 7), the subsequent risk of an initial embolus is not inconsequential (table I). As a result, we also recommend anticoagulation therapy to a patient with established atrial fibrillation who has mitral valve disease or ASH, regardless of the length of time that atrial fibrillation has been present.

The results of the present study also suggest that there may be a relation between left atrial size and the long-term success or failure of cardioversion. For instance, we found that although most patients achieved sinus rhythm during attempted cardioversion, relapse to atrial fibrillation was common; only six of 24 (25%) patients with isolated mitral valve disease and three of 13 (23%) patients with ASH maintained sinus rhythm for longer than six months (fig. 6), a long-term cardioversion success rate consistent with that of previous studies6, 22, 23 of patients with mitral valve disease. Some investigators have suggested that the long-term success rate of cardioversion is low primarily when atrial fibrillation has been present for a prolonged duration prior to the cardioversion attempt.14, 22, 23 The observation in the present study that none of the five patients with atrial fibrillation present for longer than one year was able to be cardioverted and maintained in sinus rhythm for longer than six months is consistent with that suggestion. However, in those patients in whom cardioversion was attempted within one year of onset of atrial fibrillation, we found that only six of 19 (32%) patients with isolated mitral valve disease and three of 13 (23%) patients with asymmetric septal hypertrophy were able to be cardioverted and maintained in sinus rhythm for at least six months. Since all these patients had a left atrial dimension exceeding 45 mm, it is conceivable that once moderate left atrial dilatation occurs, maintenance of normal sinus rhythm for at least six months following cardioversion is relatively uncommon, even if cardioversion is attempted shortly after the onset of the arrhythmia.

In the 24 patients with mitral valve disease in whom DC cardioversion was attempted, no embolic events occurred, a finding consistent with the low embolic rates (1-3%) previously reported in patients with rheumatic heart disease.14 However, emboli did occur in three of the 13 (23%) patients with ASH when DC cardioversion was performed, despite the fact that two of the three were adequately anticoagulated with warfarin at the time of cardioversion. Thus, patients with ASH appear to be particularly susceptible to embolization following cardioversion. In addition, it appears that patients with asymmetric septal hypertrophy who have a left atrial dimension of 50 mm or greater are a particularly vulnerable group; DC cardioversion was successful in achieving and maintaining sinus rhythm for at least six months in only one of ten (10%) patients in this group, while emboli occurred in three of the ten (30%) patients (three of 20 cardioversion attempts).

The strong association of left atrial size and atrial fibrillation in our study appears to indicate a cause and effect relation, a conclusion suggested by Probst et al. in a recent study of patients with mitral stenosis.13 These investigators argued that if left atrial dilatation is the cause of atrial fibrillation, then left atrial size should be smallest in patients with normal sinus rhythm, largest in patients with chronic atrial fibrillation, and intermediate in size in patients with paroxysmal atrial fibrillation.13 Using a qualitative angiographic assessment of left atrial size, these investigators did not find such a relation and therefore concluded that atrial fibrillation precedes and produces atrial dilatation. In the present study, echocardiography was used in order to obtain a quantitative index of left atrial size. With this more quantitative technique, a stepwise relation between left atrial size and atrial fibrillation was demonstrated. Thus, patients who had a left atrial dimension of 40 mm or less, with rare exception, were in normal sinus rhythm and patients whose left atrial dimension was 55 mm or greater, with rare exception, had chronic atrial fibrillation. Patients with paroxysmal atrial fibrillation had intermediate left atrial dilatation with 19 of 21 (90%) such patients having left atrial dimensions between 40 and 55 mm. Although possible, it seems unlikely that left atrial size in the patients with paroxysmal atrial fibrillation was normal prior to the first occurrence of the arrhythmia and that atrial dilatation was produced in these patients by the relatively short and infrequent paroxysms of atrial fibrillation they had experienced. In this regard, we were able to measure left atrial dimensions in three patients both before and after an episode of paroxysmal atrial fibrillation. In all three patients, no change in left atrial dimension was found. In addition, several patients without a history of atrial fibrillation had moderate left atrial enlargement indicating that atrial dilatation can be produced by a hemodynamic burden alone.

The pathophysiology of atrial fibrillation in patients with valvular heart disease or asymmetric septal hypertrophy that is best supported by our echocardiographic data and that we presently are using as a working hypothesis is outlined in figure 7: a chronic hemodynamic burden initially
LA SIZE (ECHO) & ATRIAL FIB/Henry et al.

produces atrial dilatation (and possibly structural damage to the atrial wall); atrial dilatation in turn increases the likelihood of the development of atrial fibrillation, perhaps through re-entrant mechanisms; once atrial fibrillation is present, atrial dilatation could progress as a consequence of either the continued hemodynamic burden, the loss of atrial systole, or both. Whether the mechanisms outlined in Figure 7 will apply to the pathogenesis of atrial fibrillation that occurs in patients with other abnormalities (for example, thyrotoxicosis or ischemic heart disease) is unclear.

We should point out that we did not routinely perform coronary arteriography in our patients. Hence, it is not possible to exclude coronary artery disease as a possible factor in the pathogenesis of atrial fibrillation in some of our patients, particularly since patients in atrial fibrillation were usually older than 40 years of age. We do not believe, however, that coronary artery disease could have been a major precipitating factor, for several reasons. First, symptoms suggesting coronary artery disease are rare in patients with isolated mitral valve disease. Second, in our experience, most patients with asymmetric septal hypertrophy have normal coronary arteries as evidenced by both angiographic and necropsy findings. And finally, even in the unlikely possibility that many of our patients had unsuspected coronary disease, the prevalence of chronic or paroxysmal atrial fibrillation in coronary disease patients does not nearly approach the almost 90% prevalence of the arrhythmia that was found in patients above age 40 years who also had left atrial dilatation.

In conclusion, the results of the present investigation demonstrate a quantitative relation between atrial size, measured echocardiographically, and the presence or absence of atrial fibrillation. In addition, the data suggest that left atrial size may be an important factor in determining whether cardioversion will be successful in achieving and maintaining sinus rhythm for at least six months. Although these data suggest that the detection of left atrial dilatation by echocardiography may be useful in decisions concerning prophylactic anticoagulation, only a prospective study can determine whether the benefit of prophylactic therapy will outweigh the potential hazards.

References
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W L Henry, J Morganroth, A S Pearlman, C E Clark, D R Redwood, S B Itscoitz and S E Epstein

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