Echocardiography in stroke: Which probe when?

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Patients who have experienced a transient ischemic attack or stroke are often extremely concerned about the event and the likelihood of recurrence. Although they want "straight answers" from their physicians, it is often difficult to provide definitive answers to even the simple question, "What caused my stroke?" Physicians try to answer one aspect of the question by performing echocardiograms to identify a possible cardiac source of brain embolism.

In their review (page 989), Moira Kapral and Frank Silver, with the Canadian Task Force on Preventive Health Care, have been careful in their selection of studies.[1] To derive a pooled estimate of the yield of echocardiography in detecting intracardiac masses requiring therapy in patients with stroke, they included only studies that examined consecutive stroke patients — a critical restriction. Series of nonconsecutive patients referred for echocardiography are biased by selection, because the yield of echocardiography is clearly higher among those with evidence of heart disease.

Many publications describing the value of echocardiography or other diagnostic tests do not elucidate the effect of test results on patient management. In this regard, the task force should be commended for recognizing that the appropriate use of imaging requires an understanding of both the capabilities of the imaging technique and the value of therapy used to treat the identified condition. The task force's statement that the identification by echocardiography of a condition requiring anticoagulation in a patient with another known condition requiring anticoagulation (e.g., atrial fibrillation or a mechanical heart valve) will not lead to a change in patient management bears emphasis.[2] After all, a test useful to a clinician is one that provides important new information leading to a change in approach or important confirmation of a previously selected approach. A test useful to a patient is one that leads to a change in understanding and treatment associated with a better outcome: new information that does not improve outcome has no value.

That this is true is highlighted by the authors' discussion of the role of patent foramen ovale and atrial septal aneurysm in stroke. Many series suggest a considerable risk of recurrent events in these patients. The treatment of these abnormalities, which are present in a very high proportion of young patients with stroke, can include antiplatelet therapy, anticoagulant therapy, closure with a device delivered by means of a catheter or surgical closure in an open-heart procedure.[3,4] Several small series have demonstrated low rates of recurrent events after anticoagulation or closure of the defects,[5–7] whereas others have demonstrated less impressive benefit.[8] There is, as yet, no evidence from rigorous randomized trials that antiplatelet therapy, anticoagulant therpay or closure of defects is associated with a better outcome.

The issue of whether transesophageal echocardiography (TEE) should be used as the initial screening test is more controversial, however. In the early days of TEE Pearlman[9] raised a concern that physicians might use it directly without performing transthoracic echocardiography (TTE).[9] The task force has endorsed this practice, presumably based on the much higher yield of TEE for the detection of atrial thrombi and atheromatous aortic debris. Given the data reviewed, it is not surprising that the task force arrived at this conclusion. In the cost-effectiveness analysis performed by McNamara and colleagues[10] the "yield" was limited to identification of these thrombi; since TTE has a very low sensitivity for identifying atrial thrombi, TEE emerged as the preferred initial strategy.

Most echocardiographers view the 2 techniques as complementary and perform TTE initially. They then proceed with a "focused" transesophageal study. It seems prudent to begin with a less invasive, although less sensitive, procedure if there is a reasonable likelihood that the result will influence therapy and avoid the need for a substantial number of more resource-intensive, invasive transesophageal procedures. Furthermore, although the sensitivity of TTE for identifying patent foramen ovale (about 50%) is lower than that of TEE (about 100%), it is substantial. Indeed, the high prevalence of patent foramen ovale among young stroke patients was first identified through the use of contrast TTE. Thus, if an echocardiogram is requested for a patient under age 50 presenting with stroke or transient ischemic attack without an identified source of embolus, the approach in our laboratory is to perform TTE with injection of agitated saline. Patients with identified patent foramen ovale and moderate to marked right-to-left shunt are often prescribed anticoagulant therapy and occasionally undergo closure of the defect. If the transthoracic study is negative we would proceed to TEE if the attending physician wishes to pursue a cardiac source of embolus. I believe that many neurologists make therapeutic decisions in cases of patent foramen ovale identified by TTE.[3]

To date, no definitive series has compared outcomes and costs associated with TEE used as the initial procedure ver-

sus TTE followed by TEE if the transthoracic study does not reveal a treatable source of embolus. In the absence of direct comparison of the strategies, I believe that there is continued room for our approach of performing TTE first, with subsequent TEE in cases in which identification of an intracardiac thrombus, a patent foramen ovale or an atrial septal aneurysm would provoke a change in the attending physician's management (usually anticoagulant therapy or consideration of defect closure).

The task force has made recommendations on the basis of what is known and has (correctly) made limited excursions beyond the data. Clinicians are forced to decide whether to wait for the evidence or to follow intuition and trends. Delineation of the role of patent foramen ovale and atrial septal aneurysm in stroke and assessment of available treatment strategies is proceeding rapidly. Until the definitive results are available, the article and recommendations of the task force provide a useful guide to clinicians. I disagree with its recommendation of proceeding directly to TEE. I believe that many clinicians will continue to follow an approach similar to that currently used in our laboratory, as I have outlined here.

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