Assessment of Left Atrial Appendage Function by Transesophageal Echocardiography

Implications for the Development of Thrombus

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Background. The predilection of the left atrial appendage (LAA) for thrombus formation has long been known.

Methods and Results. We prospectively studied the two-dimensional echocardiographic and Doppler patterns of LAA function in 82 patients by transesophageal echocardiography. In the 63 patients in sinus rhythm, LAA area was measured during LAA diastole at the onset of the electrocardiographic (ECG) P wave (LAA\textsubscript{max}) and after LAA systole at the ECG R wave (LAA\textsubscript{min}) and LAA ejection fraction was calculated as (LAA\textsubscript{max}−LAA\textsubscript{min})/LAA\textsubscript{max}; peak Doppler velocity was recorded from the LAA outlet. The 58 patients in sinus rhythm without LAA thrombus were grouped according to left atrial size on transthoracic echocardiography; 39 patients had a left atrial size of less than 40 mm (group 1) and 19 had a left atrial size of 40 mm or greater (group 2). Five patients in sinus rhythm had LAA thrombus. In the 19 patients with atrial fibrillation or flutter LAA\textsubscript{max} was measured independent of the ECG; three of these patients had LAA spontaneous contrast, four had thrombus, and one had both. Patients in sinus rhythm without LAA thrombus demonstrated a characteristic pattern of a contractile LAA apex and a noncontractile base with color flow and pulsed Doppler evidence of LAA emptying that coincided with the P wave. Patients in sinus rhythm with LAA thrombus had a mean±SD LAA\textsubscript{max} (8.0±1.5 cm\textsuperscript{2}) larger than that in group 1 (5.0±1.9 cm\textsuperscript{2}) (p<0.01) but not group 2 (6.7±3.1 cm\textsuperscript{2}), LAA\textsubscript{min} (6.5±1.0 cm\textsuperscript{2}) larger than that in both group 1 (2.3±1.5 cm\textsuperscript{2}) and group 2 (4.2±2.7 cm\textsuperscript{2}) (p<0.01), and LAA ejection fraction (17±11%) and LAA velocity (0.24±0.10 m/sec) less than those in both group 1 (55±21% and 0.48±0.24 m/sec, respectively) and group 2 (45±27% and 0.46±0.24 m/sec, respectively) (p<0.01). Patients with atrial fibrillation or flutter with LAA spontaneous contrast and/or thrombus had LAA\textsubscript{max} (10.4±6.6 cm\textsuperscript{2}) greater than that in patients with atrial fibrillation or flutter without LAA contrast and/or thrombus (6.8±3.0 cm\textsuperscript{2}) (p<0.05). The LAA appeared as a static pouch in seven of eight of the former compared with in two of 11 of the latter. When attempted, Doppler demonstrated a recognizable fibrillatory LAA outflow velocity pattern in none of three in the former versus four of seven in the latter group.

Conclusions. We conclude that the LAA has a characteristic pattern of emptying in sinus rhythm. LAA thrombus formation in sinus rhythm and atrial fibrillation is associated with both poor LAA contraction and LAA dilatation. (Circulation 1991;84:223–231)

The predilection of the left atrial appendage (LAA) for thrombus formation has been known since the beginning of this century.\textsuperscript{1} Among 51 cases of mitral stenosis studied at autopsy, Jordan et al\textsuperscript{2} in 1951 reported the presence of left atrial thrombi in 40 patients. In 20 cases (50%) the thrombus was restricted to the LAA, in 14 (35%) thrombi were present in both the LAA and the left atrium, and in six (15%) the thrombus was found only in the left atrium. Shrestha et al\textsuperscript{3} in 1983 reported the surgical findings in 293 patients with mitral stenosis. Left atrial thrombi were found in 51 patients; in 11 the thrombus was confined to the LAA, in 21 thrombi were present in both the LAA and the left atrial cavity, and in 19 the thrombus was found only in the left atrial cavity.

Transthoracic echocardiography detects thrombus in the body of the left atrium with a sensitivity

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ranging from 33% to 60%\(^1,4\) but is less able to detect LAA thrombus. Herzog et al\(^5\) reported the detection of LAA thrombus by transthoracic echocardiography in three patients; in two the findings were confirmed at surgery and in the other at autopsy. Shrestha et al,\(^3\) however, reported that preoperative transthoracic echocardiography did not detect the presence of LAA thrombus observed at surgery in 11 patients with mitral stenosis. Using transesophageal echocardiography (TEE), Aschenberg et al\(^6\) detected LAA thrombus confirmed at surgery in six patients with mitral stenosis; transthoracic echocardiography failed to detect the LAA thrombus in these patients.

LAA thrombus, like left atrial cavity thrombus, is a potent risk factor for stroke. Pathologic evidence of systemic emboli was found in 32 of 51 cases of mitral stenosis.\(^1\) Thrombus in the LAA only accounted for 13 (41%), in both the left atrial cavity and LAA for eight (25%), and in the left atrial cavity only for five (16%). Malone et al\(^7\) examined the left atrium in 63 patients with nonvalvular atrial fibrillation; group 1 consisted of 11 patients with systemic embolus and group 2 of 52 patients without recent systemic embolus. Left atrial thrombus was detected in four (36%) group 1 patients and three (6%) group 2 patients (\(p<0.005\)). All but one of these thrombi were confined to the LAA, and none were detected by transthoracic echocardiography. Mugge et al\(^8\) studied 1,910 patients by TEE and detected LAA thrombus in 68 (3.6%); 19 were in sinus rhythm and the other 49 had atrial fibrillation. Of these 68 patients, 38 (56%) had a history compatible with arterial embolism (16 had nonhemorrhagic strokes, 19 had peripheral embolism, and three had both). Left atrial spontaneous contrast is also associated with an increased incidence of left atrial thrombi and arterial embolic episodes.\(^9\)

The pathogenesis of LAA thrombus has not been specifically studied. It is assumed that thrombus within the body of the left atrium arises from stagnation of the blood secondary to dilatation of the left atrium or poor contraction. There are two potential anatomic reasons for predisposition to thrombus formation in the LAA. First, the LAA is a long, narrow chamber with a narrow tip or apex.\(^10\) Second, the inner surface of the LAA is marked by muscular ridges.\(^10\)

The purpose of this study was to examine the appearance and function of the LAA in health and in various cardiac disease states to elucidate the cause of LAA thrombus formation.

**Methods**

**Patients**

Over 6 months, 118 patients had TEE performed; 82 had visualization of the LAA adequate to permit detailed measurements. Primary indications for TEE were prosthetic valve evaluation (40%), stroke (24%), congenital heart disease (11%), endocarditis (11%), and miscellaneous (12%). The patients' age range was 22–78 years; there were 40 men and 42 women.

**Echocardiography**

TEE was performed using an ATL Ultramark 9 device (Bothell, Wash.) with a single-plane probe.
equipped with a 5-MHz transducer. Sedation with 2–5 mg i.v. diazepam was used in approximately 10% of the patients. The pharynx was anesthetized with topical lidocaine spray, and the probe was introduced into the esophagus with the patient lying on the left side. The LAA was viewed from the basal short axis, with the tip of the probe slightly flexed to observe the whole length and breadth of the LAA.11

In the 63 patients who were in sinus rhythm, the area of the LAA just before the P wave (LAA_{max}) and at or just after the electrocardiographic (ECG) QRS complex at the end of LAA systole (LAA_{min}) was determined by planimetry. The perimeter extended from the top of the limbus between the upper left pulmonary vein along a straight line drawn to the aorta at its shortest point at the base of the LAA (Figure 1). The ejection fraction of the LAA was calculated as \((LAA_{max} - LAA_{min})/LAA_{max}\). Pulsed Doppler interrogation of the outlet of the LAA provided the peak LAA systolic velocity (Figure 2). One representative cardiac cycle was used for each patient in sinus rhythm. Measurements were taken by scanning the videotape after the study was performed and choosing the cycle that had the largest LAA cavity with the clearest display of the LAA endocardium.

In the 19 patients with atrial dysrhythmias (atrial fibrillation in 17, atrial flutter in two) the maximum area of the LAA was obtained independent of the ECG, representing the mean of two or three cardiac cycles. Where possible, a minimum LAA area was also determined by planimetry and pulsed Doppler interrogation of the LAA outlet was performed. These measurements were taken from the videotape after completion of the study using the same guidelines as for patients in sinus rhythm.

Five patients in sinus rhythm exhibited LAA thrombus, and three of them also had LAA spontaneous contrast. Eight patients with atrial dysrhythmias exhibited LAA spontaneous contrast (three), LAA thrombus (four), or both (one). There were two characteristic locations of LAA thrombus (Figure 3): at the LAA apex either alone or with extension into the body of the LAA (three patients in sinus rhythm, two with atrial dysrhythmia) or attached to the lateral LAA wall (two patients in sinus rhythm, three with atrial dysrhythmia).

**Statistical Methods**

Groups were compared using the two-tailed unpaired t test. Values are given as mean±SD in the text.

**Results**

**Sinus Rhythm**

In sinus rhythm, the LAA demonstrated a characteristic pattern of emptying. The apex of the LAA was highly contractile and would usually obliterate, whereas the base or neck of the LAA, which accounts for approximately 30–50% of the length of the LAA, was relatively noncontractile. A definite pulsed
Doppler signal that coincided with LAA systole could be obtained in 42 of the 63 patients in sinus rhythm. Color imaging showed flow leaving the LAA that coincided with the cavity contraction and pulsed Doppler signal (Figure 4).

For the purpose of comparison, the 58 patients in sinus rhythm without LAA thrombus were divided into two groups (Table 1) according to left atrial cavity size on transthoracic echocardiography: less than 40 mm (group 1, 39 patients) or 40 mm or greater (group 2, 19 patients). In the five patients in sinus rhythm with LAA thrombus (Table 2) LAA\textsubscript{max} was larger than that in group 1 (p < 0.01) but not group 2, LAA\textsubscript{min} was larger than that in both groups (p < 0.01), and LAA ejection fraction and peak velocity were less than those in both group 1 and group 2 (p < 0.01) (Figure 5).

**Atrial Dysrhythmia**

*Without thrombus and/or contrast.* In nine (82%) of these 11 patients, the LAA filled during ventricular systole and emptied toward the end of diastole: mean LAA ejection fraction was 18% and the range was 8–41%. In these patients the walls of the LAA could be seen to fibrillate, but each fibrillatory contraction was without sufficient inward movement to account for LAA emptying. Rather, during ventricular filling the left ventricular wall adjacent to the LAA seemed to physically push the medial wall of the LAA inward and upward toward the lateral LAA (which seemed relatively static) to account for LAA emptying (Figure 6). Seven of the nine patients had pulsed Doppler interrogation of LAA outflow; in four of these, there was a characteristic signal (Figure 7) suggesting rapid minimal LAA emptying and filling with fibrillatory contractions (velocities of 0.12, 0.20, 0.25, and 0.45 m/sec). Two of the seven patients demonstrated a prolonged low-velocity outflow (Figure 8), which may represent the Doppler equivalent of passive LAA emptying from left ventricular compression. The two remaining patients of the nine had a virtually akinetic LAA; one of these had pulsed Doppler interrogation that showed no recordable velocity contour.

*With thrombus and/or contrast.* Seven of these eight patients did not demonstrate significant LAA emptying or the characteristic Doppler signal (interrogation attempted in three patients) associated with fibrillatory contraction, and the LAA behaved as a static pouch. The other patient displayed a pattern of passive emptying as in Figure 3; this patient demonstrated a low-velocity outflow as in Figure 8. LAA\textsubscript{max} was greater in this group than in the group with atrial dysrhythmia but no thrombus and/or clot (Table 1).

### Table 1. Echocardiographic and Doppler Measurements in All Patients

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>LAA\textsubscript{max} Area (cm\textsuperscript{2})</th>
<th>LAA\textsubscript{min} Area (cm\textsuperscript{2})</th>
<th>LAA ejection fraction (%)</th>
<th>Peak LAA velocity (m/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus rhythm</td>
<td></td>
<td>Mean±SD</td>
<td>Range</td>
<td>Mean±SD</td>
<td>Range</td>
</tr>
<tr>
<td>Without clot</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LA &lt;40 mm</td>
<td>39</td>
<td>5.0±1.9</td>
<td>2.1–11.9</td>
<td>2.3±1.5</td>
<td>0.4–7.5</td>
</tr>
<tr>
<td>LA ≥40 mm</td>
<td>19</td>
<td>6.7±3.1</td>
<td>2.1–13.0</td>
<td>4.2±2.7</td>
<td>0.5–9.6</td>
</tr>
<tr>
<td>Clot</td>
<td>5</td>
<td>8.0±1.5</td>
<td>5.6–9.5</td>
<td>6.5±1.0</td>
<td>5.4–8.0</td>
</tr>
<tr>
<td>Atrial dysrhythmia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without clot</td>
<td>11</td>
<td>6.8±3.0</td>
<td>2.6–12.8</td>
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<td>...</td>
</tr>
<tr>
<td>Clot</td>
<td>8</td>
<td>10.4±6.6</td>
<td>3.3–21.1</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

LAA, left atrial appendage; LA, left atrial size; Clot, LAA contrast and/or thrombus.
FIGURE 4. Transesophageal echocardiogram with color imaging portraying flow leaving left atrial appendage (LAA) following LAA contraction. Arrow points to small area of high velocity (yellow) just beyond obliterated apex. Red area represents "stroke volume" of LAA contraction in this frame taken just after QRS complex (same patient as in Figure 1). AO, aorta; LA, left atrium.

$\textit{Pollick and Taylor Left Atrial Appendage Function}$

$p<0.01$). Left atrial size did not differ significantly between the groups (55.3±9.1 versus 49.7±13.2 mm).

**Left Atrial Size**

Left atrial cavity size measured by transthoracic echocardiography correlated weakly with LAA size on TEE ($r=0.34, p<0.0001$). LAA max in 15 patients in whom the heart was deemed normal ranged from 2.9 to 5.8 (mean 4.05) cm². Using 6.0 cm² as the upper normal value for LAA max 33 (85%) of 39 patients with a left atrial size of less than 40 mm had LAA max of less than 6.0 cm²; 12 (41%) of 29 patients with a left atrial size of 40 mm or greater had LAA max of less than 6.0 cm². Thus, an enlarged left atrium on transthoracic echocardiography was associated with an enlarged LAA in 59% of patients. Conversely, a normal-sized left atrium was associated with an enlarged LAA in 15% of patients.

Left atrial size was larger (52.1±9 mm) in patients with LAA contrast and/or thrombus than in patients without contrast or thrombus (39.6±11 mm) ($p=0.0002$). However, left atrial size in patients with LAA contrast and/or thrombus was not larger than that in patients with a left atrial size of 40 mm or greater without contrast and/or thrombus (47.5±7 mm) ($p=0.13$).

**Reproducibility**

Two observers, each blinded to the other's results, separately analyzed LAA max and LAA min for 10 patients in sinus rhythm (five patients in group 1 and five patients in group 2). The mean (range) differ-

<p>| Table 2. Patients in Sinus Rhythm With Left Atrial Appendage Thrombus |
|-------------------------|--------------------------|-------------------|-------------------|-----------------|---------------------------|</p>
<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (yr)</th>
<th>Diagnosis</th>
<th>Thrombus</th>
<th>LA size (mm)</th>
<th>Area (cm²)</th>
<th>LAA ejection fraction (%)</th>
<th>Peak LAA velocity (m/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>74</td>
<td>Mitral stenosis</td>
<td>+</td>
<td>46</td>
<td>9.5</td>
<td>7.0</td>
<td>26</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
<td>Mitral stenosis</td>
<td>-</td>
<td>54</td>
<td>7.3</td>
<td>6.1</td>
<td>16</td>
</tr>
<tr>
<td>3</td>
<td>35</td>
<td>Mitral stenosis</td>
<td>-</td>
<td>48</td>
<td>9.0</td>
<td>8.0</td>
<td>11</td>
</tr>
<tr>
<td>4</td>
<td>69</td>
<td>Mitral bioprosthesis stenosis</td>
<td>+</td>
<td>38</td>
<td>8.5</td>
<td>5.9</td>
<td>30</td>
</tr>
<tr>
<td>5</td>
<td>67</td>
<td>Restrictive cardiomyopathy</td>
<td>+</td>
<td>49</td>
<td>5.6</td>
<td>5.4</td>
<td>3</td>
</tr>
</tbody>
</table>

Pt, patient number; LA, left atrium; LAA, left atrial appendage. All patients were female.
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Figure 5. Transesophageal echocardiogram depicting patient in sinus rhythm with left atrial appendage (LAA) thrombus (arrow at LAA apex) before (left) and after (middle) atrial systole. LAA is large and contracts poorly. Weak Doppler signal of LAA systole (0.15 m/sec) is depicted in right panel.

ences for LAAmax, LAAmin, and LAA ejection fraction were 0.6 cm² (0.1–1.5 cm²), 0.8 cm² (0.1–2.3 cm²), and 10.8% (1–24%), respectively. The mean (range) coefficients of variation [(observer 1–observer 2)/observer 1] were 7.9% (1–19%), 24% (5–130%), and 32.9% (3–81%), respectively.

Insufficient cardiac cycles were captured on each patient to quantify interbeat variability. Qualitatively, if the LAA was fully visualized at its maximum size, there appeared to be little interbeat variability in size or contraction for patients in sinus rhythm or with atrial fibrillation.

Discussion

The trabecular LAA is a remnant of the original embryonic left atrium that develops during the third week of gestation. The main smooth-walled left atrial cavity develops later and is formed from an outgrowth of the pulmonary veins. The function of the LAA is unknown, and little work has been performed recently. In studies performed more than 50 years ago, it was speculated that the atrial appendages fill the space that is created within the pericardial sac during ventricular systole as the ventricles eject blood and decrease in size. The appendages passively fill during ventricular systole and then passively empty during ventricular diastole. We saw this phenomenon in 10 patients with atrial dysrhythmia. While the contractile nature of the LAA is common surgical knowledge (G.F.O. Tyers, personal communication), there have been no previous angiographic or detailed echocardiographic studies of the precise timing and extent of the contraction. One case report of a herniated giant LAA describes

Figure 6. Transesophageal echocardiogram demonstrating passive change in left atrial appendage size in patient with atrial fibrillation. Left: Ventricular systole. Small arrow points to closed mitral valve. Right: Ventricular diastole. Small arrow points to open mitral valve and large arrow shows filled left ventricle (LV), which seems to have pushed medial wall of left atrial appendage, thereby partly emptying it. A, left atrial appendage.
vigorously LAA contraction during which the "distal tip would flip in an arch of approximately 3 inches with each systole." The timing with the ECG was not reported.

The results of this current study confirm these previous physiological theories, surgical findings, and a recent case report\textsuperscript{15} and suggest that, in sinus rhythm, the LAA is a highly contractile muscular sac that obliterates its apex during atrial systole. We do not believe that translocation of the LAA during the cardiac cycle is the cause of the LAA's decrease in size because the LAA velocity and color flow in sinus rhythm are concordant with an LAA reduction in size, reflecting true contraction. A decrease in LAA contraction is seen in patients in sinus rhythm with thrombus within the LAA, with a decrease in the LAA ejection fraction from 55% to 18% and a decrease in the peak velocity from 0.48 to 0.24 m/sec.

In patients with atrial fibrillation or flutter without LAA contrast or thrombus, the LAA seems to display passive filling and emptying due to compression from the adjacent left ventricle onto the medial LAA wall. Interestingly, there is a propensity for thrombus to locate on the relatively immobile lateral LAA wall (the LAA wall not adjacent to the left ventricle); this observation provides corroboration for this theory. Aschengberg et al\textsuperscript{6} portrayed three illustrations of LAA thrombus, one at the LAA apex and the other two attached to the lateral wall. A predilection for the lateral wall, however, was not mentioned.

In patients with atrial fibrillation or flutter with LAA contrast and/or thrombus, the LAA invariably behaved as a static pouch. The LAA was larger in these patients than in those without contrast or thrombus (10.4 versus 6.8 cm\textsuperscript{2}, \(p<0.01\)), although there was substantial overlap. These findings on LAA size and function may have particular relevance with reference to the recently published reports on atrial fibrillation and stroke. In those studies, the risk of atrial fibrillation producing stroke and the decrease in stroke risk from warfarin is evident.\textsuperscript{16,17} No high-risk atrial fibrillation group (e.g., based on increased left atrial size), however, has been identified.\textsuperscript{18} The results of this current prospective study suggest that the contraction pattern and size of the atrial appendage may be a risk factor in producing LAA thrombus in patients with atrial fibrillation, just as in patients in sinus rhythm.

Measurement of the LAA is prone to observer variability, as demonstrated in the reproducibility study. This, in part, likely reflects the irregularity of the LAA cavity, the arbitrary definition of the upper border with the left atrium, and some interbeat variability. Maintaining the same landmarks of the left atrial cavity for LAA_{\text{max}} and LAA_{\text{min}} should help to make the measurements as accurate as possible. Also, the simultaneous use of pulsed Doppler and color flow imaging can ensure that the LAA pattern represents contraction and not translocation of the LAA during the cardiac cycle.
In conclusion, this study confirms an earlier case report\(^1\) that the LAA is a highly contractile pump with a pattern of contraction quite distinct from that of the main body of the left atrium. LAA thrombus formation is associated with decreased LAA contraction as well as with LAA dilatation. Our observations suggest that a severely hypokinetic LAA, usually also associated with an enlarged LAA, would predispose to thrombus formation just as left ventricular aneurysm predisposes to thrombus formation. An alternative explanation for this association is that the presence of thrombus could physically impede LAA contraction. This would seem a less likely explanation because the thrombus is usually not large enough to account for the LAA hypokinesis. We therefore conclude that it is not only the distinct anatomy, but also the peculiar physiology, of the LAA that lends itself to disease to a predilection to the formation of thrombus.

Acknowledgments

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\(^1\)Reported by guest on April 16, 2017.
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