As in the report of Manning et al,2 anticoagulation management was not uniform in the present series, reflecting physician judgments regarding the requirement for and contraindications to anticoagulation. Our study did not address the optimal duration of anticoagulation after cardioversion, which may depend on the time course of atrial function after cardioversion3,4 and the likelihood of arrhythmia recurrence. Most cases of cardioversion-related embolism occur in the first week after cardioversion.1,5–8 Although the minimum anticoagulation duration of 24 hours after cardioversion recommended by Manning et al may provide some benefit, it appears prudent to continue anticoagulation after cardioversion for at least one week and as long as one month, until further data are available.

As noted in the discussion, the 2.4% incidence of embolism in our study overestimates the true incidence of embolism after TEE-guided cardioversion, as the denominator of 712 patients was drawn only from centers in which embolic events occurred. However, this incidence is consistent with the 95% confidence intervals for embolism of 0.4–6.6% in Manning’s study2 and is comparable with the incidence reported by Arnold et al.8 Although we agree with Manning et al that TEE is helpful for patients in whom anticoagulation is absolutely contraindicated, clinicians should understand the potential risks of such an approach.

The possibility that thrombus may have developed before cardioversion in the three patients with delays after TEE has already been addressed in our paper.1 As in Dr Manning’s study,2 a minority of our patients was studied with single plane TEE. Their study showed no difference in the sensitivity of the transverse and longitudinal planes for left atrial thrombi. Nevertheless, we recommend that TEE-guided cardioversion be performed using biplane or multiplane TEE probes. Finally, we note that this multicenter cooperative study included all cases of embolism after TEE-guided cardioversion known to the investigators, including seven patients included in other studies. This multicenter approach enabled comprehensive, uniform evaluation of all known cases.

In response to Dr Josephson’s remarks, there was no “two year hiatus” in data collection. None of the patients undergoing TEE-guided cardioversion in 1989 or 1990 had embolic events. Quality control is an important issue, which we specifically addressed. All videotapes were independently reviewed by an experienced echocardiographer, and all studies except one unavailable videotape were reviewed in a central reference laboratory. The absence of atrial thrombus was confirmed in all cases. Excluding the 50% of patients with atrial fibrillation who have left atrial “smoke” from cardioversion is not the practice of physicians experienced with the procedure, except in occasional cases when left atrial thrombus cannot be confidently excluded or the patient is not therapeutically anticoagulated. It is clear from our study that no precardioversion characteristic reliably predicts patients at risk of embolism after cardioversion.

Correspondence

Ian W. Black MBBS, FRACP
Diane Fatkin MBBS, BSc (Med), FRACP
Kirin B. Sagar, MD
Bijoy K. Khandheria MBBS
Dominic Y. Leung MBBS, MRCP(UK), FRACP
James M. Galloway, MD
Michael P. Feneley MD, FRACP
Warren F. Walsh MBBS, FRACP
Richard A. Grimm, DO
Claudia Stollberger, MD
Patrick M.J. Verhorst, MD
Allan L. Klein MD, FRCP(C)

References


Thromboembolism After Negative TEE

To the Editor:

We read with interest the report by Black et al regarding thromboembolism following a negative transesophageal echocardiogram (TEE) and cardioversion among patients with atrial fibrillation.

As stated in our preliminary report regarding the use of TEE to guide early cardioversion,2 we continue to advocate therapeutic anticoagulation with heparin/warfarin before TEE and extending to at least 24 hours and preferably one month after cardioversion. Almost 90% of the patients in our report were treated in this manner.2 Therapeutic anticoagulation was prescribed prior to cardioversion because of the finite spatial resolution of TEE as well as for prophylaxis during return of atrial mechanical function. We now have experience in over 200 patients utilizing such a strategy with similar results. To our knowledge, no cardiovascular associated thromboembolism has been reported in any patient who has been treated in this manner (1 month of postcardioversion anticoagulation).

While we do not advocate the use of a TEE-guided strategy without therapeutic anticoagulation, we do believe that this approach “may be reasonable for the very small minority of patients who are poor candidates for any anticoagulation.”2 The report by Black et al states that the embolic complications occurred in 2.4% of 712 patients screened by TEE before cardioversion at the participating centers. If all 712 patients did not receive adequate anticoagulation prior to cardioversion, then the TEE strategy would appear preferable to the 3.3% incidence of thromboembolism following “cardioversion without anticoagulation” recently reported by Arnold et al.3 The lack of a uniform treatment of these patients, however, makes the data difficult to interpret.

We are concerned about the implication made by the authors that a negative TEE should have excluded thromboembolism in their group of 17 patients. Three patients did not undergo cardioversion until 7 to 21 days following TEE. Certainly a TEE performed weeks earlier cannot exclude the development of a small thrombus in a nonanticoagulated patient with atrial fibrillation. It is unfortunate that these investigators did not use our strategy of immediate post-TEE electrical cardioversion for patients not receiving therapeutic anticoagulation. Four of their patients were in atrial fibrillation at the time of thromboembolism,
not sinus rhythm. The development of a thrombus during the period of atrial fibrillation cannot be excluded. Finally, 8 patients underwent monoplane TEE examination. While no randomized trials of monoplane vs biplane or multiplane have been published, the latter two techniques are widely acknowledged to be superior. Several recent reports have documented the visualization of thrombi in the vertical (90°) plane which are not seen in the horizontal plane.

We are in partial agreement with the authors’ conclusion that a “negative TEE for thrombus not obviate the requirement for anticoagulant therapy during and after cardioversion.” In addition, we strongly recommend that patients undergoing TEE guided strategy for early cardioversion should be therapeutically anticoagulated at the time of the TEE and extending at least 24 hours after cardioversion and (preferably) 1 month after cardioversion. Atrial thrombi may form between TEE and cardioversion, and stunning of the atria following successful cardioversion has been well documented.3

Finally, since it appears that many of the patients in this report have been described before,3-7 this should be acknowledged so as to avoid the impression that these represent 17 new patients with embolic complications following TEE guided cardioversion.

Warren J. Manning, MD
David J. Silverman, MD
Pamela S. Douglas, MD
Harvard Medical School
Beth Israel Hospital
Boston, Massachusetts

References


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

To the Editor:

I believe the article “Exclusion of Atrial Thrombus by Transesophageal Echocardiography Does Not Preclude Embolism After Cardioversion of Atrial Fibrillation” by Black et al1 has numerous methodological problems.

The fundamental conclusion is that 17 patients who had precardioversion TEEs, which were interpreted as having no evidence of atrial thrombus, subsequently had cardioembolic events after cardioversion. These 17 patients are said to be out of a total of 712 patients; this denominator is likely a minimum number, as the methods employed to ensure ascertainment of the total number of TEEs in the various institutions is not explained. The fact that some patients were evaluated in 1988 and then there was 2-year hiatus until the next 9 patients were enrolled in 1991 strongly suggests the possibility of ascertainment bias.

There is a specter of lack of quality control, as the echos were not read in a blinded fashion, or in a central reference laboratory, nor overread by another echocardiographer. All of these techniques are commonly employed in other studies of cardiovascular imaging to ensure quality control.

Half the patients underwent evaluation with single-plane probes; a technique that is known to have diminished sensitivity. It is inappropriate, particularly in 1994, to lump the single-plane studies with the multiplane studies, particularly if the above quality control measures were not employed.

Left atrial “smoke” is known to be associated with a prothrombotic state and to be associated with atrial thrombus. It is not particularly surprising that some patients with smoke would subsequently have emboli. Many consider the presence of smoke, particularly if it is moderate to severe, to be a contraindication to cardioversion.

There was a significant delay between the TEE and subsequent cardioversion in several patients; in 3 of 17 patients, this lag was in excess of 1 week. It is not surprising that a negative TEE at one point in time does not guarantee the subsequent (precardioversion) development of atrial thrombus.

There is no guarantee that all the strokes in this patient population of nearly 1000, many of whom undoubtedly had hypertension, diabetes, and atherosclerosis, were of cardioembolic origin, despite the temporal coincidence.

The authors briefly mention some of the study limitations does not really negate the fact that the 17 patients are incompletely characterized both clinically and by echos that have not been subject to quality review.

What is needed to better evaluate this case series is a description of the total denominator, including an explanation for the 2-year hiatus in patient recruitment, more detailed individual patient characteristics (perhaps in tabular form) so that one can assess the number of patients who had TEEs with state-of-the-art equipment that were free of “smoke,” who underwent cardioversion in a relatively short period of time after the TEE, and had no other likely source of their neurologic event. Improved quality control of the echocardiographic interpretations would appear mandatory.

While I applaud the notion of further study of the clinical utility of TEE in patients considered for cardioversion of atrial fibrillation, it would appear that the current report provides insufficient justification for the authors’ planned ACUTE study.

Richard A. Josephson, MS, MD
Director of Cardiology Research & Education
Codirector Echocardiography Laboratory
Akron City Hospital/Summa Health System
Akron, Ohio

Effects of Dietary Iron Intake on Stored Iron, Free Iron, and Coronary Disease

To the Editor:

The study by Ascherio et al1 is an important contribution to our understanding of the role of iron in ischemic heart disease. It provides strong new support for the “iron theory”2,3 and the key hypothesis that iron depletion protects against ischemic heart
Thromboembolism after negative TEE.
W J Manning, D I Silverman and P S Douglas

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The online version of this article, along with updated information and services, is located on the World Wide Web at:
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