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Transesophageal Echocardiographically Facilitated Early Cardioversion From Atrial Fibrillation Using Short-Term Anticoagulation: Final Results of a Prospective 4.5-Year Study

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Objectives. **We sought to validate the safety of transesophageal echocardiographically guided early cardioversion in conjunction with short-term anticoagulation as a strategy for guiding early cardioversion in hospitalized patients with atrial fibrillation.**

Background. **Because atrial thrombi are poorly seen by conventional imaging techniques, several weeks of prophylactic anticoagulation is routinely prescribed before cardioversion. Transesophageal echocardiography is a superior test for identifying atrial thrombi; preliminary feasibility studies have supported its use to guide early cardioversion for patients in whom no thrombus is observed, but safety has not been validated in any large series.**

Methods. **All patients admitted to hospital with atrial fibrillation during a 4.5-year period were screened. The inclusion criterion was a clinical duration of atrial fibrillation >2 days or of unknown duration. Patients received anticoagulation with heparin/warfarin and underwent conventional transthoracic echocardiography followed by transesophageal study. Patients in whom transesophageal echocardiography revealed no atrial thrombus underwent pharmacologic or electrical cardioversion followed by warfarin therapy for 1 month. Cardioversion was deferred in patients with evidence of atrial thrombi, and they received prolonged warfarin treatment.**

Results. **Two hundred thirty-three patients (86% of those eligible) agreed to participate, and 230 underwent transesophageal echocardiography. Transesophageal echocardiography identified 40 atrial thrombi (left atrium 34, right atrium 6) in 34 patients (15%). One hundred eighty-six (95%) of 196 patients without thrombi had successful cardioversion to sinus rhythm, all without prolonged anticoagulation, and none (0%, 95% confidence interval 0% to 1.6%) experienced a clinical thromboembolic event. Eighteen patients with atrial thrombi underwent uneventful cardioversion after prolonged anticoagulation.**

Conclusions. **Compared with smaller series that have shown only feasibility, this large prospective and consecutive study of patients undergoing transesophageal echocardiographically facilitated early cardioversion in conjunction with short-term anticoagulation validates the safety of this strategy. This treatment algorithm has a safety profile similar to conventional therapy and minimizes both the period of anticoagulation and the overall duration of atrial fibrillation.**

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Thromboembolism as a consequence of atrial fibrillation is a major cause of morbidity and mortality. Cardioversion from atrial fibrillation is commonly used both to prevent thromboembolism (1) and to improve left ventricular function (2–5). However, up to 7% of patients undergoing cardioversion without anticoagulation experience clinical thromboembolism (6–9), presumably as a result of dislodgment of an atrial thrombus after resumption of atrial systolic activity. Several weeks of warfarin therapy before cardioversion has been shown to reduce the incidence of cardioversion-associated thromboembolism by 0% to 1.6% (8–10). This recognized benefit of prophylactic anticoagulation before cardioversion has led to its adoption as the current standard of care for patients with atrial fibrillation of >2 days in duration (11,12), although no large randomized trials have been reported. However, the use of several weeks of anticoagulation is associated with significant adverse effects, most notably an increased risk of hemorrhage (8,13). This treatment strategy also results in a 1-month delay in cardioversion for the great majority of patients who would not experience thromboembolism.

Until the development of two-dimensional transthoracic echocardiography, identification of intracardiac thrombi was rarely possible except at cardiac surgery or autopsy. Although conventional transthoracic echocardiography may identify thrombi in the body of the atria, it poorly visualizes the left

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atrial appendage, the site of most thrombi in patients with atrial fibrillation (14,15). A superior test for atrial thrombi would obviate the need for several weeks of precardioversion anticoagulation in patients found to be free of thrombus, thereby abbreviating the total duration of anticoagulation, allowing earlier return of sinus rhythm and atrial mechanical function (16) and eliminating the need for a second hospital admission for cardioversion.

Because of its excellent visualization of posterior cardiac structures, transesophageal echocardiography has rapidly become the procedure of choice for the detection of left atrial pathology, including left atrial tumor, atrial septal defect, patent foramen ovale and left atrial thrombus (17–19). The procedure has proved well tolerated and safe in both young adult and elderly patients (20,21). We and others (22–25) have been investigating the use of transesophageal echocardiography as a tool to facilitate cardioversion in patients with atrial fibrillation. These preliminary studies (22–25) have demonstrated the feasibility of the transesophageal echocardiographic approach, but the small number of patients in each series has limited validation of its safety. We report the results of a large, prospective study of a consecutive group of patients over a 4.5-year period who participated in a transesophageal echocardiographic guided strategy for early cardioversion, which now validates the safety of this approach.

Methods

Study patients. We screened 1,299 consecutive adult patients admitted (or referred to the echocardiography laboratory) to the Beth Israel Hospital from January 1, 1990 through July 31, 1994 or to the University of Connecticut Health Center from January 1, 1991 through July 31, 1994 with a diagnosis that included atrial fibrillation. Patients were excluded if they were receiving long-term anticoagulation with warfarin (and had maintained a therapeutic prothrombin time ≥15 s), if their clinically estimated duration of atrial fibrillation was <2 days, if they spontaneously reverted to sinus rhythm before we obtained informed consent, if cardioversion was not indicated or desired or if transesophageal echocardiography was contraindicated (esophageal stricture, n = 1) (26). Patients with previous or acute thromboembolic events were not excluded. Of the 270 patients who qualified, 37 declined to participate, 23 at their primary physician's request. The 233 patients who agreed to participate include 94 described in a preliminary report (22).

Study protocol (Fig. 1). After obtaining written informed consent, we began therapeutic heparin anticoagulation in 211 patients (91%) using an intravenous heparin bolus (3,000 to 5,000 U) and continuous heparin infusion to maintain a partial thromboplastin time of 1.4 to 1.9 times baseline. Twenty-two patients were believed (decision of their primary physicians) not to be candidates for short-term heparin therapy because of recent or previous gastrointestinal bleeding (n = 13), advanced age (n = 7) or hemorrhagic stroke (n = 2). So as not to delay hospital discharge we also initiated warfarin in 163 patients (70%) considered suitable by their primary physicians for postcardioversion anticoagulation, with a target prothrombin time of 1.4 to 1.6 times baseline.

Conventional transthoracic examination was initially performed in all subjects. Two-dimensional imaging and Doppler transthoracicstudies were obtained using an HP Sonos 500, 1000 or 1500 (Hewlett-Packard Co.) combined imaging/Doppler echocardiograph equipped with a 2.0-, 2.5- or 3.5-MHz phased-array transducer. M-mode left atrial dimension was measured in the parasternal long-axis view (27). The extent of mitral regurgitation was assessed by pulsed and color Doppler and graded as 0 (none) to 3 (severe) (28). Left ventricular systolic function was defined as abnormal if there was evidence of global or regional hypokinesis.

If no definite thrombus was seen on transthoracic study, transesophageal echocardiography was performed within 48 h after at least a 6-h fast. Patients received pharyngeal anesthesia with 10% lidocaine or Cetacaine spray (Cetylite Industries, Inc.) and mild sedation with intravenous meperidine, midazolam, fentanyl or morphine sulfate, as necessary. Prophylactic antibiotics were administered to patients with substantial valvular abnormalities (29). Transesophageal echocardiography was attempted using a commercial 5.0-MHz single-plane (n = 50), biplane (n = 95) or multiplane probe (n = 88). The left atrial appendage was initially viewed in the horizontal (0°) plane with the tip of the probe slightly flexed and with the probe gently withdrawn until the bifurcation of the pulmonary artery was identified. Initial imaging of the left atrial appendage in the vertical (90°) plane was followed by posterior and anterior rotation of the probe until the coronary sinus and aorta were visualized, respectively. Multiplane imaging was accomplished at 0° and 90° as described earlier, followed by imaging of the left atrial appendage in the horizontal (0°) plane and rotation of the imaging sector from 0° to 180° during continuous visualization of the appendage. An atrial thrombus was defined as a well circumscribed and uniformly consistent echo-reflective mass of different texture than the atrial wall (30). Spontaneous echo contrast, a marker of blood stasis, was considered present when dynamic "smokelike" echoes were detected within the atria (31). Imaging and Doppler data were recorded on videotape for consensus review by at least two experienced observers.

If no atrial thrombi were seen, cardioversion was attempted chemically in patients who had received heparin anticoagulation using either quinidine, procainamide, disopyramide, flecainide or amiodarone (choice of their primary physician). If sinus rhythm was not achieved within 12 to 36 h, direct current cardioversion was performed (three patients refused electrical cardioversion after unsuccessful chemical cardioversion). Heparin was continued for at least 24 h after cardioversion in those who had received no warfarin, and until the prothrombin time was therapeutic for those who had received warfarin. Warfarin was continued for 1 month after cardioversion.

Because of the concern regarding thrombus development during the interval between transesophageal echocardiography

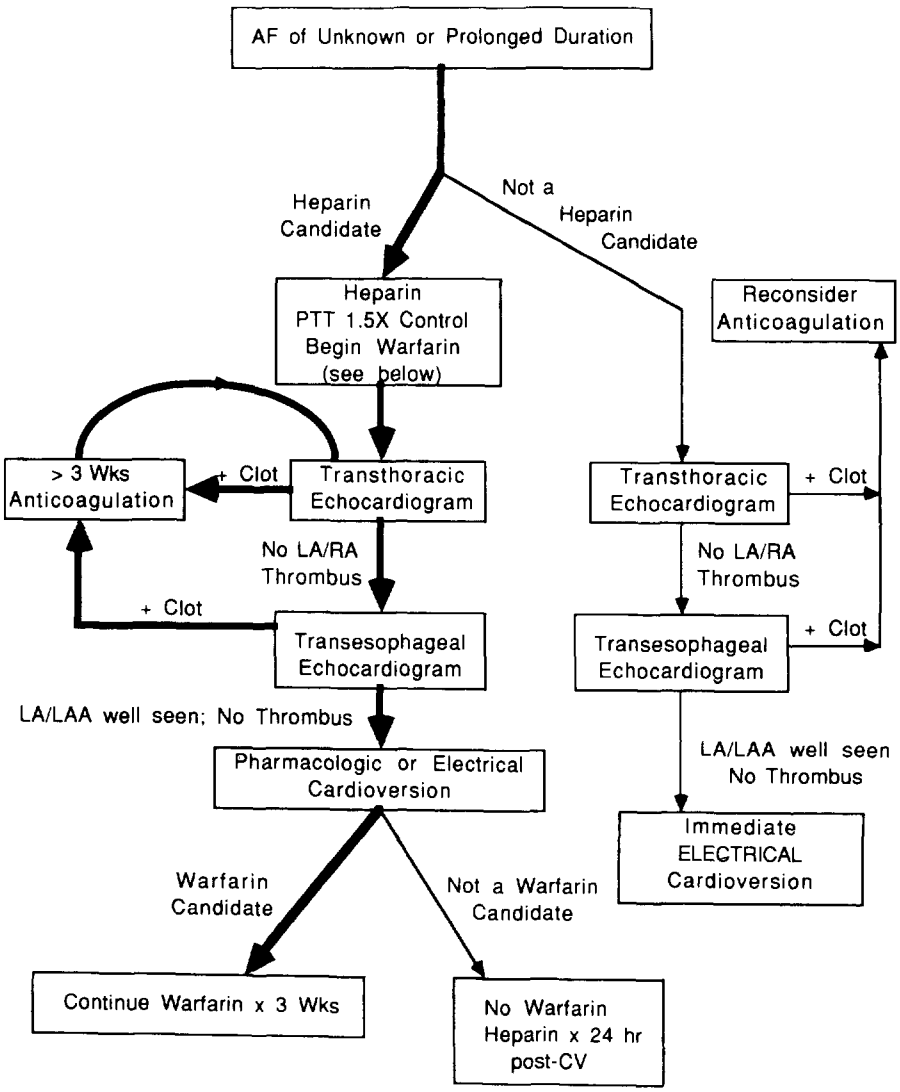

Figure 1. Schematic of the transesophageal echocardiographic protocol. AF = atrial fibrillation; CV = cardioversion; LA = left atrial; LAA = left atrial appendage; PTT = partial thromboplastin time; RA = right atrial.

and cardioversion, patients who did not receive heparin anticoagulation underwent electrical cardioversion immediately after transesophageal echocardiography.

Patients in whom an atrial thrombus was identified received >3 weeks of warfarin therapy with a target prothrombin time of 1.5 to 1.8 times baseline. Repeat transesophageal echocardiography was then recommended, with cardioversion if no thrombus was visualized on the follow-up study.

All patients were followed up daily during their hospital stay and by office visit or telephone call at 3 to 4 weeks after cardioversion. No patient was lost to follow-up. The study was approved by the Institutional Review Boards of both hospitals, and informed consent was obtained from all participants.

Statistical analysis. Results are expressed as mean value ± 1 SD. Categoric variables between groups were compared using the Fisher exact test, and continuous variables were compared using analysis of variance. Severity of mitral regurgitation was analyzed both as a continuous variable (0 to 3) and as a dichotomous variable (less than moderate or moderate to severe) (StatView II, Abacus Concepts). In all cases a two-tailed test was performed, with $p \leq 0.05$ considered significant. Confidence intervals for the percent of patients who might experience an embolic event after a negative transesophageal echocardiogram were calculated using the method of Hanley and Lippman-Hand (32).

Results

The 233 patients who agreed to participate included 105 men and 128 women (mean [±SD] age 73 ± 13 years, range 30 to 98). The underlying disorder predisposing to atrial fibrillation included one or more of the following: hypertension (n = 116 [50%]), ischemic heart disease (n = 56 [24%]), rheumatic mitral valve disease (n = 17 [8%]), alcoholism (n = 10 [4%]), pneumonia (n = 4 [2%]) and pulmonary embolism (n = 2 [1%]). In 59 patients (25%) no predisposing condition could be identified. The clinically estimated duration of atrial fibrillation was 3.4 ± 6.0 weeks, with a distribution as shown in Figure

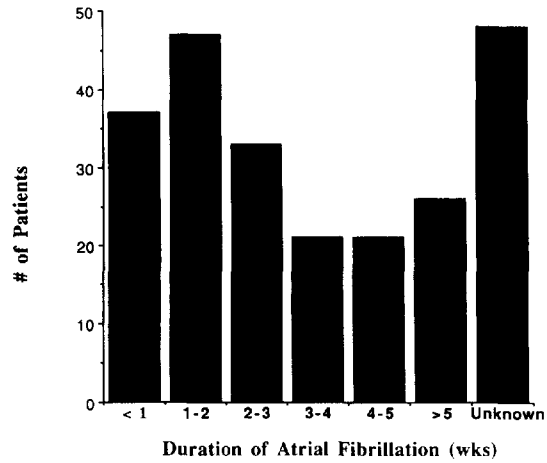

Figure 2. Histogram depicting duration of atrial fibrillation before admission to hospital for the 233 study participants.

2. The duration of atrial fibrillation was clinically indeterminate in 48 patients (21% of the total). Thirty-five patients (15%) had a history of recent or distant clinical thromboembolism. Echocardiography.

Echocardiography. Two hundred thirty-three subjects underwent transthoracic echocardiography, followed by uncomplicated transesophageal echocardiography in 230. Transesophageal echocardiography could not be performed in three patients (1.3%) because of inability to intubate the esophagus (subsequent results of barium swallows were all normal). These patients were treated with conservative therapy (3 weeks of warfarin before cardioversion), and their data were not included in the subsequent analysis. Transesophageal echocardiographic study and well defined left atrial and left atrial appendage borders were identified in all remaining subjects. The right atrial appendage was not adequately seen in three subjects (1.3%), all of whom had imaging with single-plane transesophageal probes.

Five atrial thrombi were suspected by transthoracic echocardiography (one right, four left). Of these only two left atrial thrombi were confirmed at transesophageal study (sensitivity of transthoracic echocardiography for left atrial thrombi 6%; specificity 99%; positive predictive value 50%; negative predictive value 86%).

Forty atrial thrombi (34 left atrium, 6 right atrium) were identified by transesophageal echocardiography in 34 patients (15% of the total group). Ninety-five percent of thrombi were visualized only by transesophageal echocardiographic study. Thrombus size varied from 2 to 20 mm (maximal length). All six right atrial thrombi and all but one left atrial thrombus were confined to or extended into their respective appendages. Two left atrial thrombi (7% of left atrial thrombi) and one right atrial thrombus (25% of right atrial thrombi) detected during biplane or multiplane transesophageal echocardiography were not visualized in the horizontal (0°) orientation, but all thrombi were identified in the vertical (90°) plane. Spontaneous left atrial contrast was identified by transesophageal echocardiography in 49% of the study participants.

Table 1. Characteristics and Transesophageal Echocardiographic Data for 230 Study Patients

	Left Atrial Thrombus Present (n = 34)	Left Atrial Thrombus Absent (n = 196)	p Value
Gender (female)	62%	53%	0.45
Age (yr)	75 ± 12	72 ± 13	0.30
Duration of atrial fibrillation (wk)	2.8 ± 3.1*	3.5 ± 6.5†	0.61
Recent thromboembolism	8 (24%)	15 (8%)	0.01
Left atrial dimension (cm)	4.8 ± 0.6	4.5 ± 0.8	0.065
Abnormal left ventricular systolic function	61%	32%	0.014
Left atrial spontaneous contrast	27 (79%)	85 (43%)	0.0002
Severity of mitral regurgitation (0–3+)	1.2 ± 0.7	1.4 ± 0.7	0.29
At least moderate mitral regurgitation	9 (26%)	56 (27%)	0.88

*Unknown, n = 7. †Unknown, n = 40. Data presented are mean value ± SD or number (%) or percent of patients.

Echocardiographic indexes associated with left atrial thrombi. Left atrial thrombi were significantly more frequent among patients with rheumatic mitral valve disease (24%, p = 0.019) and those with abnormal left ventricular systolic function, recent thromboembolism and spontaneous left atrial echo contrast (Table 1). However none of these variables was a reliable predictor of thrombus for an individual patient because of substantial overlap between groups. Patient age, gender, duration of atrial fibrillation and left atrial dimension (p = 0.065) were not significantly different between groups with and without left atrial thrombi (Table 1). Eighty percent of study patients had at least mild mitral regurgitation; there was no difference in the severity of mitral regurgitation between the groups with and without left atrial thrombi (p = 0.29). Twenty-seven percent of patients had at least moderate mitral regurgitation. Analysis of mitral regurgitation as a dichotomous variable based on less than or at least moderate mitral regurgitation also did not demonstrate a difference between the groups with and without atrial thrombi (p = 0.87).

Transesophageal echocardiography identified spontaneous echo contrast within the right atrium in 10% of patients (n = 24), including five of six patients with right atrial thrombi (vs. 9% of patients without thrombi, p = 0.001).

Cardioversion. Cardioversion was deferred in all 34 patients with evidence of left or right atrial thrombi. Subsequently three patients with left atrial thrombi died suddenly. One experienced a clinical embolic event before death, and a second patient was found to be asystolic 5 days after transesophageal echocardiographic identification of a left atrial thrombus (22). A limited autopsy (thorax only) in this latter patient found no residual left atrial thrombus. Another patient who presented with new atrial fibrillation and acute thromboembolism to the left leg (confirmed at thrombectomy) had a left atrial appendage thrombus identified on transesophageal

examination and died 4 days later of overwhelming sepsis. The family refused permission for an autopsy.

Eighteen (58%) of 31 surviving patients have undergone uneventful cardioversion after prolonged anticoagulation. Thirteen patients underwent cardioversion after repeat transesophageal echocardiography documented no residual thrombi; three patients had cardioversion after 4 weeks of oral anticoagulant therapy without repeat transesophageal echocardiography (decision of their primary physician), and two patients converted spontaneously and uneventfully 1 and 3 weeks after identification of a left atrial appendage thrombus on transesophageal study.

Of the 13 patients with thrombi who remained in atrial fibrillation, one underwent transesophageal echocardiography after 4 weeks of anticoagulation, which demonstrated a persistent 3-mm thrombus in the distal left atrial appendage. Cardioversion has been deferred indefinitely and she continues to receive anticoagulation. Repeat transesophageal echocardiography has been recommended for the remaining patients but 12 remain in atrial fibrillation and have not had a repeat study. Their primary physicians believed that these patients were not optimal candidates for cardioversion.

In 196 patients (85% of the total) no atrial thrombus was visualized by transesophageal echocardiography. Of these patients 186 (95%) had successful cardioversion to sinus rhythm; none received chronic anticoagulation and none (0%, 95% confidence interval 0% to 1.6%) experienced a clinical embolic event. One hundred patients (54%) had chemical cardioversion with either quinidine, procainamide, disopyramide, flecainide, amiodarone or sotalol. Three patients spontaneously converted including two who converted during transesophageal echocardiographic examination. The remaining 86 patients underwent successful electrical cardioversion either immediately after transesophageal echocardiography (n = 21) or after unsuccessful chemical cardioversion (n = 65). Ten patients failed to convert after attempted chemical or electrical cardioversion, or both, and continued to receive oral warfarin.

Discussion

Compared with smaller series (22–25), which showed only feasibility, this large prospective and consecutive study of 230 patients undergoing transesophageal echocardiographically facilitated early cardioversion in conjunction with short-term anticoagulation validates the safety of this treatment strategy for hospitalized patients with atrial fibrillation. Using the treatment algorithm described in Figure 1, with enrollment of almost 90% of all eligible patients admitted to our institutions over the past 4.5 years, we showed that the safety profile of this approach is equal to that of conventional therapy.

Advantages of early cardioversion from atrial fibrillation. Early and safe cardioversion in concert with short-term anticoagulation and guided by transesophageal echocardiography has several advantages over traditional strategies. Currently up to 4 weeks of oral anticoagulation is recommended before and after cardioversion (11,12), which exposes patients to a significant additional risk of a hemorrhagic complication by doubling their exposure to systemic anticoagulation. The incidence of major bleeding appears to be directly related to the length as well as the level of anticoagulation and is exceedingly rare in patients who have received oral anticoagulation for <1 month (8).

Early cardioversion also offers physiologic advantages over traditional therapy. The duration of atrial fibrillation is the strongest predictor of who will remain in sinus rhythm after cardioversion (33,34). Almost 60% of patients hospitalized for atrial fibrillation have been in atrial fibrillation <1 month (Fig. 2). Traditional treatment with 3 to 4 weeks of anticoagulation before cardioversion more than doubles the total period of atrial fibrillation. The time required for return of atrial mechanical function has been shown to be directly related to the duration of atrial fibrillation before cardioversion (16). Patients with atrial fibrillation <2 weeks in duration have complete return of atrial mechanical function within 24 h of cardioversion; those with atrial fibrillation <6 weeks in duration demonstrate return of atrial mechanical function within 1 week. Patients with atrial fibrillation for longer periods (>6 weeks) may require up to 3 weeks for return of atrial mechanical function. Finally, 4 weeks of anticoagulation and delayed cardioversion are often followed by a costly second hospital stay.

Echocardiographic indexes associated with left atrial thrombi. We found a significant increase in the incidence of both spontaneous echo contrast and abnormal left ventricular systolic function in patients with left atrial thrombi. Spontaneous echo contrast was also identified in nearly half of the patients in the series. Although its etiology remains controversial (35), the strong association between spontaneous echo contrast and atrial thrombus (36) suggests that efforts to identify atrial thrombus using transesophageal echocardiography should be especially diligent when spontaneous echo contrast is observed. Left ventricular dysfunction has also been associated with an increased risk of thromboembolism (37). Studies of chronic atrial fibrillation have shown a "protective effect" of mitral regurgitation against thromboembolism (38–40). Our data demonstrate that mitral regurgitation, analyzed as either a continuous or a dichotomous variable, was not significantly different between the groups with and without left atrial thrombi. This apparent conflict may reflect differences in pathophysiology of thrombus formation and thrombus migration (embolization), patient population (acute vs. chronic atrial fibrillation) or anticoagulation regimens. A recent transesophageal echocardiographic study by Fisher et al. (41) in patients with atrial fibrillation also failed to identify mitral regurgitation as protective against the formation of left atrial appendage thrombi.

Atrial thrombi were detected in 15% of the patients in the present series, a figure similar to that reported by others (42,43) but higher than the previously reported incidence of emboli from either chronic atrial fibrillation (44) or at cardioversion without anticoagulation (6–9). This higher number of patients with atrial thrombi most likely approximates the group

potentially at risk for early embolization because the number of patients who have early embolization will likely be some fraction of those with thrombi. Some thrombi may not embolize and some emboli may be clinically silent.

Role of anticoagulation in transesophageal echocardiographic studies to guide early cardioversion. Recent published reports (45–47) of patients who have experienced cardioversion-related embolic events after transesophageal echocardiography have demonstrated no thrombus before cardioversion. However the adverse events reported (45–47) have occurred uniformly in patients who have not received therapeutic anticoagulation with heparin or warfarin before transesophageal echocardiography and extending 3 to 4 weeks after cardioversion. Many underwent electrical cardioversion several days or weeks after transesophageal echocardiography, with no anticoagulation during this interval. For these patients it is impossible to exclude the possibility that atrial thrombi may have formed either between the transesophageal echocardiography and cardioversion or after cardioversion. Impaired atrial mechanical function (16,48) or new spontaneous contrast (45,49) has also been well documented after cardioversion. The lack of therapeutic anticoagulation in these cases (45–47) may have left patients vulnerable during the pericardioversion period. As in our preliminary report (22) we continue to recommend strongly that all patients admitted to the hospital for treatment of atrial fibrillation receive anticoagulation with intravenous heparin followed by transesophageal echocardiography only when a therapeutic partial thromboplastin time has been achieved. Systemic anticoagulation should then be continued for 3 to 4 weeks after cardioversion. Anticoagulation is given to minimize the potential for thrombi to develop between the periods of admission, transesophageal echocardiographic study and cardioversion. Systemic anticoagulation for 3 to 4 weeks after cardioversion should prevent thrombi from forming after cardioversion. To our knowledge this strategy has been associated with no thromboembolic events among patients undergoing early cardioversion.

Although 19 patients in our series had uneventful cardioversion immediately after transesophageal study and without any systemic anticoagulation, the small number of patients treated in this manner and the previously mentioned reports of embolic events in patients treated in this manner (45–47) preclude our recommendation for cardioversion without anticoagulation. Such a treatment strategy may be reasonable for the very small number of subjects in whom even short-term intravenous heparin is contraindicated, such as patients with active gastrointestinal bleeding or intracranial hemorrhage. A transesophageal echocardiographic approach for such a group may be safer than "blind" cardioversion, although this remains to be studied. As always the risks and benefits of cardioversion for these patients must be considered carefully.

Clinical implications. Because the previously reported incidence of cardioversion-associated thromboembolism in patients who have received prophylactic anticoagulation (conventional therapy) is also low, our nonrandomized prospective study does not prove that a transesophageal echocardiographically guided approach in concert with short-term anticoagulation is safer than the conventional strategy. A series of >7,000 randomized patients will be required to compare directly transesophageal echocardiographically guided cardioversion with prophylactic anticoagulation (22). Such a trial has been initiated recently (49), and we support such studies to identify which strategy is the safest. Our prospective report of 230 consecutive patients with atrial fibrillation undergoing cardioversion does validate the safety of this approach as equivalent to that of conventional therapy and also represents, to our knowledge, the largest single prospective study of patients undergoing cardioversion with primarily "natural" atrial fibrillation. The absence of any embolic events in our series argues favorably for this approach to cardioversion for hospitalized patients with atrial fibrillation. Recent data also have suggested that this approach is cost-effective in this population (50).

We do not advocate the use of transesophageal echocardiography in all patients with atrial fibrillation. Our study included patients hospitalized for treatment of atrial fibrillation. The role and especially the cost-effectiveness of transesophageal echocardiography in the treatment of outpatients with atrial fibrillation and its role in patients with atrial flutter, remain to be determined. We did not study patients with postoperative atrial fibrillation because they were recognized early (<2 days), thus excluding them from this study. We also do not support the routine use of transesophageal echocardiography before cardioversion in patients who have had adequate anticoagulation with prolonged (>3 weeks) warfarin therapy before planned cardioversion. It does seem reasonable to consider transesophageal echocardiographic study (even after 3 weeks of warfarin) for those patients in whom the initial presentation included acute thromboembolism or for those in whom warfarin anticoagulation has been intermittently subtherapeutic, but these groups remain to be studied.

Study limitations. Our study was not designed to compare the sensitivity or specificity of different transesophageal echocardiographic imaging planes for the detection of atrial thrombi. The superiority of biplane examination for imaging both atria and appendages has been reported by several observers (51,52), and our data corroborate those reports. The additional value of multiplane transesophageal echocardiography in this setting remains to be defined, although we currently perform only multiplane examinations. We devote considerable time during the transesophageal echocardiographic examination to studying the atria/appendage. Using this strategy the accuracy of transesophageal echocardiography in identifying atrial thrombi is extremely high (17,18,53), although certainly there is a finite resolution to transesophageal echocardiography and very small thrombi may be missed. It is for this reason that we strongly recommend systemic heparin at the time of transesophageal study, extending for 3 to 4 weeks after successful cardioversion. Treatment of patients in whom the atrial appendage is not adequately visualized by transesophageal echocardiography would be best accomplished with conserva-

tive therapy with prolonged warfarin before (and after) cardioversion.

Conclusions. This large prospective and consecutive study documents the safety of transesophageal echocardiography in concert with short-term anticoagulation to facilitate early cardioversion for hospitalized patients with atrial fibrillation of unknown or prolonged duration. This approach is as safe as conventional therapy. Such a strategy also minimizes the total durations of anticoagulation, atrial fibrillation and atrial mechanical dysfunction.

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