with the asthmatic state. We agree with Dr. Malo that nebulized agents like methacholine or histamine are the best single test to use. We only use cold air in subjects with symptoms suggesting cold intolerance who show normal responses to methacholine. Such patients are not uncommon, perhaps because we, like Dr. Malo, live in a place with a very large supply of cold air.

N. R. Anthonisen, M.D.,
University of Manitoba,
Winnipeg, Canada

Multiple Protruding, Mobile Left Ventricular Thrombi and Risk of Embolism after Cardioversion

To the Editor:

Although many patients with left ventricular aneurysms have associated mural thrombi, the reported risk of clinical cerebral embolism as a result of cardioversion during electrophysiologic study is remarkably low. The embolic risk of cardioversion in patients with intraventricular mural thrombi has not been studied systematically, but it is known that protruding and mobile left ventricular thrombi have been reported to be associated with a high risk of embolism. We report a patient with multiple, "high-risk" mural thrombi who received multiple cardioversions during serial electrophysiologic studies without clinical signs of embolization.

A 62-year-old white man suffered an acute anterior myocardial infarction for which he was initially treated with intravenous streptokinase, followed by heparin. Coumadin was subsequently added and heparin therapy discontinued. An echocardiogram on the seventh day post-infarction demonstrated no intraventricular thrombi. Angiography revealed single-vessel disease with total occlusion of the left anterior descending artery after the first septal perforator. An antero-apical aneurysm was noted without evidence of left ventricular thrombus. Three weeks after infarction, the patient underwent two serial electrophysiologic studies, during which he required five cardioversions to terminate ventricular tachycardia without embolic complications. An echocardiogram performed four weeks post-infarction revealed the presence of several protruding left ventricular thrombi of up to 2 cm in diameter; at least two were pedunculated and freely mobile (Fig 1). A repeat echocardiogram performed six weeks post-infarction revealed no significant change. A third follow-up electrophysiologic study was performed, during which cardioversion was again required without embolic complications.

The risk of embolism is greatest during the first six weeks after myocardial infarction and may be reduced by systemic anticoagulation prophylaxis therapy. It is possible that prior treatment with anticoagulants may have reduced the risk of embolization in our patient, although the first five cardioversions were performed without anticoagulant therapy. Even though cardioversions in our patient were uneventful, subclinical thromboembolism under these circumstances could not be excluded. The risk-vs-benefit ratio of electrophysiologic study in patients like ours should be individually assessed.

Y. S. Archie Lo, M.D., F.C.C.P., and
Charles D. Sirdell, M.D.,
Division of Cardiology,
Stanford University Medical Center,
Stanford, California

REFERENCES


FIGURE 1. (Right) Apical four-chamber view showing the left ventricle with two large, protruding thrombi (arrows) in the antero-apical aneurysm. (Left) Apical two-chamber view showing a pedunculated, protruding thrombus (arrow).