Cardiology Grand Rounds from

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Part I: Use of Echocardiography in the Evaluation of Patients with Suspected Cardioembolic Stroke

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ABSTRACT: One-sixth of all ischemic strokes are a result of embolization from the heart. Echocardiography serves as the cornerstone in evaluating patients who may have had a cardioembolic stroke. This article is the first of a two-part review series focusing on the role of echocardiography in the diagnosis and treatment of cardioembolic stroke. Specifically, this section will focus on nonrheumatic atrial fibrillation as well as cardiovascular masses as potential embolic sources. **KEY INDEXING TERMS:** Transesophageal echocardiography; Stroke; Atrial fibrillation; Cardiac mass; Aortic atherosclerosis. **[Am J Med Sci 2005; 329(6):310–316.]**

The diagnosis of a cardioembolic source of stroke is uncertain and relies on the identification of a potential cardiac source of embolism in the absence of significant cerebrovascular disease.[1,2] In clinical studies, cardiogenic embolism has accounted for up to one-sixth of ischemic strokes, and there is no doubt that echocardiography serves as the cornerstone in the evaluation and diagnosis of these patients.[1] Currently, evaluation for a possible cardioembolic source of stroke is the leading clinical indication for referral for transesophageal echocardiography (TEE) accounting for 26% of the 8500 patients referred for TEE in the Value Of TEE (VOTE) registry.[3] There are no sensitive nor specific clinical diagnostic criteria for cardioembolic stroke. Although less than 2% of patients found to have a cardiac source of stoke have no clinical evidence of heart disease, a potential source can be identified by echocardiography in nearly 10% of these patients. As a result, the cerebral embolism task force recommends that echocardiography be used liberally in the evaluation of patients with stroke.[1,2]

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Cardiac Sources of Emboli

Echocardiography is capable of detecting multiple potential causes of stroke. Cardioembolic sources for stroke can be separated into three distinct categories, as shown in Table 1. The first subset includes cardiac lesions that have a propensity for thrombus formation. This includes specific characteristics of the left atrial appendage and the presence of mitral annular calcification in patients with nonrheumatic atrial fibrillation (AF). A second group of causes is the presence of cardiovascular masses, which may include intracardiac tumors, thrombi, or atherosclerotic plaques of the aorta. The third subset includes passageways within the heart serving as conduits for paradoxical embolization from the venous system. The most commonly identified passageways implicated in cardiogenic strokes are atrial septal defects and patent foramen ovale.

The role of echocardiography in the diagnosis and treatment of embolic sources of stroke in nonrheumatic AF as well as cardiovascular masses as potential causes of stroke will be the focus of this review. Part two of this review will focus on the use of echocardiography for evaluation patients with potential pathways for paradoxical embolization with a discussion on the current recommendations in their management.

Nonrheumatic Atrial Fibrillation and Cardioembolic Stroke

Atrial fibrillation is a well known source of morbidity and mortality for both men and women across a broad spectrum of ages.[4] Currently the American College of Cardiology/American Heart Association and the Sixth American College of Chest Physicians Consensus Conference on Antithrombotic Therapy, in recognition of the high risk of thromboembolism, recommend anticoagulation with a target international normalized ratio (INR) of 2.5 in mitral stenosis for all patients with either a previous embolic event or an episode of AF.[5,6] Because the treatment recommendations for such patients is well established, we will focus our discussion on patients with AF in the absence of rheumatic valvular disease. Nonrheumatic AF in the general population is associated with a fivefold to sevenfold increased risk of ischemic stroke. In the Stroke Prevention in AF

Table 1. Classifications of cardiac sources of embolic stroke

1. Atrial fibrillation (nonrheumatic)
a. Left atrial appendage thrombi
b. Left atrial appendage velocity
c. Aortic atherosclerotic disease
d. Mitral annular calcification
2. Cardiovascular masses
a. Mobile aortic atherosclerotic disease
b. Left ventricular thrombus
c. Intracardiac masses
3. Paradoxical emboli pathways
a. Patent foramen ovale
4. Atrial septal defects

Study, 1330 patients with persistent or paroxysmal AF were treated with either 325 mg/day aspirin (double-blind) or warfarin with placebo. During a mean follow-up period of 1.3 years, the rate of ischemic stroke or systemic embolism in patients assigned to placebo was 6.3% per year and was reduced to 3.6% per year in the aspirin-treated group. In the subgroup of warfarin-eligible patients, the risk of stroke was reduced to 2.3%, versus 7.4% per year compared with placebo.[7] In an echocardiographic substudy from the SPAF-I trial, 568 patients assigned to the placebo arm underwent transthoracic echocardiography (TTE) at the beginning of the trial in an effort to identify echocardiographic predictors of thromboembolism. Left ventricular dysfunction ($P = 0.003$) and the size of the left atrium ($P = 0.02$) were the strongest independent predictors of later thromboembolism. In patients not having these two independent echocardiographic predictors or any of three identified clinical predictors of thromboembolism (history of hypertension, recent congestive heart failure, previous thromboembolism), the risk of thromboembolism was low (1% per year). However, in patients with no clinical predictors of thromboembolism but with either or both of the echocardiographic predictors, the risk of stroke was 6% per year.[8] This was the first study showing that echocardiography can risk-stratify patients with AF beyond clinical evaluation and guide their therapy accordingly.

In a transesophageal echocardiography (TEE) substudy from the SPAF-III trial, Goldman et al.[9] showed a pathophysiologic correlation between the risk of stroke in patients with AF and the low atrial activity as measured by left atrial appendage velocity. In 721 patients with history of nonvalvular AF who underwent TEE, the velocity in the left atrial appendage was significantly lower (20–29 cm/sec) if the patients were in AF at the time of the TEE, as compared with 50–60 cm/sec if sinus rhythm was

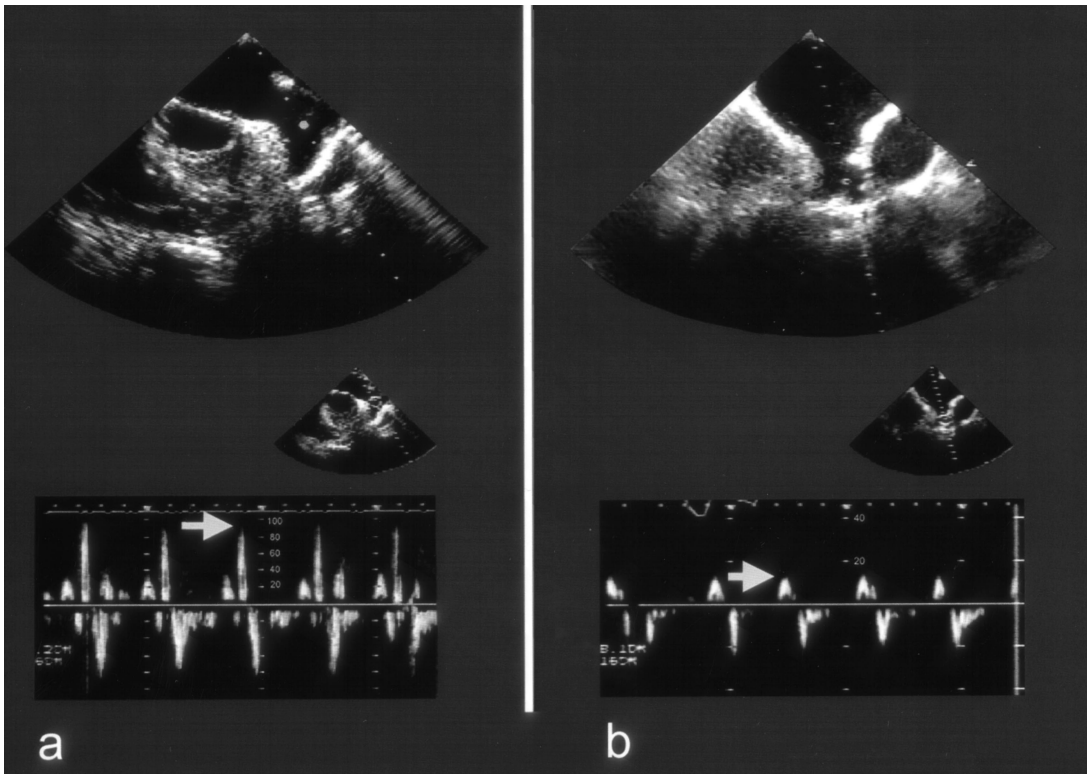

Figure 1. Transesophageal echocardiogram of the left atrial appendage in a patient in sinus rhythm as compared with a patient in atrial fibrillation. The arrow in (a) shows the velocity in the left atrial appendage to be greater than 80 cm/sec while the arrow in (b) shows the velocity to be less than 20 cm/sec.