Exclusion of Atrial Thrombus by Transesophageal Echocardiography Does Not Preclude Embolism After Cardioversion of Atrial Fibrillation

A Multicenter Study

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Background Transesophageal echocardiography (TEE) has been used recently to detect atrial thrombi before cardioversion of atrial arrhythmias. It has been assumed that embolic events after cardioversion result from embolism of preexisting atrial thrombi that are accurately detected by TEE. This study examined the clinical and echocardiographic findings in patients with embolism after cardioversion of atrial fibrillation despite exclusion of atrial thrombi by TEE.

Methods and Results Clinical and echocardiographic data in 17 patients with embolic events after TEE-guided electrical (n=16) or pharmacological (n=1) cardioversion were analyzed. All 17 patients had nonvalvular atrial fibrillation, including four patients with lone atrial fibrillation. TEE before cardioversion showed left atrial spontaneous echo contrast in five patients and did not show atrial thrombus in any patient. Cardioversion resulted in return to sinus rhythm without immediate complication in all patients. Thirteen patients had cerebral embolic events and four patients had peripheral embolism occurring 2 hours to 7 days after cardioversion.

None of the patients were therapeutically anticoagulated at the time of embolism. New or increased left atrial spontaneous echo contrast was detected in four of the five patients undergoing repeat TEE after cardioversion including one patient with a new left atrial appendage thrombus.

Conclusions Embolism may occur after cardioversion of atrial fibrillation in inadequately anticoagulated patients despite apparent exclusion of preexisting atrial thrombus by TEE. These findings suggest de novo atrial thrombosis after cardioversion or imperfect sensitivity of TEE for atrial thrombi and suggest that screening by TEE does not obviate the requirement for anticoagulant therapy at the time of and after cardioversion. A randomized clinical trial is needed to compare conventional anticoagulant management with a TEE-guided strategy including anticoagulation after cardioversion. (Circulation. 1994;89:2509-2513.)

Key Words • cardioversion • echocardiography • atrial fibrillation • thromboembolism

Recent clinical trials have focused attention on methods of reducing the thromboembolic risk of atrial fibrillation, including antithrombotic therapy and cardioversion. However, cardioversion itself may result in embolism. Although anticoagulant therapy is frequently used in patients undergoing cardioversion, such therapy does not eliminate the risk of embolism and may result in additional cost, delay, and potential morbidity.

Recently, several investigators have proposed the use of transesophageal echocardiography (TEE) to screen patients for atrial thrombi before cardioversion, potentially reducing the thromboembolic risk and requirement for anticoagulation. It has been suggested that cardioversion using TEE without prolonged anticoagulation may be safer than cardioversion using anticoagulation without TEE. However, we have identified 17 patients with embolism after cardioversion despite apparent exclusion of atrial thrombus by TEE. The purpose of this study was to analyze the clinical and echocardiographic characteristics of these patients and to examine the implications for the mechanism of postcardioversion embolism and for the role of TEE in patients undergoing cardioversion.

Methods Investigators from hospitals performing TEE-guided cardioversion were requested to provide information regarding embolic events after electrical or pharmacological cardioversion for atrial fibrillation or atrial flutter in patients screened by TEE. The investigators were identified at recent scientific meetings. All the investigators agreed to contribute information to this study and completed a standard questionnaire for each patient.

Clinical data included demographic information, type, duration, and etiology of the atrial arrhythmia; previous cardio-
### Selected Characteristics of 17 Patients With Embolism After Cardioversion of Atrial Fibrillation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/ Sex</th>
<th>Arhythmia Duration</th>
<th>Etiology</th>
<th>Antithrombotic Therapy</th>
<th>LA, mm</th>
<th>LV Dysfunction</th>
<th>LA SEC</th>
<th>Cardioversion Method</th>
<th>Interval CV Embolism, hours</th>
<th>Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>57/M</td>
<td>1 year</td>
<td>Lone</td>
<td>None</td>
<td>40</td>
<td>Normal</td>
<td>No</td>
<td>Quinidine</td>
<td>2</td>
<td>Right hemiplegia, dysphasia</td>
</tr>
<tr>
<td>2</td>
<td>57/M</td>
<td>4 weeks</td>
<td>Lone</td>
<td>Aspirin</td>
<td>43</td>
<td>Normal</td>
<td>No</td>
<td>DC</td>
<td>48</td>
<td>Hemianopia</td>
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<tr>
<td>3</td>
<td>59/M</td>
<td>3 years</td>
<td>CAD</td>
<td>SC Heparin/ Aspirin</td>
<td>48</td>
<td>Mild</td>
<td>No</td>
<td>DC</td>
<td>99</td>
<td>Brachial embolism</td>
</tr>
<tr>
<td>4</td>
<td>61/M</td>
<td>2 weeks</td>
<td>HT</td>
<td>SC Heparin</td>
<td>49</td>
<td>Normal</td>
<td>No</td>
<td>DC</td>
<td>48</td>
<td>Dysphasia</td>
</tr>
<tr>
<td>5</td>
<td>62/M</td>
<td>13 days</td>
<td>ETOH</td>
<td>IV Heparin</td>
<td>42</td>
<td>Moderate</td>
<td>Yes</td>
<td>DC</td>
<td>4</td>
<td>Dysphasia</td>
</tr>
<tr>
<td>6</td>
<td>63/M</td>
<td>7 days</td>
<td>DCM</td>
<td>Aspirin</td>
<td>NA</td>
<td>Moderate</td>
<td>Yes</td>
<td>DC</td>
<td>24</td>
<td>Femoral embolism</td>
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<tr>
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<td>5 days</td>
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<td>Normal</td>
<td>No</td>
<td>DC</td>
<td>45</td>
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</tr>
<tr>
<td>8</td>
<td>65/F</td>
<td>11 days</td>
<td>HT</td>
<td>IV Heparin/ Warfarin</td>
<td>42</td>
<td>Normal</td>
<td>No</td>
<td>DC</td>
<td>36</td>
<td>Right hemiplegia, dysphasia</td>
</tr>
<tr>
<td>9</td>
<td>66/F</td>
<td>3 weeks</td>
<td>Lone</td>
<td>IV Heparin</td>
<td>49</td>
<td>Mild</td>
<td>No</td>
<td>DC</td>
<td>24</td>
<td>Left hemiparesis</td>
</tr>
<tr>
<td>10</td>
<td>68/M</td>
<td>2 weeks</td>
<td>ETOH</td>
<td>Aspirin</td>
<td>48</td>
<td>Normal</td>
<td>No</td>
<td>DC</td>
<td>30</td>
<td>Left hemiparesis</td>
</tr>
<tr>
<td>11</td>
<td>69/F</td>
<td>2 days</td>
<td>HT</td>
<td>None</td>
<td>NA</td>
<td>Moderate</td>
<td>Yes</td>
<td>DC</td>
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<td>Cerebellar syndrome</td>
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<td>Aspirin</td>
<td>54</td>
<td>Severe</td>
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</tr>
<tr>
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<td>2 weeks</td>
<td>Postop</td>
<td>SC Heparin</td>
<td>58</td>
<td>Normal</td>
<td>Yes</td>
<td>DC</td>
<td>168</td>
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</tr>
<tr>
<td>14</td>
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<td>COPD</td>
<td>None</td>
<td>45</td>
<td>Normal</td>
<td>No</td>
<td>DC</td>
<td>39</td>
<td>Brachial embolism</td>
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<tr>
<td>15</td>
<td>84/F</td>
<td>6 weeks</td>
<td>Lone</td>
<td>SC Heparin</td>
<td>33</td>
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<td>No</td>
<td>DC</td>
<td>24</td>
<td>Left monoplegia</td>
</tr>
<tr>
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<td>87/F</td>
<td>12 days</td>
<td>HT</td>
<td>None</td>
<td>55</td>
<td>Normal</td>
<td>No</td>
<td>DC</td>
<td>9</td>
<td>Left hemiparesis</td>
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<tr>
<td>17</td>
<td>88/F</td>
<td>8 days</td>
<td>COPD</td>
<td>SC Heparin</td>
<td>58</td>
<td>Normal</td>
<td>No</td>
<td>DC</td>
<td>98</td>
<td>Mesenteric embolism</td>
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</tbody>
</table>

Antithrombotic therapy refers to the time of cardioversion. CAD indicates coronary artery disease; COPD, chronic obstructive pulmonary disease; CV, cardioversion; DCM, dilated cardiomyopathy; ETOH, alcohol; HT, hypertension; LA, left atrium; LV, left ventricle; NA, not available; Postop, postoperative; and SEC, spontaneous echo contrast.

version attempts; coexisting cardiac diseases; thromboembolic risk factors and previous embolic events; and antithrombotic and antiarrhythmic medications. Nonvalvular atrial fibrillation refers to the absence of mitral stenosis or any valve prothesis.15 Lone atrial fibrillation refers to the absence of a known predisposing cause including cardiac disease, hypertension, or diabetes.16 Duration of arrhythmia refers to the current episode of arrhythmia. Antithrombotic therapy was determined by the treating physician and did not follow a uniform protocol. Therapeutic anticoagulation was defined as International Normalized Ratio (INR) ≥2.0 for patients receiving warfarin, activated partial thromboplastin time ≥1.5× control for patients receiving heparin, and anti-Factor Xa ≥0.5 IU/mL for patients receiving low-molecular-weight heparin (dalteparin sodium, Fragmin).

Echocardiographic data comprised transducer type and frequency, left ventricular systolic function, left atrial diameter, mitral valve stenosis and regurgitation, left and right atrial spontaneous echo contrast and thrombus, and other potential cardiac sources of embolism. Transthoracic echocardiography was performed with commercially available 2.25- to 3.5-MHz transducers. TEE was performed with standard techniques17 using commercially available, 5-MHz biplane (n=8), singleplane (n=8), or multiplane (n=1) transducers. There were no complications of TEE. The left atrial cavity and appendage were visualized by TEE in all patients. Left atrial spontaneous echo contrast refers to dynamic, smokelike echoes with a characteristic swirling motion, distinct from echoes caused by excessive gain.18 Spontaneous echo contrast was graded as mild or severe.19 Atrial thrombus refers to the presence of a clearly defined intracavitary mass acoustically distinct from underlying endocardium and not caused by the pectinate ridges of the atrial appendage.20 Left atrial appendage Doppler flow velocities were not routinely determined.

Cardioversion data collected included interval between TEE and cardioversion, cardioversion method, number and strength of DC shocks, nonembolic complications, and recurrence of atrial fibrillation after cardioversion. Data regarding embolic events included interval between cardioversion and embolism, clinical description of event, results of investigations, clinical management, and outcome. Cardioversion-related embolism was defined as any clinically evident acute cerebral or systemic ischemic event or pulmonary embolism within 1 month after cardioversion.

### Results

#### Clinical Characteristics

Clinical and echocardiographic characteristics of all 17 patients with embolism after TEE-guided cardioversion are summarized in the Table. The 17 patients represent 2.4% of the 712 patients screened by TEE before cardioversion at the participating centers. The patients underwent cardioversion in 1988 (n=2), 1991 (n=9), 1992 (n=5), and 1993 (n=1). There were 11 men and 6 women aged 68±13 years. All patients had nonvalvular atrial fibrillation, including 4 patients with lone atrial fibrillation. The duration of arrhythmia was 2 days to 3 years. Seven patients had congestive heart failure, 7 patients had hypertension, and 2 patients had diabetes mellitus. Four patients had undergone previous cardioversion without embolism. Two patients had previous embolism. No patient had undergone cardiac

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surgery or recent (≤1 month) noncardiac surgery. Eleven patients were receiving class I or III antiarrhythmic therapy; amiodarone (n=4), procainamide (n=3), sotalol (n=3), flecainide (n=2), and quinidine (n=1). Four patients were considered to have clinical contraindications to anticoagulant therapy.

**Echocardiography**

Transthoracic echocardiography did not detect mitral stenosis, intracardiac thrombus, spontaneous echo contrast, or other source of embolism in any patient. Left ventricular systolic function was normal in 11 patients and impaired in 6 patients. Two patients had left ventricular hypertrophy. Mean left atrial diameter was 47±7 mm.

TEE did not detect left or right atrial thrombus or other intracardiac thrombus or masses in any patient. Left atrial spontaneous echo contrast was detected in the left atrial cavity and appendage in 4 patients and in the left atrial cavity alone in 1 patient and was graded as mild intensity in all 5 patients. Other potential sources of embolism included patent foramen ovale (n=2), protruding aortic atheroma (n=1), and mitral annular calcification (n=1). Seven patients had at least mild (≥1+) mitral regurgitation.

Antithrombotic therapy at the time of TEE comprised no therapy (n=7); aspirin (n=4); intravenous heparin (n=3), including 1 patient also receiving warfarin; subcutaneous dalteparin (low-molecular-weight heparin) (n=2), including 1 patient also receiving aspirin; and subcutaneous heparin (n=1). The duration of anticoagulant therapy in the 6 patients receiving heparin and/or warfarin was 1 to 11 days. Only 3 of the 17 patients were therapeutically anticoagulated at the time of TEE.

**Cardioversion**

Sixteen patients underwent electrical cardioversion using synchronized DC shocks. Electrical cardioversion was performed within 24 hours after TEE in 13 patients and 7 to 21 days after TEE in 3 patients. Patients received 2.2±1.3 shocks (range, 1 to 6), with a maximum single shock of 227±100 J (range, 100 to 360) and cumulative of 419±356 J (range, 100 to 1420). All 16 patients were successfully cardioverted to sinus rhythm. The single patient receiving pharmacological cardioversion commenced quinidine 3 hours after TEE and reverted to sinus rhythm 19 hours after TEE. There were no immediate complications of cardioversion in the 17 patients. Three patients reverted to atrial fibrillation within 24 hours after cardioversion.

Antithrombotic therapy at the time of cardioversion comprised no therapy (n=5); aspirin (n=4); intravenous heparin (n=3), including 1 patient also receiving warfarin; subcutaneous heparin (n=3); and subcutaneous dalteparin (n=2), including 1 patient also receiving aspirin. Only 2 of the 17 patients were therapeutically anticoagulated at the time of cardioversion.

**Embolic Events**

Thirteen patients had cerebral embolic events after cardioversion, and 4 patients had brachial (n=2), femoral (n=1), or mesenteric (n=1) embolism. Embolism occurred 2 hours to 7 days (mean, 46±42 hours) after cardioversion. ECG after embolism showed that 13 patients were in sinus rhythm and 4 patients had reverted to atrial fibrillation. The cerebral embolic events were classified as cerebrovascular accident in 9 patients and transient ischemic attack in 4 patients. Cerebral computed tomography scanning was performed in 12 of 13 patients with cerebral embolism and showed cerebral infarction in 8 patients and normal findings in 4 patients. Carotid duplex scanning in 7 patients showed no significant (>50%) stenosis. The 13 patients with cerebral embolism were treated with intravenous heparin (n=9), aspirin (n=3), or no antithrombotic therapy (n=1). The 4 patients with limb or mesenteric embolism were treated with surgical thromboembolectomy (n=3) or intravenous heparin (n=1). Eleven of the 17 patients returned home, 4 patients were transferred to nursing homes, and 2 patients died.

Antithrombotic therapy at the time of embolism comprised no therapy (n=6); aspirin (n=5); intravenous heparin (n=3), including 1 patient also receiving warfarin; subcutaneous dalteparin (n=2), including 1 patient also receiving aspirin; and subcutaneous heparin (n=1). None of the 17 patients was therapeutically anticoagulated at the time of embolism.

**Echocardiography After Cardioversion**

Five patients underwent repeat TEE 0 to 96 hours after cardioversion. Left atrial spontaneous echo contrast was detected in 4 of the 5 patients. In all 4 patients, spontaneous echo contrast was not previously present (n=2) or showed markedly increased intensity (n=2) compared with the precadioversion study. One patient with mild left atrial spontaneous echo contrast without thrombus at the precadioversion study had increased spontaneous echo contrast and a fresh thrombus adja- cent to the lateral wall of the left atrial appendage detected by TEE 4 days after embolism.

**Discussion**

Embolism represents an uncommon but feared complication after cardioversion of atrial arrhythmias. Although prolonged anticoagulation before and after cardioversion has been recommended, such therapy is not universally used, particularly in patients with recent-onset or postoperative atrial fibrillation. This may reflect the absence of randomized trials, the occurrence of embolism despite anticoagulation, and the risk of bleeding, and additional cost, inconvenience, and delay.

Recently, investigators have used TEE to screen for atrial thrombi before cardioversion of atrial arrhythmias. The rationale for TEE is based on two assumptions. Embolism after cardioversion has been assumed to result from propulsion of preexisting atrial thrombus into the circulation. It also has been assumed that TEE is a highly sensitive method of detecting atrial thrombus. The finding in the present study that embolism may occur despite apparent exclusion of preexisting atrial thrombus by TEE requires that these assumptions be reassessed.

**Characteristics of Patients With Embolism**

Patients in the present series featured a spectrum of risk factors for embolism. The Stroke Prevention in Atrial Fibrillation (SPAF) investigators identified five clinical or echocardiographic predictors of increased thromboembolic risk in nonvalvular atrial fibrillation, the arrhythmia present in all patients in the present series. The clinical risk factors, congestive heart failure,
previous embolism, and hypertension, were present in 11 patients in the present series. Six patients in this series had left ventricular dysfunction, and most patients had left atrial dilation. However, the series included 4 patients with lone atrial fibrillation, which is associated with low embolic risk. We also assessed the presence of left atrial spontaneous echo contrast detected by TEE. This echocardiographic phenomenon reflects atrial stasis and altered systemic hematoletic parameters, thereby reflecting two arms of Virchow’s triad of factors influencing thrombosis, and is a marker of left atrial thrombus and previous embolism in patients with nonvalvular atrial fibrillation. Spontaneous echo contrast was detected before cardioversion in only 5 patients, suggesting that this finding has limited predictive value for embolism in patients undergoing cardioversion.

The most characteristic feature of patients in this series is inadequate anticoagulation. No patient was therapeutically anticoagulated at the time of embolism, and no patient received prolonged anticoagulation before cardioversion. Manning et al recently used TEE to avoid prolonged anticoagulation before cardioversion. Although not all patients in that study received anticoagulants at the time of and after cardioversion, no embolic events were detected (95% confidence interval, 0% to 4.6%). It was suggested that cardioversion using TEE without prolonged anticoagulation may be safer than cardioversion using anticoagulation without TEE. The present series shows that screening by TEE without adequate anticoagulation does not preclude the risk of embolism and supports the use of anticoagulant therapy at the time of and for a period after cardioversion. This series also suggests that aspirin and/or subcutaneous heparin may not provide adequate protection against embolism in the cardioversion setting.

Detection of Atrial Thrombus by TEE

The rationale for TEE-guided cardioversion assumes that TEE is an accurate method for the detection of atrial thrombus. Could TEE have missed preexisting left atrial thrombi in the present series? Five studies in 389 patients have shown an overall 92% sensitivity and 98% specificity, compared with surgical findings, for detection of left atrial thromb by TEE in patients with mitral valve disease. The imperfect sensitivity of TEE may reflect the complex three-dimensional structure of the left atrial appendage. The sensitivity of TEE may be further reduced in patients with nonvalvular etiology, in whom thrombi are typically smaller and located in the left atrial appendage rather than the main atrial cavity. Several patients in the present series had single-plane TEE imaging only. Biplane or multiplane imaging improves visualization of the atrial appendage and may improve the diagnostic accuracy of TEE for thrombus. Although most patients in the present series underwent cardioversion shortly after TEE, intervals up to 21 days occurred. Thrombus may have formed in these patients between TEE and cardioversion.

Although missed thrombi may in part account for the embolic events observed, several factors suggest that this is an incomplete explanation. The left atrial appendage and cavity were visualized in all patients and were assessed by echocardiographers with considerable expertise in TEE. The majority of patients were studied shortly before cardioversion by biplane or multiplane TEE imaging. Left atrial spontaneous echo contrast was infrequent before cardioversion. Nevertheless, this series highlights both the potential limitations and the importance of diagnostic accuracy when TEE is used to screen patients for atrial thrombi.

Mechanisms of Embolism After Cardioversion

It has been assumed that postcardioversion embolism results from dislodgment of preexisting atrial thrombus after the return of mechanical atrial contraction. However, recent studies have suggested an alternate mechanism for thromboembolism. Grimm et al performed TEE immediately before and after successful electrical cardioversion of atrial fibrillation. Left atrial spontaneous echo contrast developed de novo or increased after cardioversion in 35% of the patients and was associated with a decrease in blood flow velocity in the left atrial appendage. Faktin et al reported new or increased left atrial spontaneous echo contrast after cardioversion in 40% of patients studied. The pathogenesis of this apparently paradoxical deterioration in atrial appendage mechanical function after cardioversion despite reversion to sinus rhythm is not well understood. Nevertheless, these recent studies suggest an alternate mechanism for embolism in which persistent or increased atrial stasis after cardioversion results in the formation of fresh, loosely adherent thrombus and subsequent embolism.

The findings of the present study support the hypothesis that cardioversion may result in a thrombogenic milieu. New or increased left atrial spontaneous echo contrast after cardioversion was found in 4 of the 5 patients in whom it was assessed, including 1 patient with de novo thrombosis documented by TEE. It is apparent that even if TEE were a perfect test for atrial thrombus, screening before cardioversion would not preclude all cases of postcardioversion embolism. The finding of residual thrombus in only 1 of 5 patients is consistent with previous reports and presumably reflects complete embolization of loosely adherent thrombus. The remaining 12 patients were not restudied after embolism, leaving it uncertain as to whether de novo atrial thrombosis occurred.

Conversely, these findings do not preclude a role for preexisting atrial thrombus in the pathogenesis of postcardioversion embolism. Detection of preexisting atrial thrombus by TEE presumably identifies a subgroup of patients with increased risk for embolism in whom cardioversion can be deferred and antithrombotic therapy can be commenced or continued.

Although not all embolic events during atrial fibrillation result from left atrial thrombi, only 4 patients in the present series had a potential source of embolism other than the left atrium. The four patients with recurrent atrial fibrillation also illustrate the probable heterogeneity of embolism related to cardioversion. One patient had embolism after quinidine cardioversion. Previous reports suggest that the risk of embolism after pharmacological and electrical cardioversion is similar.

Study Limitations

The precise incidence of embolism after TEE-guided cardioversion cannot be determined from the present study, which comprises a selected patient population. Nevertheless, the study demonstrates that embolic events still may occur despite screening by TEE.
tailed left atrial and appendage mechanics were not assessed, and not all patients underwent repeat TEE after cardioversion. We cannot be certain whether emboli occurred because of preexisting thrombi missed by TEE or de novo atrial thrombosis resulting from atrial stasis after cardioversion.

Clinical Implications

This study has shown that exclusion of preexisting atrial thrombus by TEE in inadequately anticoagulated patients does not abolish the risk of embolism after cardioversion of atrial fibrillation. The study provides clinical correlation for recent mechanistic studies suggesting that persistent or increased left atrial stasis after cardioversion may result in a thrombogenic milieu. These findings suggest that a negative TEE for thrombus may not obviate the requirement for anticoagulant therapy during and after cardioversion. Small left atrial appendage thrombus missed by TEE also may have contributed to the embolic events observed. It therefore remains to be determined whether screening by TEE removes the need for precardioversion anticoagulation and whether screening by TEE decreases the embolic risk in patients receiving conventional anticoagulant therapy. A randomized clinical trial, the Assessment of Cardioversion Utilizing Transesophageal Echocardiography (ACUTE) Study, is under way to assess the potential benefits and limitations of TEE in preventing thromboembolism associated with cardioversion.

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References

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