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Cardiovascular Sources of Embolic Stroke: II

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Cardiovascular Sources of Embolic Stroke: II

Craig M. Brodsky, MD

I. INTRODUCTION

This article details the cardiac evaluation of patients with a suspected embolic stroke. This is the second part of a 2-part review on cardiovascular sources of embolic stroke (**Appendix 1**). The first part of the series (*see* “Cardiovascular Sources of Embolic Stroke: I” in the *Hospital Physician Cardiology Board Review Manual*, Volume 8, Part 4) reviewed atrial fibrillation, atrial flutter, left ventricular thrombosis, myxoma, and aortic atherosclerosis. This part of the review focuses on strokes caused by patent foramen ovale (PFO), valvular excrescences, endocarditis, cardiac catheterization, and mitral valve prolapse.

Patients with a suspected cardiovascular source of embolic stroke typically present to a neurologist. The neurologist will often request either a consultation by a cardiologist or an echocardiogram to help determine the etiology of the event. The cardiologist and neurologist often must work together to determine the etiology of the event. For those patients who present directly to the cardiologist with acute neurologic symptoms, consultation with a neurologist may be helpful. Although this part of the review focuses on less common causes of embolic stroke, familiarity with these potential sources of cardiovascular embolism is essential to the practice of cardiology.

II. ETIOLOGY OF EMBOLIC STROKE

PATENT FORAMEN OVALE

Approximately 10% to 15% of the general population has PFO.¹⁻³ Paradoxical embolism through a PFO is thought to be a frequent cause of embolic stroke, particularly in young patients with cryptogenic stroke. Two studies found PFO in 40% to 50% of patients with ischemic stroke compared with 10% to

15% of controls.^{2,3} A recent meta-analysis confirmed these findings, noting that both PFO and atrial septal aneurysms are unequivocally associated with an increased incidence of stroke in patients younger than 55 years.⁴ The combination of a PFO and atrial septal aneurysm represents a higher risk than either finding alone.⁵

Echocardiography is the test of choice for the detection of a PFO (**Figures 1 and 2**). After injecting agitated saline into a peripheral intravenous line, images are obtained with either transthoracic or transesophageal imaging. The use of transesophageal imaging is ideal because the mid-esophageal position brings the probe to within a few centimeters of the atrial septum, yielding superior anatomic detail. The membrane of the foramen ovale and the presence of an atrial septal aneurysm can be easily discerned. The overall yield for detecting a potential source of embolism by transesophageal echocardiography is 43%, compared with 14% for transthoracic echocardiography.⁶

The risk of paradoxical embolism in those with a PFO can be determined by several echocardiographic variables. The size of the PFO is associated with the risk of paradoxical embolism. In a case-controlled study of 244 patients, those with stroke or transient ischemic attack who had a PFO had a mean PFO diameter of 4 ± 2 mm, versus 2 ± 1 mm in control patients.⁷ The passage of microbubbles across the shunt as visualized by transesophageal echocardiography also is associated with embolism. Those who have passage of greater than 20 microbubbles across the PFO are much more likely to have a recurrent event than those with a smaller shunt (31% versus 0%; $P = 0.03$).⁸ Atrial septal aneurysms are similarly associated with stroke.⁹ The presence of an atrial septal aneurysm has a synergistic effect on risk of stroke when combined with a PFO.¹⁰ Finally, those with patency of the PFO at rest (ie, those in whom microbubbles cross without a Valsalva maneuver) and those with greater membrane mobility have a higher likelihood of stroke recurrence.¹¹