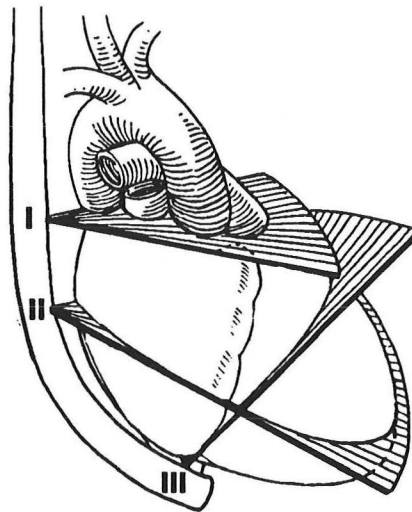


Clinical Applications of Transesophageal Echocardiography

Paul A. Grayburn, M.D.



Internal Medicine Grand Rounds

August 26, 1993

Echocardiography has become the single most widely used cardiac imaging modality because it provides real-time noninvasive assessment of the anatomy and physiology of the heart. Unfortunately, high quality echocardiograms cannot be obtained in all patients because of obesity, chest wall abnormalities, pulmonary disease, and mechanical ventilation, all of which interfere with transmission of ultrasound through the chest wall. Accordingly, transesophageal echocardiography (TEE) was developed primarily to overcome these limitations in the 10-15% of patients with "technically difficult studies."

TEE was first reported by Frazin in 1976 using M-mode echocardiography (1). However, the technique did not become widely used until the late 1980's when technological improvements enabled phased array transducers to be placed on the tip of a standard gastroscope (2-4). Over the past 5 years, TEE has emerged as an important clinical tool for the assessment of various cardiovascular disorders. The purpose of these Grand Rounds is to review the strengths and weaknesses of TEE and to discuss its current clinical applications. It should be emphasized that TEE is a young technique that is still evolving clinically and technologically.

Guidelines for Performing TEE

Physician Training. TEE should only be performed by a physician with proper training in esophageal intubation and sophisticated understanding of cardiac anatomy, pathophysiology, and echocardiography (5). The former can be obtained under the tutelage of an experienced gastroenterologist. The latter is best obtained by cardiology fellowship. Other physicians who wish to learn TEE should spend at least 6 months training in an experienced echocardiography laboratory (5).

Procedure. Awake patients are studied in the left lateral decubitus position to avoid aspiration. Patients are kept NPO overnight unless the study is emergent (ie aortic dissection). Topical anesthesia is utilized to suppress the gag reflex and conscious sedation is achieved with intravenous midazolam and meperidine. Blood pressure, ECG, and oximetry are monitored during the procedure by a nurse. The American Heart Association does not recommend routine SBE prophylaxis for upper endoscopy unless biopsies are taken (6). In our laboratory, SBE prophylaxis is administered only for patients with prosthetic heart valves.

Contraindications. Patients with esophageal pathology, including varices, strictures, carcinoma, or diverticulae should not undergo TEE. Patients who are at risk of atlantoaxial dislocation with neck flexion should not undergo TEE. Finally, patients with severe lung disease may have respiratory compromise during TEE.

Safety. TEE has been shown to be a safe procedure. Daniel, et al reported the experience of 15 European centers in 10,419 consecutive patients (7). Unsuccessful esophageal intubation occurred in 1.9% of patients and the TEE was aborted due to intolerance of the probe in 0.6%. There were 18 complications (0.18%) including death (n=1), bronchospasm (n=6), ventricular tachycardia (n=3), atrial fibrillation (n=3), hypoxia (n=2), AV block (n=1), angina (n=1), and minor pharyngeal bleeding (n=1). The patient who died exsanguinated from an esophageal perforation at the site of an undiagnosed esophageal carcinoma. In a smaller study of the safety of TEE in critically ill patients in an intensive care unit (n=62), there were no complications (8).

Anatomic Considerations

As an ultrasound beam passes through the body, it is reflected and refracted by the various tissues that it encounters. Thus, the strength of the signal returning to the transducer is attenuated in the far field of the image as compared to the near field. The practical application of ultrasound attenuation is that structures far away from the transducer are not visualized as well as structures closer to the transducer. Transthoracic echocardiography, in which the transducer is placed on the chest wall is limited in its ability to visualize posterior cardiac structures such as the left atrium, the left atrial appendage, and the pulmonary veins. These structures are well visualized by TEE because the transducer in the esophagus is posterior to the heart. On the other hand, TEE generally does not image certain anterior structures, such as the pulmonic valve and left ventricular apex, as well as transthoracic echocardiography.

Figure 1 shows the primary imaging planes for single plane TEE. The basal position (labeled I) allows short-axis views of the base of the heart. From this view, the left atrium, left atrial appendage, pulmonary veins, aortic valve, and pulmonary veins can be seen. Slightly advancing the probe to position II yields the 4 chamber view, which shows the atrioventricular valves, interatrial septum, interventricular septum, and all 4 cardiac chambers. Advancing the probe into the stomach (position III) yields the transgastric short-axis view of the left ventricle.

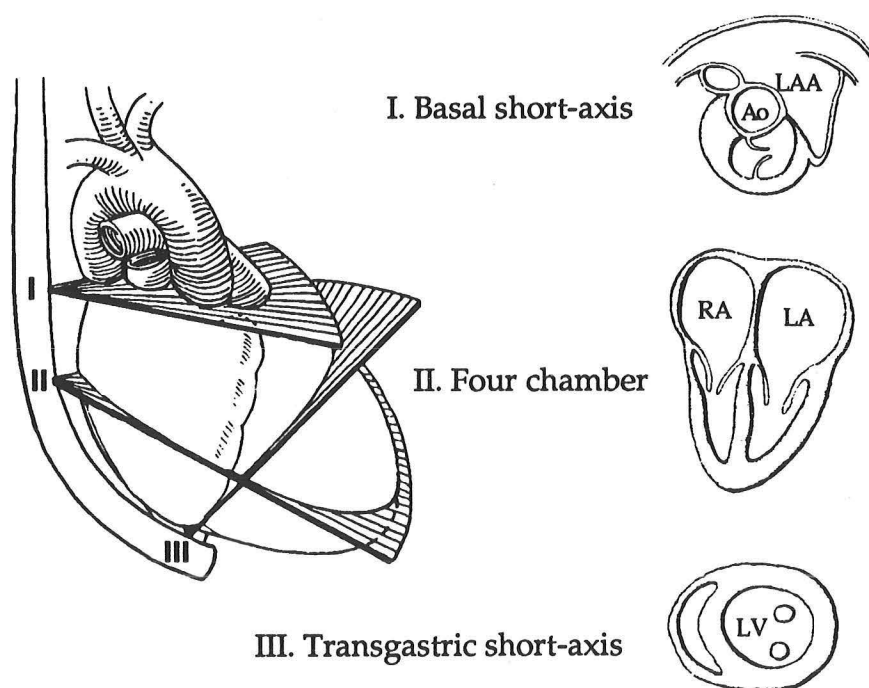


Figure 1. Primary imaging locations for single plane TEE.

Initial TEE probes were limited to a single imaging plane transverse to the probe (figure 2). Subsequently, biplane probes were developed with allowed long-axis views of the heart and improved the ability to image the ascending aorta, interatrial septum, left ventricular outflow tract, and tricuspid valve (3). The recent development of multiplane TEE allows complete rotation of the imaging plane and greatly improves the ability to visualize the aortic valve, pulmonic valve, ascending aorta, and pulmonary veins. Because multiplane TEE is inherently 3-dimensional, it offers more complete evaluation of structures that are well seen by single plane TEE (ie the mitral valve and left ventricle).

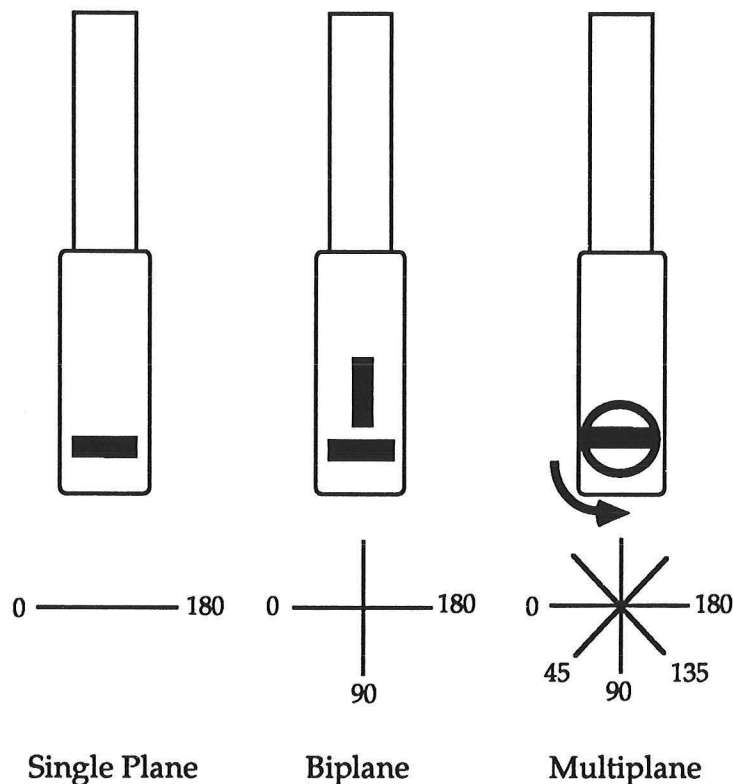


Figure 2. Evolution of TEE transducers.

Because the TEE probe is limited to the esophagus, it is not well suited to performing pulsed and continuous wave Doppler measurements of velocities across stenotic valves. Transthoracic echocardiography (TTE) allows interrogation of velocities from multiple sites (apex, parasternal, suprasternal, subxiphoid) and is therefore much better than TEE for calculating aortic and mitral valve areas, pressure gradients, and cardiac outputs. In the case of Doppler color flow mapping, TTE is superior to TEE in detecting pulmonic and tricuspid regurgitation. TEE is superior in detecting atrial septal defects, patent foramen ovale, and prosthetic valve regurgitation. Both techniques are good for detection of mitral regurgitation, aortic regurgitation, and ventricular septal defects.

CLINICAL APPLICATIONS

Aortic dissection

Aortic dissection is a life-threatening condition requiring prompt diagnosis and treatment. The prognosis of untreated aortic dissection is dismal with 25% mortality within 24 hours, 50% within 1 week, 75% within 1 month, and 90% within 1 year (9). Dissection involving the ascending aorta (DeBakey Type I and II) requires surgical therapy, whereas uncomplicated dissection limited to the descending aorta (Type III) is managed medically (10). Recently, a consensus has emerged that TEE and MRI are superior to angiography and CT scanning in diagnosing and classifying aortic dissection (11).

Erbel et al (12), compared the diagnostic accuracy of TEE, CT scanning, and aortography in 164 patients, 82 of whom had confirmed aortic dissection. The results are shown in table I.

Table I. Diagnostic accuracy of TEE, CT, and angiography in aortic dissection.

Test	Sensitivity	Specificity	+ Pred Value	- Pred Value
TEE	99%	98%	98%	99%
CT	83%	100%	100%	86%
Angio	88%	94%	96%	84%

Other investigators have also reported that TEE has a diagnostic accuracy of nearly 100% in classifying aortic dissection (13,14). In addition, TEE is highly accurate in identifying the entry site of the dissection, thrombosis of the false lumen, aortic regurgitation, and pericardial effusion (13-16). Nienaber et al (17,18), have reported a lower specificity of TEE in identifying proximal dissection due to false positive results in the ascending aorta. Their data showed that both TEE and MRI had sensitivities of nearly 100% but that the specificity of TEE for ascending aortic dissection was only 68% and 77% in two separate studies (17,18). Unfortunately, they used angiography rather than surgical or autopsy confirmation as the gold standard. In addition, the diagnosis of dissection by TEE was made by identification of a flap in the lumen. It has been shown that the appearance of a flap in the ascending aorta by single plane TEE can be emulated by reverberation artifacts (19). Had Nienaber required the identification of another TEE finding such as entry site, thrombosis of the false lumen, aortic regurgitation, or pericardial effusion, all of the 7 false positive studies in his first study (17) would have been correctly classified. Such information is not available in his second study. Perhaps even more importantly, the development of multiplane TEE now offers superior visualization of the ascending aorta. Thus, the artifacts that may yield false positive results in the ascending aorta by single-plane TEE can now be clearly distinguished by multiplane imaging.

Table II compares angiography, CT, MRI, and TEE in the assessment of aortic dissection. As can be seen, both TEE and MRI are highly accurate in making the diagnosis and identifying pericardial effusion. MRI has an advantage in identifying

branch vessel involvement, whereas TEE has a slight advantage in identifying aortic regurgitation and coronary artery involvement. Because TEE is portable and readily available, it is the diagnostic method of choice for the initial evaluation of patients with aortic dissection. If the TEE shows a proximal dissection with aortic regurgitation, pericardial effusion, or clear identification of the entry site, the patient can be taken to the operating room without further workup. If a proximal flap is seen without any other findings in a stable patient, an MRI should be obtained to confirm the diagnosis. If the TEE shows a distal dissection, medical therapy can be instituted and MRI can be obtained later if it is needed to assess branch vessel involvement (ie, progressive deterioration in renal function). A negative TEE is sufficient to exclude the diagnosis of aortic dissection.

Table II. Comparison of angiography, CT, MRI, and TEE in aortic dissection.

	Angiography	CT	MRI	TEE
diagnostic accuracy	++	++	+++	+++
aortic regurgitation	+++	-	+	+++
pericardial effusion	-	++	+++	+++
branch vessel involvement	+++	+	+++	+
coronary involvement	++	-	++	++

+++ excellent; ++ good; + fair; - not detected

Endocarditis

Transthoracic echocardiography (TTE) has been widely used to evaluate the presence and size of vegetations, the degree of valvular regurgitation, and left ventricular function in patients with known or suspected endocarditis. However, TTE is only moderately sensitive in detecting vegetations found at surgery or autopsy, and is very insensitive in detecting perivalvular abscess. Thus, TEE has emerged as a superior imaging tool for the assessment of endocarditis.

Mugge et al (20), compared TTE and TEE in assessing both the presence and prognostic significance of vegetations in a prospective series of 105 consecutive patients. Patients with vegetations >10 mm had a higher incidence of embolic events than those with vegetations ≤ 10 mm (22 of 47 versus 11 of 58, $p < 0.01$). There was no association between vegetation size and congestive heart failure or death. In a subset of 80 patients in whom the echo findings were confirmed by autopsy or surgery, sensitivity was 96% for TEE and 77% for TTE. Specificity was not reported. Shively et al (21), reported a sensitivity and specificity for TEE of 94% and 98%, respectively compared to 69% and 92% for TTE. Although it is clear that TEE is much more accurate than TTE for detecting vegetations, several caveats must be kept in mind. First, vegetations may represent old, healed endocarditis rather than active infection. Second, it may be difficult to distinguish vegetation from prolapsed, flail, or thickened leaflets. Finally, acoustic shadowing from prosthetic valves may mask the presence of vegetations.

Daniel et al (22), evaluated the ability of TEE to detect perivalvular abscesses in 118 consecutive patients with endocarditis confirmed at autopsy or surgery. A total

of 46 abscesses were found at surgery or autopsy in 44 patients. TEE had a sensitivity of 87% and a specificity of 95%. In comparison, TTE has a sensitivity of only 28%. All 44 patients with abscesses either died or underwent surgery for heart failure or persistent fever. Similar findings were reported by Karalis et al (23), who found that aortic valve abscesses often spread to the mitral valve causing leaflet perforation and extensive destruction of the mitral-aortic annular fibrosa. Thus, TEE performed early in the course of endocarditis offers the ability to detect abscesses and perform surgery before the patient develops major complications and before progressive tissue destruction.

The role of TEE in endocarditis is evolving. Clearly, all patients with fever and suspected endocarditis do not need a TEE. Patients with right-sided endocarditis generally have a good prognosis (24) and there is evidence to suggest that TEE is no better than TTE in diagnosing right-sided vegetations (25). However, all patients with left-sided or prosthetic valve endocarditis should undergo TEE early in their course to exclude an abscess. The presence of an abscess or severe valvular regurgitation warrants early valve replacement (26). Patients with large, mobile vegetations on the initial TEE may be considered for surgical debridement of the valve to prevent embolic complications, although this remains somewhat controversial. If an abscess is not present and there is no evidence of severe valvular regurgitation, appropriate antibiotic therapy should be instituted. Repeat TEE should be performed if the patient develops congestive heart failure, AV block, or persistent fever. Although a negative TEE does not "rule out" endocarditis, it is associated with a good prognosis and a high likelihood that another source of fever will be found (27).

Mitral Valve Disease

Because TEE provides excellent visualization of the mitral valve, it is particularly useful in assessing mitral valve disease. Mitral stenosis is easily identified and accurately quantitated by TTE (28). TTE is also very accurate in determining whether a stenotic mitral valve is suitable for balloon valvuloplasty or surgical commissurotomy (29). Accordingly, the role of TEE in mitral stenosis is generally limited to patients with technically difficult TTE, or the need to assess the presence of left atrial thrombus prior to balloon valvuloplasty (30).

TEE plays a much greater role in assessing the structure and function of the mitral valve apparatus in patients with mitral regurgitation. This is largely due to the development of mitral valve reparative techniques (31,32). Valve repair has a much lower surgical mortality (33) and does not result in postoperative depression of left ventricular function (34). TEE provides high resolution images of the anatomic details of the papillary muscles, chordae, leaflets, and annulus, enabling these structures to be viewed simultaneously as a functional unit (35). TEE is also capable of estimating the severity of mitral regurgitation (36-38). Thus, TEE has become the method of choice to determine which patients are candidates for valve repair and to decide which type of repair is needed (39-41). Intraoperative TEE is now routinely used to assess the adequacy of mitral valve repair (42).

Prosthetic Valves

Doppler echocardiography has greatly facilitated the noninvasive assessment of prosthetic valve integrity (43,44). Nevertheless, several problems have limited the ability of TTE to detect prosthetic valve dysfunction. Obviously, technically difficult studies occur in some patients. More importantly, acoustic shadowing by the prosthetic valve itself renders detection of vegetations and perivalvular leaks difficult. Acoustic shadowing refers to the inability of the ultrasound beam to penetrate a mechanical prosthesis such that structures lying on the other side of the prosthesis cannot be visualized. TEE can partly overcome these difficulties by providing an acoustic window that lies behind the prosthesis. This is especially true for the mitral position, where perivalvular mitral regurgitation is masked by the prosthesis from standard TTE views. Importantly, all normally functioning mechanical prostheses have small regurgitant leaks; TEE can distinguish these from abnormal regurgitation. Khandheria et al (45), found a sensitivity of 96% and specificity of 98% in detecting abnormalities of mitral valve prostheses in 50 patients undergoing catheterization and/or valve replacement. Of the abnormalities detected by TEE, 48% were missed by TTE. Daniel et al (46), studied 126 patients in whom prosthetic valve dysfunction was confirmed at surgery. TTE only identified 65% of the abnormal mitral valves compared to 97% for TEE. Aortic valve dysfunction was only detected in 50% by TTE and 77% by TEE. When analyzed by type of prosthesis, TTE identified 65% of abnormal bioprostheses and 22% of abnormal mechanical prostheses. In contrast, TEE correctly detected 87% of abnormal bioprostheses and 83% of abnormal mechanical prostheses. Thus, TEE is clearly superior to TTE in assessing prosthetic valve dysfunction. TEE is superb in assessing prosthetic mitral valves and good for aortic valves. TTE probably remains superior in determining bioprosthetic valve area for suspected stenosis of aortic bioprostheses.

Routine followup of patients with prosthetic valves depends on physical examination and TTE. For patients with suspected stenosis of a bioprosthesis, TTE is sufficient in most instances. However, patients with suspected thrombosis, infection, or regurgitant leak in a mechanical prosthetic valve, TEE is the procedure of choice.

Cardiac Source of Embolus

Although evaluation of cardiac source of embolus is currently the most common reason for performing TEE, this remains a very controversial topic. Stroke is a major cause of death and disability and it is estimated that 15-20% of strokes are cardioembolic in origin (47). Unfortunately, traditional clinical features of embolic stroke, such as abrupt onset, middle cerebral artery distribution, and seizures, are not reliable (47,48). Two-dimensional echocardiography has been shown to have an extremely low yield in detecting intracardiac thrombus in patients without overt clinical evidence of heart disease (49,50). Because TEE is far superior to TTE in imaging the left atrial appendage, there has been hope that TEE will improve the ability to identify cardioembolic strokes. Table III lists studies comparing TTE and

TEE in assessing potential sources of embolism in patients with strokes or TIA's (51-56). One study by Zenker et al (57) is excluded because it found mitral valve prolapse in 50% of patients and inappropriate diagnostic criteria for prolapse were used.

Table III. Studies comparing TTE and TEE in stroke/TIA patients.

Study	Patients (n)	PSE by 2D Echo (%)	PSE by TEE (%)
Pop (51)	72	10	24
Hofmann (52)	153	36	58
Pearson (53)	79	15	57
Cujec (54)	63	14	41
DeRook (55)	66	14	57
Lee (56)	50	0	52

At first glance, this table suggests that TEE shows a high prevalence of potential cardiac embolic sources. However, there are several limitations to these data. First, none of the studies were controlled. It is very likely that TEE would have found potential sources of embolism in appropriate control groups of similar age and risk factors. Second, most of the studies (51-53,56) specifically excluded patients with carotid disease, such that the data represent a selected group of patients in whom cardioembolic stroke is more likely than stroke due to cerebrovascular disease. Finally, most of the potential sources of embolism are of questionable significance and would not lead to anticoagulation. Table IV lists the potential sources of embolism and the effect that such a finding would have on management.

Table IV. Potential cardiac sources of embolism found by TEE.

Finding	Associated with Clinical Heart Disease	Effect on Management
Left atrial thrombus	yes	anticoagulate
LV thrombus	yes	anticoagulate
Vegetation	yes	antibiotics
Spontaneous echo contrast	yes	probably anticoagulate
Atrial septal aneurysm	no	uncertain
Aortic atherosclerotic debris	no	uncertain
Patent foramen ovale	no	none
Mitral valve prolapse	no	none
Mitral annular calcification	no	none
Mitral valve "strands"	no	none
Valve thickening	no	none

Likely Sources of Embolism. It is useful to consider the above TEE findings as either likely or possible sources of embolization. Clearly, intracardiac thrombus would mandate anticoagulation. Although intracardiac thrombus is found in 10-12% of patients with stroke in the aforementioned studies, it is almost always associated with clinical evidence of cardiac disease. In the studies of Pop (51), Pearson (53), Cujec (54), and Lee (56), left atrial thrombus was seen in only 2 of

151(1.3%) patients without clinical evidence of heart disease. In contrast, Hofmann (52) reported intracardiac masses in 20 (19%) of 107 patients without clinical heart disease. It is not clear how many of these masses were left atrial thrombus. Importantly, echodensities in the left atrial appendage do not always represent thrombus. Prominent pectinate muscles and calcifications may be mistaken for thrombus.

Spontaneous echo contrast refers to an echodense "swirling" that is thought to be echocardiographically visible stasis of blood flow occurring in the setting of mitral stenosis, atrial fibrillation, or left ventricular dysfunction (57). Black et al (57) reported the prevalence of spontaneous echo contrast in 400 consecutive patients undergoing TEE for various indications. Spontaneous echo contrast was seen in 75 (19%) patients, 71 of whom had atrial fibrillation or mitral stenosis. Three other patients had severe left ventricular dysfunction and one had marked sinus bradycardia. Thus, while spontaneous echo contrast is generally thought to be associated with thromboembolism (59), it is rarely found in patients without clinically apparent heart disease. In the studies of Pop (51), Pearson (53), Cujec (54), and Lee (56), no stroke patient without clinical evidence of heart disease had spontaneous echo contrast. Although there are no data specifically addressing the need to anticoagulate patients with spontaneous echo contrast, most such patients already have a cardiac condition for which anticoagulation is indicated.

Possible Sources of Embolism. Atrial septal aneurysm has been associated with stroke (60,61). The mechanism for this remains uncertain, but is thought to be thrombus formation within the aneurysm or paradoxical embolism through a concomitant patent foramen ovale. Although the treatment for atrial septal aneurysm is uncertain, anticoagulation should be considered in patients with unexplained stroke.

Although paradoxical embolism is known to occur through a patent foramen ovale, it is probably uncommon (62). Patent foramen ovale is a normal variant that occurs in 25-30% of individuals. Therefore, it should not be surprising that it is frequently found in stroke patients. Paradoxical embolism through a patent foramen ovale requires a source of venous thrombus, pulmonary embolism, or rarely, venous air embolism (62). A stroke should not be attributed to a patent foramen ovale unless a such a source is found in a patient with otherwise unexplained stroke. Similarly, mitral valve prolapse, mitral annular calcification, and mitral valve "strands," although associated with stroke in some series, may not be directly causative and are not indications for anticoagulation.

Aortic atherosclerotic debris identified by TEE in the thoracic aorta has been associated with stroke (63-65). Because it is difficult to imagine that descending aortic "debris" embolizes backwards into the cerebral circulation, it may merely be a marker for diffuse atherosclerosis involving the cerebral circulation. Occasionally, a large protruding atheroma in the ascending aorta is seen and may well be a source of embolism to the brain. The treatment for this condition is uncertain. Both coumadin, aspirin, and surgical removal have been proposed (65).

Parkland Stroke Data. Brickner et al (66), reported the TEE findings of 200 consecutive stroke patients admitted to Parkland Memorial Hospital. Patients with documented stroke or TIA's were prospectively evaluated by history, physical

examination, TTE, and TEE. Patients were grouped according to the presence of clinically apparent heart disease (n=65), hypertension (n=91), and no apparent cardiovascular disease (n=44). Likely sources of embolism were considered to be intracardiac thrombus, prosthetic heart valves, mitral stenosis, or vegetations. Potential sources of embolism were defined as spontaneous echo contrast, atrial septal aneurysm, aortic atherosclerotic debris, mitral valve prolapse, mitral annular calcification, and mitral valve strands. Patent foramen ovale was not included as a source of embolism because it is a normal variant that occurs in 25-30% of the population. Figure 3 shows the results of the study according to patient group. As can be seen, likely sources of embolism were rarely found in patients without clinically apparent heart disease.

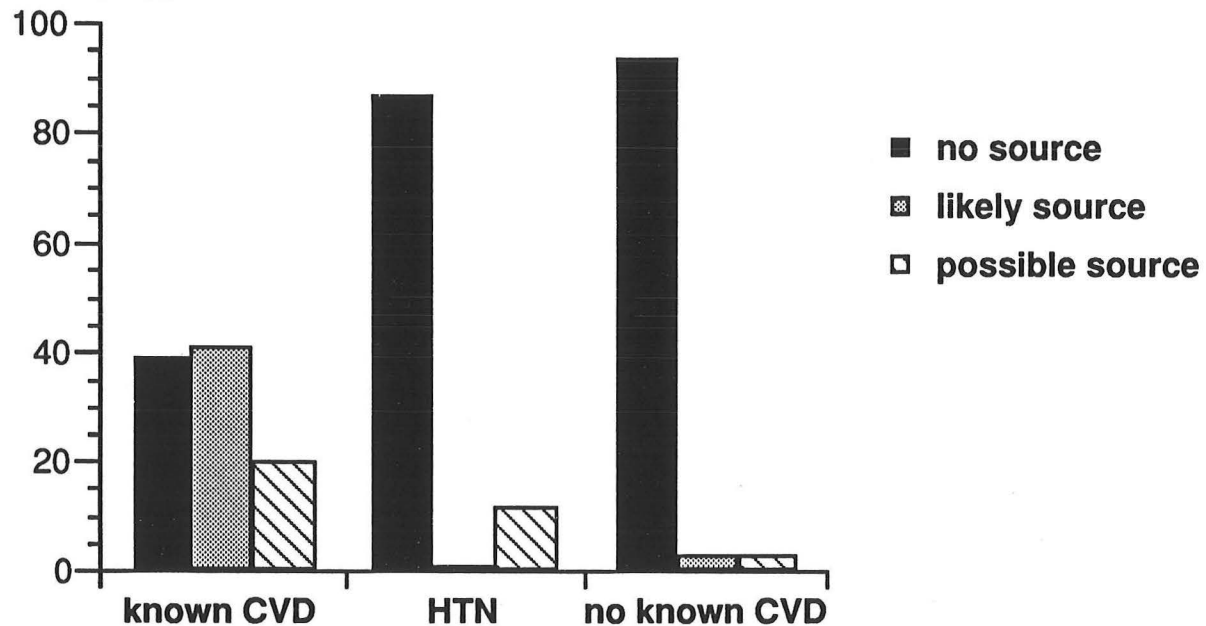


Figure 3. TEE findings in 200 consecutive stroke/TIA patients at Parkland.

As a general rule, the reason to perform TEE in stroke patients is to identify a source of embolism that would require anticoagulation. Because there is a risk to anticoagulating stroke patients, only definite cardioembolic sources such as left atrial thrombus would result in such therapy. Thus, although TEE finds "potential" sources of embolism 40-50% of the time, most of these findings are not indications for anticoagulant therapy. Furthermore, it is uncommon for stroke patients with no clinical evidence of heart disease to have left atrial thrombus. Finally, the important question to ask in a stroke patient is not whether there is a clot in the left atrium, but whether the patient is at risk for developing left atrial thrombus. For example, a patient with chronic atrial fibrillation, dilated cardiomyopathy, or mitral stenosis should be anticoagulated regardless of the TEE findings. After all, the TEE may not show left atrial clot if the clot has already migrated to the head. Accordingly, I would propose the following approach to deciding which stroke patients would benefit from TEE.

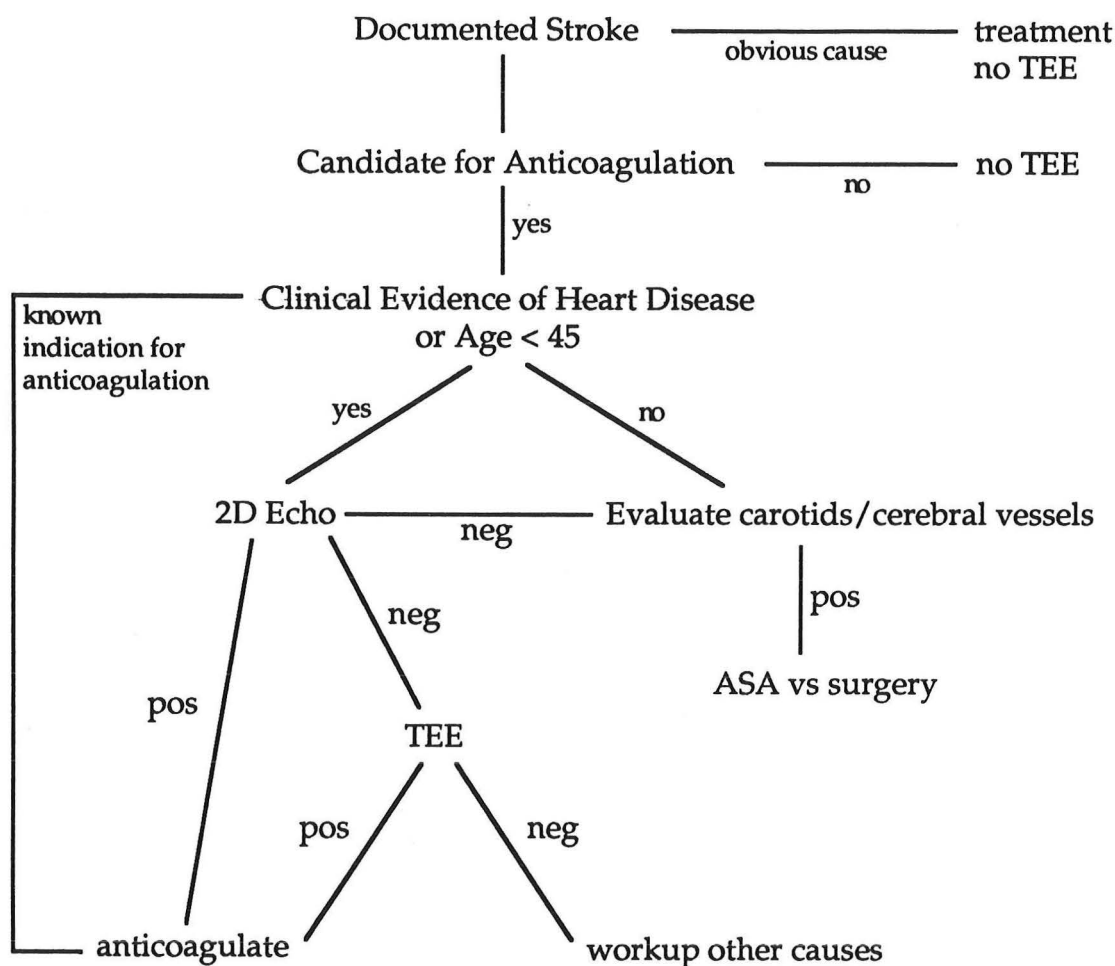


Figure 4. Suggested approach to evaluation of stroke patients by TEE.

Congenital Heart Disease

TEE is not commonly used in infants and children with congenital heart disease because of the size of the probes and the fact that most children make technically good transthoracic echocardiograms. In adults, TEE is useful in a variety of congenital abnormalities including ASD, Ebstein's anomaly, partial anomalous pulmonary venous return, subaortic membranes, and assessment of surgically corrected congenital heart disease.

ASD's are well visualized by TEE and can easily be classified as ostium secundum, ostium primum, or sinus venosus type. In addition, the presence of association partial anomalous pulmonary venous return can be assessed. Color flow mapping can identify the direction and magnitude of shunting across the interatrial septum (67).

Technically Difficult Echocardiogram

Critically ill patients in the intensive care unit often make poor TTE studies because of mechanical ventilation or inability to properly position the patient. TEE may be very useful in such patients to assess LV function, valvular lesions, intracardiac shunts, or mechanical complications of acute myocardial infarction (68). Calculation of LV volumes and ejection fraction is possible but tends to underestimate angiographic volumes because of failure of the imaging plane to transect the true LV apex (69).

Intraoperative Monitoring

As noted previously, TEE is very useful intraoperatively in assessing the results of mitral valve repair. It is also useful in valve replacement to assess perivalvular leaks, restriction of disk excursion, and left ventricular function after surgery (70). Patients with impaired left ventricular function may benefit from TEE during bypass surgery to evaluate postoperative LV function and the need for hemodynamic support.

There has been great interest in using intraoperative TEE to monitor wall motion as a marker for ischemia in patients undergoing non-cardiac surgery (71,72). However, the only prospective trial of this has shown that intraoperative TEE has little incremental value over 2-lead ECG monitoring (73). In this study, 332 men undergoing noncardiac surgery underwent monitoring with 2-lead ECG, 12-lead ECG, and TEE. These techniques were assessed for evidence of intraoperative ischemia by observers who were blinded to the postoperative outcomes of death, myocardial infarction, or unstable angina. A total of 111 patients had evidence of intraoperative ischemia by at least one monitoring technique. Table V illustrates the data.

Table V. Intraoperative monitoring of ischemia by TEE and ECG.

Method	Odds ratio (95% CI)	Intra-op ischemia	Ischemic outcome
2-lead ECG	3.0 (0.9-11.0)	73	5
12-lead ECG	2.4 (0.6-9.5)	46	3
TEE	2.2 (0.6-8.5)	50	3

As can be seen, neither TEE nor 12-lead ECG offered any incremental benefit over 2-lead ECG in predicting postoperative outcomes. This is partly because an apical ischemic event would be missed by monitoring LV function in the short-axis view. In addition, wall motion is by far the most difficult aspect of echocardiography to learn and it is easy to either miss minor wall motion abnormalities or overstate small changes resulting from alterations in heart rate or loading conditions. Given that no data are available to indicate that detection of wall motion abnormalities by intraoperative TEE alters patient management or outcome, TEE should not be routinely used for intraoperative monitoring.

Aortic Valve Disease

Single-plane TEE produces off-axis images of the aortic valve and therefore has not been accurate in assessing the severity of aortic stenosis or regurgitation. This may change with the development of multiplane TEE. The multiplane technique offers spectacular short-axis and long-axis views of the aortic leaflets and should prove to be valuable in assessing aortic regurgitation. TEE will not supplant TTE for aortic stenosis because the esophagus provides only limited windows for aligning the Doppler beam with the stenotic jet. The ability of TEE to assess aortic ring abscess, aortic vegetations, and aortic dissection have already been discussed.

Cardiac Masses

TEE may be indicated to further evaluate cardiac masses noted on TTE. It should be remembered that echocardiography does not make a histologic diagnosis. In most cases, masses will require excisional biopsy to define the diagnosis. However, TEE may be helpful in confirming that a mass is real as opposed to artifact. It may also be better than TTE in defining location, site of attachment, and extension or impingement on adjacent structures.

Cardioversion of Atrial Fibrillation

Recently, it has been reported that TEE may be useful in assessing left atrial thrombus in patients with atrial fibrillation undergoing cardioversion (74). The traditional approach to such patients is to anticoagulate them for 2-3 weeks prior to elective cardioversion. This reduces the risk of embolism during cardioversion from about 5% to 1.6% (75). By using TEE immediately prior to cardioversion, one can identify patients with left atrial thrombus in whom cardioversion can be postponed until after a period of anticoagulation. Patients without left atrial thrombus can be safely cardioverted to sinus rhythm. This approach would eliminate the need for repeat hospitalization, interim anticoagulation, and would offer the potential hemodynamic benefit of earlier restoration of sinus rhythm. Such patients should be started on IV heparin 24 hours before the procedure and coumadin for an uncertain period (probably 6 weeks) because atrial thrombus may form shortly after cardioversion due to the delayed recovery of atrial mechanical function or recurrence of atrial fibrillation.

SUMMARY

TEE is valuable cardiac imaging tool that continues to evolve clinically and technologically. It is currently the procedure of choice for diagnosis of aortic dissection. It is also useful in evaluating endocarditis, mitral valve disease, prosthetic heart valves, and congenital heart disease. Although the role of TEE in stroke is controversial, it appears to be useful in selected patients. Intraoperative TEE plays an important role in cardiac surgery but is not routinely indicated to monitor ischemia in patients undergoing noncardiac surgery.

REFERENCES

1. Frazin L, Talano JV, Stephanides L, Loeb HS, Kopel L, Gunnar RM. Esophageal echocardiography. *Circulation* 1976; 54: 102-108.
2. Seward JB, Khandheria BK, Oh JK, et al. Transesophageal echocardiography: technique, anatomic correlations, implementation, and clinical applications. *Mayo Clin Proc* 1988; 63:649-680.
3. Seward JB, Khandheria BK, Edwards WD, Oh JK, Freeman WK, Tajik AJ. Biplanar transesophageal echocardiography: anatomic correlations, image orientation, and clinical applications. *Mayo Clin Proc* 1990; 65:1193-1213.
4. Seward JB, Khandheria BK, Freeman WK, et al. Multiplane transesophageal echocardiography: image orientation, examination technique, anatomic correlations, and clinical applications. *Mayo Clin Proc* 1993; 68:523-551.
5. Pearlman AS, Gardin JM, Martin RP, et al. Guidelines for physician training in transesophageal echocardiography. *J Am Soc Echo* 1992; 5:187-194.
6. Dajani AS, Bisno AL, Chung KJ, et al. Prevention of bacterial endocarditis: Recommendations by the American Heart Association. *JAMA* 1990; 264:2919-2922.
7. Daniel WG, Erbel R, Kasper W, et al. Safety of transesophageal echocardiography: a multicenter survey of 10,419 examinations. *Circulation* 1991; 83:817-821.
8. Pearson AC, Castello R, Labovitz AJ. Safety and utility of transesophageal echocardiography in the critically ill patient. *Am Heart J* 1990; 119:1083-1094.
9. DeSanctis RW, Doroghazi RM, Austen WG, Buckley MJ. Aortic dissection. *N Engl J Med* 1987; 317:1060-1067.
10. DeBakey ME, Henly WS, Cooley DA, Morris GC Jr, Crawford ES, Beall AC Jr. Surgical management of dissecting aneurysms of the aorta. *J Thorac Cardiovasc Surg* 1965; 49:130-149.
11. Cigarroa JE, Isselbacher EM, DeSanctis RW, Eagle KA. Diagnostic imaging in the evaluation of suspected aortic dissection: old standards and new directions. *N Engl J Med* 1993; 328:35-43.
12. Erbel R, Daniel W, Visser C, et al. Echocardiography in diagnosis of aortic dissection. *Lancet* 1989; 1:457-461.

13. Hashimoto S, Kumada T, Osakada G, et al. Assessment of transesophageal Doppler echography in dissecting aortic aneurysm. *J Am Coll Cardiol* 1989; 14:1253-1262.
14. Ballal RS, Nanda NC, Gatewood R, et al. Usefulness of transesophageal echocardiography in assessment of aortic dissection. *Circulation* 1991; 84:1903-1914.
15. Omoto R, Kyo S, Matsumura M, Shah PM, Adachi H, Yokote Y, Kondo Y. Evaluation of biplane color Doppler transesophageal echocardiography in 200 consecutive patients. *Circulation* 1992; 85:1237-1247.
16. Erbel R, Oelert H, Meyer J, et al. Effect of medical and surgical therapy on aortic dissection evaluated by transesophageal echocardiography: implications for prognosis and therapy. *Circulation* 1993; 87:1604-1615.
17. Nienaber CA, Spielmann RP, von Kodolitsch Y, et al. Diagnosis of thoracic aortic dissection: magnetic resonance imaging versus transesophageal echocardiography. *Circulation* 1992; 85:434-447.
18. Nienaber CA, von Kodolitsch Y, Nicolas V, et al. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. *N Engl J Med* 1993; 28:1-9.
19. Appelbe AF, Walker PG, Yeoh JK, Bonitatibus A, Yoganathan AP, Martin RP. Clinical significance and origin of artifacts in transesophageal echocardiography of the thoracic aorta. *J Am Coll Cardiol* 1993; 21:754-760.
20. Mugge A, Daniel W, Frank G, Lichtlen PR. Echocardiography in infective endocarditis: reassessment of prognostic implications of vegetation size determined by the transthoracic and transesophageal approach. *J Am Coll Cardiol* 1989; 14:631-638.
21. Shively BK, Gurule FT, Roldan CA, Leggett JH, Schiller NB. Diagnostic value of transesophageal compared with transthoracic echocardiography in infective endocarditis. *J Am Coll Cardiol* 1991; 18:391-397.
22. Daniel WG, Mugge A, Martin RP, et al. Improvement in the diagnosis of abscesses associated with endocarditis by transesophageal echocardiography. *N Engl J Med* 1991; 324:795-800.
23. Karalis DG, Bansal RC, Hauck AJ, et al. Transesophageal echocardiographic recognition of subaortic complications in aortic valve endocarditis: clinical and surgical implications. *Circulation* 1992; 86:353-362.

24. Hecht S, Berger M. Right-sided endocarditis in intravenous drug abusers: prognostic features in 102 episodes. *Ann Intern Med* 1992; 117:560-566.
25. San Roman JA, Vilacosta I, Zamorano JL, Almeria C, Sanchez-Harguindey L. Transesophageal echocardiography in right-sided endocarditis. *J Am Coll Cardiol* 1993; 21:1226-1230.
26. Middlemost S, Wisenbaugh T, Meyerowitz C, et al. A case for early surgery in native left-sided endocarditis complicated by heart failure: results in 203 patients. *J Am Coll Cardiol* 1991; 18:663-667.
27. Sochowski RA, Chan K-L. Implication of negative results on a monoplane transesophageal echocardiographic study in patients with suspected infective endocarditis. *J Am Coll Cardiol* 1993; 21:216-221.
28. Pearlman AS. Role of echocardiography in the diagnosis and evaluation of mitral and tricuspid stenosis. *Circulation* 1991; 84(Suppl I):I-193-I-197.
29. Abascal VM, Wilkins GT, Choong CY, et al. Echocardiographic evaluation of mitral valve structure and function in patients followed for at least 6 months after percutaneous balloon mitral valvuloplasty. *J Am Coll Cardiol* 1988; 12:606-615.
30. Kronzon I, Tunick PA, Glassman E, Slater J, Schwinger M, Freedberg RS. Transesophageal echocardiography to detect atrial clots in candidates for percutaneous transseptal mitral balloon valvuloplasty. *J Am Coll Cardiol* 1990; 16:1320-1322.
31. Carpentier A, Deloche A, Dauptain J, et al. A new reconstructive operation for correction of mitral and tricuspid insufficiency. *J Thorac Cardiovasc Surg* 1971; 61:1-13.
32. Galloway AC, Colvin SB, Baumann FG, Harty S, Spencer FC. Current concepts of mitral valve reconstruction for mitral insufficiency. *Circulation* 1988; 78:1087-1098.
33. Perier P, Deloche A, Chauvand S, et al. Comparative evaluation of mitral valve repair and replacement with Starr, Bjork, and porcine valve prostheses. *Circulation* 1984; 70(Suppl I): 187-192.
34. Goldman ME, Mora F, Guarino T, et al. Mitral valvuloplasty is superior to valve replacement for preservation of left ventricular function: an intraoperative echocardiographic study. *J Am Coll Cardiol* 1987; 10:568-574.

35. Fehske W, Grayburn PA, Omran H, DeFilippi CR, Manz M, Moosdorf R, Luderitz B. Morphology of the mitral apparatus as displayed by multiplane transesophageal echocardiography. *Am J Cardiol* 1993; submitted.
36. Castello R, Lenzen P, Aguirre F, Labovitz AJ. Quantitation of mitral regurgitation by transesophageal echocardiography with Doppler color flow mapping: correlation with cardiac catheterization. *J Am Coll Cardiol* 1992; 19:1516-1521.
37. Klein AL, Obarski TP, Stewart WJ, et al. Transesophageal echocardiography of pulmonary venous flow: a new marker of mitral regurgitation. *J Am Coll Cardiol* 1991; 18:518-526.
38. Tribouilloy C, Shen WF, Quere J-L, et al. Assessment of severity of mitral regurgitation by measuring regurgitant jet width at its origin with transesophageal Doppler color flow mapping. *Circulation* 1992; 85:1248-1253.
39. Stewart WJ, Currie PJ, Salcedo EE, et al. Evaluation of mitral leaflet motion by echocardiography and jet direction by Doppler color flow mapping to determine the mechanism of mitral regurgitation. *J Am Coll Cardiol* 1992; 20:1353-1361.
40. Freeman WK, Schaff HV, Khandheria BK, et al. Intraoperative evaluation of mitral valve regurgitation and repair by transesophageal echocardiography: incidence and significance of systolic anterior motion. *J Am Coll Cardiol* 1992; 20:599-609.
41. Sheikh KH, DeBruijn NP, Rankin JS, et al. the utility of transesophageal echocardiography and Doppler color flow imaging in patients undergoing cardiac valve surgery. *J Am Coll Cardiol* 1990; 15:363-372.
42. Maurer G, Czer LSC, Chaux A, et al. Intraoperative Doppler color flow mapping for assessment of valve repair for mitral regurgitation. *Am J Cardiol* 1987; 60:333-337.
43. Panidis I, Ross J, Mintz GS. Normal and abnormal prosthetic valve function as assessed by Doppler echocardiography. *J Am Coll Cardiol* 1986; 8:317-326.
44. Sagar KB, Wann LS, Paulsen WHJ, Romhilt DW. Doppler echocardiographic evaluation of Hancock and Bjork-Shiley prosthetic valves. *J Am Coll Cardiol* 1986; 7:681-687.
45. Khandheria BK, Seward JB, Oh JK, Freeman WK, Nichols BA, Sinak LJ, Miller FA, Tajik AJ. Value and limitations of transesophageal echocardiography in assessment of mitral valve prostheses. *Circulation* 1991; 83:1956-1968.

46. Daniel WG, Mugge A, Grote J, et al. Comparison of transthoracic and transesophageal echocardiography for detection of abnormalities of prosthetic and bioprosthetic valves in the mitral and aortic positions. *Am J Cardiol* 1993; 71:210-215.
47. Cerebral Embolism Task Force. Cardiogenic brain embolism: the second report of the Cerebral Embolism Task Force. *Arch Neurol* 1989; 46:727-743.
48. Bogousslavsky J, Hachinski VC, Boughner DR, Fox AJ, Vinuela F, Barnett HJ. Clinical predictors of cardiac and arterial lesions in carotid transient ischemic attacks. *Arch Neurol* 1986; 43:229-233.
49. Lovett JL, Sandok BA, Giuliani ER, Nasser FN. Two dimensional echocardiography in patients with focal cerebral ischemia. *Ann Intern Med* 1981; 95:1-4.
50. Greenland P, Knopman DS, Mikell FL, Asinger RW, Anderson DC, Good DC. Echocardiography in diagnostic assessment of stroke. *Ann Intern Med* 1981; 95:51-53.
51. Pop G, Sutherland GR, Koudstaal PJ, Sit TW, de Jong G, Roelandt JR. Transesophageal echocardiography in the detection of intracardiac embolic sources in patients with transient ischemic attacks. *Stroke* 1990; 21:560-565.
52. Hofmann T, Kasper W, Meinertz T, Geibel A, Just H. Echocardiographic evaluation of patients with clinically suspected arterial emboli. *Lancet* 1990; 336:1421-1424.
53. Pearson AC, Labovitz AJ, Tatineni S, Gomez CR. Superiority of transesophageal echocardiography in detecting cardiac source of embolism in patients with cerebral ischemia of uncertain etiology. *J Am Coll Cardiol* 1991; 17:66-72.
54. Cujec B, Polasek P, Voll C, Shuaib A. Transesophageal echocardiography in the detection of potential cardiac source of embolism in stroke patients. *Stroke* 1991; 22:727-733.
55. DeRook F, Comess K, Albers G, et al. Prevalence of transesophageal echocardiographic findings in stroke patients with and without carotid stenosis. *J Am Soc Echo* 1991; 4:293 (abstract).
56. Lee RJ, Bartzokis T, Yeoh T-K, Grogan HR, Choi D, Schnittger I. Enhanced detection of intracardiac sources of cerebral emboli by transesophageal echocardiography. *Stroke* 1991; 22:734-739.

57. Castello R, Pearson AC, Labovitz AJ. Prevalence and clinical implications of atrial spontaneous contrast in patients undergoing transesophageal echocardiography. *Am J Cardiol* 1990; 65:1149-1153.
58. Black IW, Hopkins AP, Lee LCL, Walsh WF. Left atrial spontaneous echo contrast: a clinical and echocardiographic analysis. *J Am Coll Cardiol* 1991; 18:398-404.
59. DeRook FA, Comess KA, Albers GW, Popp RL. Transesophageal echocardiography in the evaluation of stroke. *Ann Intern Med* 1992; 117:922-932.
60. Belkin RN, Hurwitz BJ, Kisslo J. Atrial septal aneurysm: association with cerebrovascular and peripheral embolic events. *Stroke* 1987; 18:856-862.
61. Hanley PC, Tajik AJ, Hynes JK, et al. Diagnosis and classification of atrial septal aneurysm by two-dimensional echocardiography: report of 80 consecutive cases. *J Am Coll Cardiol* 1985; 6:1370-1382.
62. Falk R. PFO or UFO? The role of a patent foramen ovale in cryptogenic stroke. *Am Heart J* 1991; 121:1264-1266.
63. Karalis DG, Chandrasekaran K, Victor MF, Ross JJ, Mintz GS. Recognition and embolic potential of intraaortic atherosclerotic debris. *J Am Coll Cardiol* 1991; 17:73-78.
64. Tunick PA, Culliford AT, Lamparello PJ, Kronzon I. Atheromatosis of the aortic arch as an occult source of multiple systemic emboli. *Ann Intern Med* 1991; 114:391-392.
65. Tunick PA, Perez JL, Kronzon I. Protruding atheromas in the thoracic aorta and systemic embolization. *Ann Intern Med* 1991; 115:423-427.
66. Brickner ME, Friedman DB, Unwin H, Lacker M, Abraham M, Grayburn PA. Value of transesophageal echocardiography in the evaluation of patients with stroke or transient ischemic attack: a prospective study. *Circulation* 1992 (Suppl I); 86:I-398 (abstract).
67. Pollick C, Sullivan H, Cujec B, Wilansky S. Doppler color-flow imaging assessment of shunt size in atrial septal defect. *Circulation* 1988; 78:522-528.
68. Patel AM, Miller FA, Khandheria BK, et al. Role of transesophageal echocardiography in the diagnosis of papillary muscle rupture secondary to myocardial infarction. *Am Heart J* 1989; 118:1330-1332.

69. Smith MD, MacPhail B, Harrison MR, Lenhoff SJ, DeMaria AN. Value and limitations of transesophageal echocardiography in determination of left ventricular volumes and ejection fraction. *J Am Coll Cardiol* 1992; 19:1213-1222.
70. Sheikh KH, DeBruijn NP, Rankin JS, et al. The utility of transesophageal echocardiography and Doppler color flow imaging in patients undergoing cardiac valve surgery. *J Am Coll Cardiol* 1990; 15:363-372.
71. Gewertz BL, Kremser PC, Zarins CK, et al. Transesophageal echocardiographic monitoring of myocardial ischemia during vascular surgery. *J Vasc Surg* 1987; 5:607-613.
72. Roizen MF, Beaupre PN, Alpert RA, et al. Monitoring with two-dimensional transesophageal echocardiography. *J Vasc Surg* 1984; 1:300-305.
73. Eisenberg MJ, London MJ, Leung JM, et al. Monitoring for myocardial ischemia during noncardiac surgery: a technology assessment of transesophageal echocardiography and 12-lead electrocardiography. *JAMA* 199; 268:210-216.
74. Manning WJ, Silverman DI, Gordon SPF, Krumholz HM, Douglas PS. Cardioversion from atrial fibrillation without prolonged anticoagulation with use of transesophageal echocardiography to exclude the presence of atrial thrombi. *N Engl J Med* 1993; 328:750-755.
75. Arnold AZ, Mick MJ, Mazurek RP, Loop FD, Trohman RG. Role of prophylactic anticoagulation for direct current cardioversion in patients with atrial fibrillation or atrial flutter. *J Am Coll Cardiol* 1992; 19:851-855.