Transesophageal Echocardiography in the Detection of Potential Cardiac Source of Embolism in Stroke Patients

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To compare the diagnostic yields of transesophageal and transthoracic echocardiography in the detection of potential cardiac sources of embolism, 63 patients (mean±SD age 63±15 [range 18–87] years) with transient ischemic attacks or stroke underwent both procedures. Transthoracic echocardiography revealed a potential cardiac source of embolism in 14% (nine) of the patients, all of whom had clinical evidence of heart disease. Transesophageal echocardiography revealed a potential cardiac source of embolism in 41% (26) of the patients; 27% (seven) of these patients had no clinical cardiovascular abnormalities. Abnormalities detected only by transesophageal echocardiography in the patients with unsuspected cardiac disease included atrial septal aneurysm in two, patent foramen ovale in two, left atrial appendage thrombus in one, and myxomatous mitral valve in two. The 26 patients with an identified cardiac source of embolism were older (67.5 versus 59.4 years, \textit{p}=0.04), more frequently in atrial fibrillation (62% [16] versus 8% [3], \textit{p}<0.0001), had a larger left atrium (43 versus 37 mm, \textit{p}=0.01) and more commonly had left ventricular hypertrophy (62% [16] versus 32% [12], \textit{p}<0.02) than the 37 patients in whom no cardiac source of embolism was identified. Thus, transesophageal echocardiography is more sensitive than transthoracic echocardiography in the detection of potential cardiac sources of embolism in patients with cerebral ischemic events. (Stroke 1991;22:727–733)

Ischemic stroke is a major cause of morbidity and mortality in North America.\textsuperscript{1,2} It is estimated that 15% of all ischemic strokes result from an embolus originating in the heart.\textsuperscript{3–4} Traditional non-invasive methods of assessing potential cardiac sources of embolism have yielded disappointing results. Precordial echocardiography\textsuperscript{5–10} and Holter monitoring\textsuperscript{5} add little to the clinical examination of the patient in detecting a cardiac source of embolism. Transesophageal echocardiography is a new application of echocardiography that allows high-resolution imaging of the atria, atrioventricular valves, and aorta by inserting an ultrasonic transducer in the esophagus.\textsuperscript{11} Recent studies have suggested that transesophageal echocardiography may detect a potential cardiac source in up to 65% of patients with cerebral ischemic events or systemic arterial embolism.\textsuperscript{12–14}

This study was undertaken to compare the diagnostic yield of transthoracic echocardiography with that of transesophageal echocardiography in the detection of a cardiac source of embolism in patients with cerebral ischemic events. The clinical characteristics of patients with an identified cardiac source of embolism were compared with the characteristics of patients who did not have such a source identified by transesophageal echocardiography.

Subjects and Methods

We enrolled 63 patients with a recent transient ischemic attack or cerebrovascular accident\textsuperscript{15} in this study between September 1989 and July 1990. Patients were referred to the Echocardiography Laboratory by neurologists who made this diagnosis. Three time slots were made available every week in the Echocardiography Laboratory for these patients. The first three patients whose requisitions were received during the week were selected for participation in this study. All patients had a history taken, with note made of previous myocardial infarction, heart failure, or arrhythmias. A cardiac physical examination was oriented toward the detection of pathological heart murmurs. Twelve-lead electrocar-
diography was performed and the results were analyzed for the presence of atrial fibrillation/flutter or pathological Q waves in keeping with myocardial infarction.

Transesophageal color Doppler echocardiography was performed on the same day as transesophageal echocardiography. Standard parasternal and apical views were obtained using an Aloka 870 imaging system (Aloka Co., Tokyo, Japan) interfaced with a 2.5- or 3.5-MHz transducer. Intravenous saline contrast was not given during transesophageal echocardiography.

Transesophageal color Doppler echocardiography was performed after the patient gave informed consent. A biplane transesophageal 5-MHz transducer interfaced with the Aloka 870 imaging system was used. A complete biplane transesophageal examination was performed in all patients. Thirty patients who had an intravenous line inserted for sedation also had saline contrast injected during transesophageal echocardiography to exclude an intracardiac shunt. All transthoracic and transesophageal echocardiograms were recorded on a Panasonic 6300 VHS videocassette recorder (Matsushita Electric of Canada Ltd., Mississauga, Canada) and were reviewed on two separate occasions by a single observer. Results of all studies were reported to referring physicians.

The following echocardiographic abnormalities were considered a possible cardiac source of embolism: left atrial or ventricular thrombus, left atrial spontaneous echo contrast, mitral stenosis, myxomatous degeneration of the mitral valve with prolapse, severe mitral annular calcification, atrial septal defect, patent foramen ovale, left ventricular aneurysm, left ventricular free wall, left ventricular aneurysm or apical akinesis, aortic/mitral vegetation, atrial myxoma, moderate-to-severe global left ventricular hypokinesia, and prosthetic heart valve. Protruding thrombus in the ascending aorta was considered a possible source of embolism. Isolated left atrial enlargement, segmental inferior or lateral left ventricular hypokinesia, aortic sclerosis, or systolic buckling of the mitral leaflets less than 3 mm into the left atrium without leaflet redundancy were not considered sources of embolism.

Patients were divided into two groups based on clinical evidence of heart disease. Group I consisted of 24 patients with a clinically suspected cardiac source of embolism. Three patients had mitral stenosis, all of whom were also in atrial fibrillation. Eight patients had both historical and electrocardiographic evidence of transmural myocardial infarction (anterior in three, inferior in four, and both anterior and inferior in one); four of these patients were in atrial fibrillation. One patient had native mitral valve endocarditis. The 12 patients without clinical evidence of mitral valve disease or myocardial infarction had atrial fibrillation documented by electrocardiography during their current hospital admission or within the preceding 6 months. Group II consisted of 39 patients in sinus rhythm without evidence of myocardial infarction, mitral stenosis, mitral valve prolapse, or endocarditis.

Mean age and left atrial size were compared between groups of patients with an identified cardiac source of embolism and those without using the unpaired t test. Nonparametric data (sex, presence of atrial fibrillation, and left ventricular hypertrophy) were compared using Fisher’s exact test or the χ² test. Significance was assessed as p<0.05.

Results

We studied 32 men and 31 women. The mean age of the patients was 63 (range 18–87) years. The mean time from the cerebral ischemic event to echocardiography was 34 (range 1–210) days.

Among the 24 group I patients, nine (38%) had a possible cardiac source of embolism identified by transthoracic echocardiography (Figure 1). The three patients with clinical evidence of mitral stenosis had this confirmed. Of the eight patients with myocardial infarction, four had an area of apical dyskinesis that may have resulted in thrombus formation and subsequent embolization. Only one of these four patients had a layered nonprotruding apical thrombus. The patient with endocarditis had a vegetation on the mitral valve. Of the 12 patients with atrial fibrillation, only one had a possible cardiac source of embolism detected on transthoracic echocardiography. She had severe mitral annular calcification.

Transesophageal echocardiography revealed a potential cardiac source of embolism in 19 (79%) of the 24 patients in group I. In the three patients with mitral stenosis transesophageal echocardiography revealed left atrial spontaneous echo contrast, increasing the likelihood of a left atrial thrombus as a cause of cerebral embolism. Six of the eight patients with myocardial infarction had a possible cardiac source of embolism identified with transesophageal echocar-
diography. One of the four patients with apical dyskinesis had spontaneous echo contrast in the left atrium, raising the possibility of a left atrial source of embolism. Two of the four patients with inferior myocardial infarction had left atrial spontaneous echo contrast, and one of these had a left atrial appendage thrombus and a mobile right atrial thrombus. Both of these patients were in atrial fibrillation. The patient with mitral valve endocarditis had improved resolution of the vegetation attached to the atrial aspect of the posterior mitral leaflet with transesophageal echocardiography. Of the 12 pa-
patients with atrial fibrillation not associated with myocardial infarction or mitral stenosis, nine had abnormal transthoracic echocardiographic findings: one patient had mitral annular calcification also seen on the transthoracic echocardiogram, seven had left atrial spontaneous echo contrast (including one patient with a left atrial appendage thrombus and a second patient with a right atrial thrombus), and one had a myxomatous mitral valve with severely thickened leaflets and a small perforation in the posterior mitral leaflet. Only three patients with atrial fibrillation had negative transthoracic echocardiograms.

In group II, all 39 patients had a negative transthoracic echocardiogram. However, the transesophageal echocardiogram was abnormal in seven (18%). Two patients had a patent foramen ovale with right-to-left shunting diagnosed by contrast study and color flow imaging (one had an associated atrial septal aneurysm), two patients had a myxomatous mitral valve (with prolapse in one), one patient had an isolated left atrial appendage thrombus (Figure 2), and two patients had atrial septal aneurysms without evidence of shunting.

The thoracic aorta was carefully examined with biplane transesophageal echocardiography in all patients. Although atherosclerotic plaques were frequently detected, no protruding thrombus was identified.

Of the 63 patients, 26 (41%) ultimately had a potential cardiac source of embolism identified (Table 1). The patients with a potential cardiac source of embolism were older (67.5 versus 59.4 years, p=0.04), more frequently in atrial fibrillation (62% [16] versus 8% [3], p<0.0001), had a larger left atrium (43 versus 37 mm, p=0.01), and more commonly had left ventricular hypertrophy (62% [16] versus 32% [12], p<0.02) than the patients without an identifiable cardiac source (Table 2).

Of the 63 patients, 44 (70%) were in sinus rhythm and 19 (30%) were in atrial fibrillation. All but three of the patients with atrial fibrillation had a potential cardiac source of embolism identified. The most common cardiac source was left atrial spontaneous echo contrast. This abnormality was detectable only by transesophageal echocardiography (Table 1).

Follow-up was available in 58 patients. Only three (5%) had a change in management because of the results of transesophageal echocardiography. One patient in sinus rhythm with a left atrial appendage thrombus received anticoagulants. A patient with restrictive cardiomyopathy and atrial fibrillation received anticoagulants after transesophageal echocardiography demonstrated a fixed right atrial thrombus and spontaneous echo contrast in the left atrium. A third patient had anticoagulation therapy discontinued after transesophageal echocardiography did not reveal any cardiac source of embolism.

**Discussion**

This study demonstrates the increased yield of transesophageal echocardiography in the detection

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**TABLE 1. Cardiac Abnormalities Detected by Echocardiography in 26 Patients With Stroke or Transient Ischemic Attacks**

<table>
<thead>
<tr>
<th>Cardiac abnormality</th>
<th>Detected by transthoracic echocardiography</th>
<th>Detected by transesophageal echocardiography</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left atrial spontaneous echo contrast</td>
<td>0</td>
<td>2</td>
<td>9</td>
</tr>
<tr>
<td>With left atrial appendage thrombus</td>
<td>0</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Without left atrial appendage thrombus</td>
<td>3</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Mitral stenosis± spontaneous echo contrast</td>
<td>4</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Apical dyskinesis± apical thrombus</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Mitral vegetation</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Mitral annular calcification (severe)</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Patent foramen ovale</td>
<td>0</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Isolated left atrial appendage thrombus</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Atrial septal aneurysm</td>
<td>0</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

Values are number of patients.

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**TABLE 2. Clinical Characteristics of Patients With and Without Cardiac Source of Embolism**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>With (n=26)</th>
<th>Without (n=37)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean±SD years)</td>
<td>68±12</td>
<td>59±17</td>
<td>0.04</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>12 (46%)</td>
<td>20 (54%)</td>
<td>NS</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>16 (62%)</td>
<td>8 (22%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Left atrial size (mean±SD mm)</td>
<td>43±11</td>
<td>37±7</td>
<td>0.01</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>16 (62%)</td>
<td>12 (32%)</td>
<td>&lt;0.02</td>
</tr>
</tbody>
</table>

NS, not significant.
of a potential cardiac source of embolism in stroke patients compared with transthoracic echocardiography. This is in keeping with previous studies. Pop et al. found that five (9%) of 53 patients with recent transient ischemic attack or stroke and without clinical cardiac abnormalities had an abnormal transesophageal echocardiogram. Six (32%) of 19 patients with a clinically suspected cardiac source of embolism had both a positive transesophageal echocardiogram and a positive transthoracic echocardiogram. The lower yield of transesophageal echocardiography in that study may be due to the lack of color flow imaging and contrast studies to detect intracardiac shunts. The proportion of patients with atrial fibrillation is not mentioned and was likely lower than in our study. Zenker et al. found that in nine (45%) of 20 patients with a cerebral ischemic event under the age of 45 years and a normal transthoracic echocardiogram, transesophageal echocardiography showed pathological findings, which consisted primarily of mitral valve prolapse, atrial septal defects, and atrial aneurysms. In the European Multicenter Study reported by Daniel et al., 479 patients with unexplained arterial embolism were studied with transthoracic and transesophageal echocardiography. The majority of these patients had had a cerebral ischemic event. Potential sources of arterial embolism were detected by transthoracic echocardiography in 176 (37%) of 479 patients and by transesophageal echocardiography in 310 (65%) of 479 patients. Transesophageal echocardiography identified mitral valve prolapse, patent foramen ovale, left atrial and atrial appendage thrombi, spontaneous echo contrast, atrial septal aneurysm, and valvular vegetations significantly more frequently than transthoracic echocardiography. The yield of transesophageal echocardiography was higher in that study because of the inclusion of patients with peripheral arterial emboli.

In our study, transthoracic echocardiography did not reveal any potential cardiac source of embolism in patients without clinically evident cardiac disease. This is in keeping with previous reports. In 38% of the patients with clinical evidence of heart disease, a potential cardiac source of embolism was detected by transthoracic echocardiography. The incidence of transthoracic echocardiographic abnormalities in stroke patients varies from 4% to 47% depending on the criteria used. A definitive source of embolism such as valvular vegetations, thrombus, or myxoma is detected in very few patients.

Patients with clinical evidence of cardiac disease frequently have a potential cardiac source of embolism detected by transesophageal echocardiography. Although its diagnostic yield in patients without obvious heart disease is low, transesophageal echocardiography may have therapeutic impact and should be performed in young patients or those with multiple strokes or systemic emboli.

Transesophageal echocardiography is superior to transthoracic echocardiography for the detection of atrial thrombus, patent foramen ovale, atrial septal aneurysm, left atrial spontaneous echo contrast, and myxomatous degeneration of the mitral valve associated with mitral valve prolapse. All of these cardiac abnormalities may be a source of embolism.

Atrial fibrillation is a well-known contributor to stroke. In our study only three of the 19 patients with atrial fibrillation had a transesophageal echocardiogram negative for a cardiac source of embolism. Belder et al. recently found that patients in atrial fibrillation with left atrial spontaneous echo contrast were four times more likely to have suffered a thromboembolic event than patients in atrial fibrillation without spontaneous echo contrast. In our study, spontaneous echo contrast was detected by transesophageal echocardiography in 13 of the 19 patients with atrial fibrillation. Two patients with spontaneous echo contrast had a left atrial appendage thrombus. In patients with atrial fibrillation the importance of transesophageal echocardiography may lie in detecting with greater certainty those with "lone atrial fibrillation" who do not have any underlying heart disease. This will prevent the risks of long-term anticoagulation in such patients.

One of the major limitations of this study is the lack of an age-matched control group of volunteers without strokes. The cardiac abnormalities associated with systemic embolism and detected by transesophageal echocardiography can also occur in persons who have not had strokes although the prevalence of all these abnormalities is higher in patients with strokes.

Saline contrast studies were not performed during transthoracic echocardiography. The two patients with a patent foramen ovale detected by color flow imaging only on transesophageal echocardiography might have had a positive saline contrast study with transthoracic echocardiography, thus improving the diagnostic yield of this procedure. Other studies have suggested that transthoracic contrast echocardiography is both insensitive and nonspecific for the detection of right-to-left shunting through a patent foramen ovale.

Because of the delay from onset of the cerebral ischemic event to transesophageal echocardiography, certain abnormalities such as left atrial appendage thrombi may have been missed. Apical thrombi may also have resolved following the embolic episode. However, all patients with apical dyskinesia, even those without the presence of thrombus, were considered to have a potential cardiac source of embolism.

Because of the high incidence of concomitant carotid artery disease in patients with a potential cardiac source of embolism, the definitive cause of stroke in these patients cannot be established. The total prevalence of a possible cardiac source of embolism in our 63 patients with stroke is therefore higher than the 15% reported in the literature. Bogousslavsky et al. found that 19% of patients with carotid-territory transient ischemic attacks had a
potential cardiac source of embolism associated with appropriate carotid artery disease. Rem et al. found that 29 (49%) of 59 patients with a possible cardiac source of cerebral embolism also had a vascular lesion in an appropriate carotid artery and that definitive diagnosis of the cause of cerebral embolism was not possible.

Although we attempted to avoid referral bias by performing transesophageal echocardiography on the first three patients with stroke referred to the echocardiography laboratory each week, bias could not be entirely eliminated. Echocardiography was probably more frequently requested by neurologists for patients with a suspected cardiac source of embolism as evidenced by the high percentage of patients with atrial fibrillation.

Transesophageal echocardiography is superior to transthoracic echocardiography in the detection of a cardiac source of embolism in patients with a cerebral ischemic event. Transesophageal echocardiography is most useful in patients with clinical evidence of heart disease. A small percentage of patients with a clinically unsuspected cardiac source of embolism will have an abnormal transesophageal echocardiogram. Transthoracic echocardiography is not useful in such patients. The group of patients with the highest yield from transesophageal echocardiography are those with atrial fibrillation, an enlarged left atrium, advanced age, and left ventricular hypertrophy.

It is debatable whether transesophageal echocardiography should be performed in all patients with stroke in the absence of clinical suspicion of a cardiac embolic source. Transesophageal echocardiography is probably most indicated in young patients or those with several embolic events in whom the superior detection of a patent foramen ovale, myxomatous mitral valve, atrial septal aneurysm, or left atrial thrombus may alter therapy.

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References


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