David C. Warltier, M.D., Ph.D., Editor

Anesthesiology 2007; 107:333–46

Transesophageal Echocardiography and Cardiovascular Sources of Embolism

Implications for Perioperative Management

Christine Ngukhoon Tan, M.B., B.S., F.R.C.A., Alan G. Fraser, F.R.C.P., F.E.S.C.†*

Transesophageal echocardiography has become an invaluable investigation in patients with cardioembolic events because of its high sensitivity and specificity for defining detailed structure and function of the cardiovascular system. Patients who receive anesthesia and critical care may be at risk of systemic embolism from various cardiovascular sources. The main factors associated with embolism include intracardiac lesions such as thrombi, vegetations, and tumors; cardiac anomalies; and vascular disease, *e.g.*, aortic atheroma. In this review article, the authors describe how transesophageal echocardiography may be used to identify various cardiovascular sources of embolism, provide risk stratification, influence medical therapy, and refine clinical decision making in patients receiving critical care and anesthesia. With these improvements, it is hoped that better patient outcomes may be achieved in the perioperative period.

PATIENTS who receive anesthesia and critical care are at risk of cardioembolic stroke, which is an uncommon but highly important cause of perioperative mortality and morbidity.[1,2] Embolism is associated with intracardiac lesions, cardiac anomalies, and vascular disease[3] (table 1), all of which may be assessed by transesophageal echocardiography (TEE).[4,5]

In this review, we describe how cardiovascular sources of embolism may be detected by TEE, and how TEE is important for stratifying risk and providing prognostic information to prevent, predict, or manage perioperative embolism.

This article is featured in "This Month in Anesthesiology." Please see this issue of ANESTHESIOLOGY, page 5A.

Additional material related to this article can be found on the ANESTHESIOLOGY Web site. Go to http://www.anesthesiology.org, click on Enhancements Index, and then scroll down to find the appropriate article and link. Supplementary material can also be accessed on the Web by clicking on the "ArticlePlus" link either in the Table of Contents or at the top of the Abstract or HTML version of the article.

Basics of TEE

Detection of Sources of Embolism

Transesophageal echocardiography is used increasingly in the perioperative period,[6] and it is considered to be superior to transthoracic echocardiography for detecting cardiovascular sources of embolism.[7] It has high sensitivity and specificity for defining posterior and inferior structures in the heart because of their proximity to the esophagus and stomach. High-frequency transduction (4–8 MHz) allows superior image resolution, and so TEE is better than transthoracic echocardiography at evaluating thrombi,[8,9] vegetations,[10] and other masses.[11] There are 20 cross-sectional views for comprehensive intraoperative TEE examination, as recommended by the American Society of Echocardiography and Society of Cardiovascular Anesthesiologists.[12] Of them, key TEE views for detecting cardiovascular sources of embolism can be obtained from the upper esophageal and midesophageal positions (fig. 1).

Limitations, Complications, and Contraindications

Masses located in the left ventricular (LV) apex, distal ascending aorta, and proximal aortic arch may be missed by TEE. Image acquisition is limited in these areas, and other imaging modalities should be considered, *e.g.*, helical computed tomography and magnetic resonance imaging.[13] In addition, TEE has caused complications that are primarily associated with esophageal intubation. These include trauma to the oropharynx, esophagus, and stomach resulting in odynophagia, esophageal perforation, and upper gastrointestinal bleeding.[14] Therefore, the main contraindications of TEE are oropharyngeal pathology, esophageal stricture, varices, and recent upper gastrointestinal bleeding.[15]

* Consultant Anesthesiologist: Department of Anaesthesia and Intensive Care Medicine, University Hospital of Wales. † Honorary Consultant in Cardiology, Wales Heart Research Institute, Cardiff University, Cardiff, United Kingdom.

Received from the Department of Anaesthesia and Intensive Care Medicine and the Wales Heart Research Institute, University Hospital of Wales, Cardiff, United Kingdom. Submitted for publication September 25, 2006. Accepted for publication April 2, 2007. Support was provided solely from institutional and/or departmental sources.

Address correspondence to Dr. Tan: Department of Anaesthesia and Intensive Care Medicine, University Hospital of Wales, Cardiff, CF14 4XN, United Kingdom. ngukhoon@yahoo.co.uk.

Table 1. Cardiovascular Sources of Embolism

Factors Associated with Embolism	Examples
Intracardiac lesions	Thrombi
	Vegetations
	Tumors
Cardiac anomalies	Patent foramen ovale
	Atrial septal defect
	Atrial septal aneurysm
	Chiari network
Vascular	Aortic atheroma
Others	Intracardiac air
	Mitral annular calcification
	Valve strands

Intracardiac Lesions

Intracardiac sources of embolism include thrombi, vegetations, and tumors. They may be distinguished by their echocardiographic features and associated risk factors (table 2). The risk of embolism and examples of TEE use in these conditions are also summarized.

Left Atrial Thrombus

Thrombi in the left atrium (LA) appear as intracavitary masses that are usually distinct from surrounding structures. Often, they reside between the trabeculae of the left atrial appendage (LAA)[16] (fig. 2), which is a multilobed end-pouch, transversed by pectinate muscles with a ridge-like appearance. (This TEE image is available on the ANESTHESIOLOGY Web site at http://www.anesthesiology.org.) Sometimes, thrombi are echo-lucent and difficult to recognize. In this situation, the diagnosis may be confirmed by giving an intravenous echo-contrast agent to improve discrimination between blood and intracavitary masses.[17,18]

The LAA is assessed at the mid-esophageal level. This technique involves rotation of the TEE transducer between 0° and 150° along the LAA axis, shown as dotted line along the middle of the scan views (figs. 1A–C). The sensitivity and specificity of detecting thrombi in the LA and LAA are between 81% and 98%, and between 98% and 100%, respectively.[19]

The presence of thrombus in the LA is highly predictive of transient ischemic attack in patients with atrial fibrillation (AF). In a prospective study of 261 patients, the odds ratio (OR) (95% confidence interval [CI]) for transient ischemic attack in patients with LA thrombus compared with those without was 7.7 (2.1–21.6), and the annual rate of transient ischemic attack was 9.2% compared with 1.9%.[20] Embolic risk would seem to correlate with mobility and shape; in a prospective study of 41 patients with LA thrombi, the incidences of embolism in patients with mobile ball-like thrombi, fixed ball thrombi, and "mountain"-type thrombi were 77, 18, and 9%, respectively.[21]

Predisposing factors for LA thrombus include mitral valve pathology and abnormal LA contractile function. Structural or anatomical risk factors include an enlarged[22] and bifid LAA.[23] In a multicenter, observational follow-up study of 409 patients, the relative risks (95% CIs) of stroke or embolism in patients with increased length and width of LAA were 1.6 (1.05–2.5) and 2.4 (1.2–4.8), respectively.[24]

Mitral Valve Disease. Left atrial thrombus has been reported in as many as 29–33% of patients with rheumatic mitral stenosis and AF[25,26] in whom it was associated with a high incidence (41%) of symptomatic arterial embolism including stroke.[26] Thrombi in the LA are less common in patients with mitral regurgita-

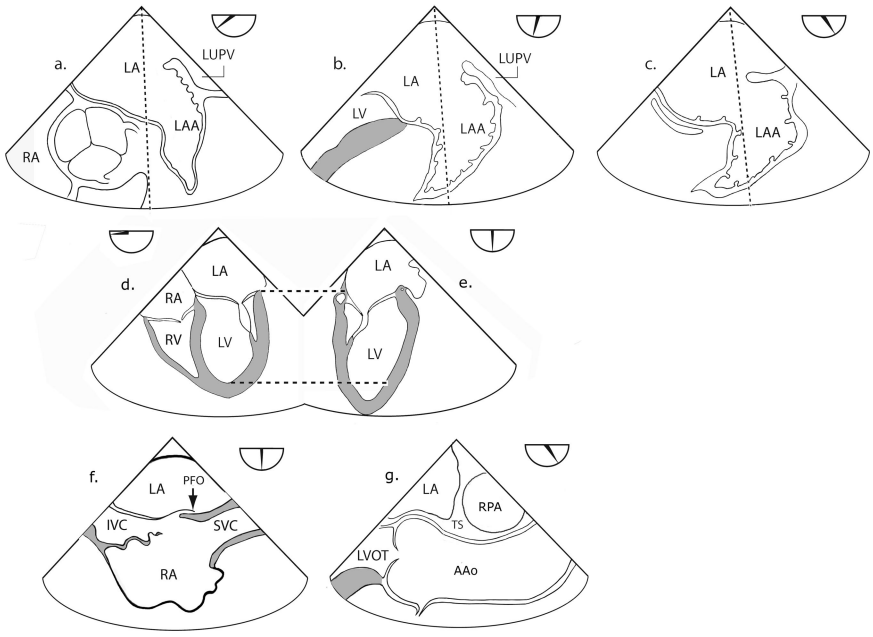

Fig. 1. Essential views for detection of cardiovascular sources of embolism. (*A*) Mid-esophageal view of the left atrium (LA) and left atrial appendage (LAA) at 20°. (*B*) Mid-esophageal view of the LA and LAA at 70°. (*C*) Mid-esophageal view of the LA and LAA at 120°. (*D*) Mid-esophageal four-chamber view (0°) of the foreshortened left ventricle (LV). (*E*) Mid-esophageal two chamber view (90°) of the LV which is not foreshortened. Apex is shown. (*F*) Mid-esophageal bicaval view (90°) showing a patent foramen ovale (PFO). (*G*) Upper esophageal long axis view (110°) of the mid–ascending aorta (AAo) imaged through the right pulmonary artery (RPA). AA = ascending aorta; IVC = inferior vena cava; LUPV = left upper pulmonary vein; LVOT = left ventricular outflow tract; RA = right atrium; RV = right ventricle; SVC = superior vena cava; TS = transverse sinus. (Multiplane angles may vary according to patient's anatomy.)

Table 2. Intracardiac Sources of Embolism and Their Associated Risk Factors, Echocardiographic Features, Embolic Risk, Differential Diagnosis, and Utility of TEE

Intracardiac Sources	Risk Factors	Associated Echo Findings	Marker of Embolic Risk	Differential Diagnosis	Examples of Utility of TEE
Thrombi					
LA thrombus	Enlarged LAA	SEC	Mobile	Pectinate muscle	1. Confirm thrombus
	Enlarged LA	Low LAA flow velocity	Ball-like	Tumor, *e.g.*, myxoma	2. Indicate timing of cardioversion
	Mitral stenosis			Vegetation	3. Optimize anticoagulation
	Prosthetic MV				4. Guide BMV or PLAATO
	AF				5. Target surgical removal
	Atrial septal aneurysm				6. Suggest amputation and ensure complete exclusion of LAA
LV thrombus	Poor LV function e.g. Infarction,	Low EF	Mobile	Prominent trabeculae	1. Direct LV thrombectomy
	Dilatation	Apical akinesis	Protruding	Tumor	2. Assist insertion or explantation of LVAD
	Apical aneurysm				
	Noncompaction		Nonlaminated		
Thrombus in RH	DVT	Pulmonary embolus	Mobile	Chiari remnants	1. Detect pulmonary embolism and paradoxical embolism
	Foreign bodies		Elongated	Vegetation	
	e.g. Vascular catheter	Paradoxical embolism if PFO or ASD present			2. Guide CVP placement and removal
	Pacing electrodes			Tumor	
	TV prosthesis				
	ARVC				
Vegetations	Valve disease	Perivalvular	Large, > 10 mm	Valve strands	1. Guide surgical repair and replacement
	Intravenous drug user	Abscess, Aneurysm or Fistula	Mobile	Nodules of Arantius	2. Assess valvular function and monitor hemodynamic instability
	Foreign bodies	Perforation		NBTE	
	Prosthetic valve	Rupture chordae			
	Vascular catheter	Dehiscence		Tumor, *e.g.*, fibroelastoma	
	Pacing electrodes				
Tumors					
Myxoma	Family history	Attached to atrial septum	Mobile	Vascularized thrombus	1. Confirm location, attachment, extension and migration
	Previous history	Heterogenous mass	Villous	Thrombus within atrial septal aneurysm	2. Guide surgical approach, manipulation and cannulation for CPB
			Uncapsulated	Lipomatous hypertrophy	
			Multiple	Secondary tumor	3. Alter surgical plan in acute tumor embolization
Fibroelastoma	Previous surgery	Attached to valve endothelium	Mobile	Thrombus	4. Assess valvular function
	Valvular trauma	Homogenous mass		Vegetation	5. Ensure adequate excision and exclude tumor residues
				Secondary tumor	

AF = atrial fibrillation; ARVC = arrhythmogenic right ventricular cardiomyopathy; ASD = atrial septal defect; BMV = balloon mitral valvotomy; CPB = cardiopulmonary bypass; CVP = central venous pressure catheter; DVT = deep vein thrombosis; EF = ejection fraction; LA = left atrium; LAA = left atrial appendage; LV = left ventricle; LVAD = left ventricular assist device; MV = mitral valve; NBTE = nonbacterial thrombotic endocarditis; PFO = patent foramen ovale; PLAATO = percutaneous LAA transcatheter occlusion; RH = right heart; SEC = spontaneous echo contrast; TEE = transesophageal echocardiography; TV = tricuspid valve.

tion[27] presumably due to attenuation of stasis by the regurgitant jet. In patients with a newly implanted mitral valve replacement, asymptomatic, early thrombosis was detected in 9.4% of 680 consecutive patients undergoing TEE.[28]

Abnormal Atrial Function. Severe dysfunction of the LA and its appendage correlates with increased risk of recurrent stroke and mortality.[29] Echocardiographic features of impaired LA function include spontaneous echo contrast (SEC), reduced velocities of flow in the LAA, and atrial dysrhythmias.

- SEC appears as smoke-like, swirling echoes resulting from the aggregation of erythrocytes and fibrinogen at low shear rates.[30] It is more likely to be detected using a high-frequency ultrasonic transducer (> 5 MHz, as used in TEE) and high gain settings. The degree of SEC can be quantified using integrated backscatter, which records the acoustic intensity of the signal in decibels.[31,32] SEC is relatively common (*e.g.*, 17% of 290 consecutive patients undergoing TEE) and is associated with AF, mitral stenosis, and mitral prostheses.[22] In patients with mitral stenosis and chronic AF, it

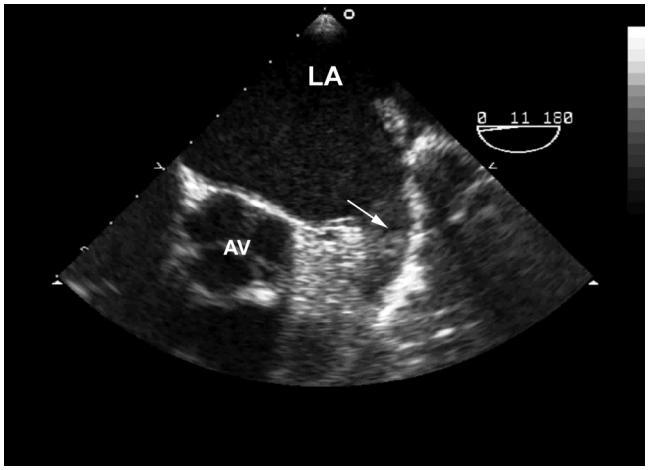

Fig. 2. Mid-esophageal short axis view of the aortic valve (AV) and left atrium (LA) showing thrombus (*arrow*) in the left atrial appendage.

predicts LA thrombus and embolic events (OR, 1.74 [1.23–2.48]).[26] In patients with severe SEC and AF, it is associated with risk of cerebral embolism.[33]

- The velocity of blood flow at the orifice of the LAA can be sampled using pulse-wave Doppler, with a low Nyquist limit and low wall-filter settings. Thrombogenic risk increases with decreasing LAA velocity; *e.g.*, in a prospective study of 500 patients with stroke, thrombus was found in 1, 10, and 29% of patients with LAA velocities greater than 40 cm/s, 20–40 cm/s, and less than 20 cm/s, respectively.[34] Above a threshold LAA velocity greater than 55 cm/s, thrombus was ruled out because this velocity has a negative predictive value of 100%.
- AF[35] and atrial flutter[36] are associated with abnormal LA function, stasis, and thrombosis.[37] The prevalence of intraatrial thrombus has been reported at 29–40% in patients with atrial flutter or AF compared with 3–5% in controls in sinus rhythm.[36,38] Despite restoration to sinus rhythm, whether spontaneously, by electrical conversion,[39] or by radiofrequency ablation,[40] patients remain at risk of LA thrombosis for up to 4–6 weeks, due to transient mechanical dysfunction of the LA called *atrial stunning*.[41]

Utility of TEE. Detection of LA thrombi by TEE has influenced clinical decision making. Examples include the following:

- TEE-guided direct current cardioversion. TEE has been performed during general anesthesia before cardioversion, to exclude the presence of thrombus in the LA.[42] This practice also involves routine anticoagulation because of the possibility of LA stunning, delayed thrombus formation, and thromboembolism.[43] Cardioversion is contraindicated in the presence of thrombus, and a follow-up TEE can guide anticoagulation and ensure its resolution.[44]
- TEE and interventional cardiology. In patients with mitral stenosis, TEE has been used to guide percutaneous transvenous mitral commissurotomy[45] and transcatheter occlusion of the LAA.[46]
- TEE in cardiac surgery. Patients with LA thrombus of high embolic potential may be scheduled to undergo open cardiac surgery. TEE should be used to reconfirm its presence. For example, in a case report, complete embolization of LA thrombus to the leg of a patient was described, and TEE showed that sternotomy was no longer required.[47] Conversely, TEE has been shown to refine intraoperative decision making, *e.g.*, removal of thrombi and closure or amputation of the LAA.[48] It is essential to verify complete surgical obliteration because residual flow within the LAA predisposes to further thromboembolism.[49]

Left Ventricular Thrombus

Left ventricular thrombi may be laminar or protruding, with a smooth or irregular shape. They are usually contiguous with areas of noncontracting myocardium. Recent or actively forming thrombi may appear echo-lucent. Patients with subacute, protruding, echo-lucent, and mobile thrombi are at high risk of embolic events, compared with those who have sessile, laminated, and organized thrombi.[50]

Because thrombi are often located in the LV apex, they should be suspected when an akinetic cardiac apex is thickened and rounded. A foreshortened image of the LV apex (fig. 1D) can be minimized by retroflexing the tip of the TEE probe or using other vertical scan views (fig. 1E). A deep transgastric view (0°–20°) may provide better resolution because it is within the near field of the transducer. To distinguish thrombi from artifacts, it is necessary to acquire optimal images in at least two different views, throughout the cardiac cycle. Some laminated thrombi may be difficult to visualize clearly, and endocardial border delineation may be improved by a higher transducer frequency ($>$ 5 MHz) and contrast ventriculography.[51] Harmonic power Doppler contrast has been used to detect thrombi in the apex or in a pseudoaneurysm.[52]

Thrombi are more common at the apex, in aneurysms[53] or pseudoaneurysms,[54] or between trabeculations and in deep recesses. An aneurysm is a dilated region of infarcted LV,[55] and a pseudoaneurysm is an area of contained rupture lined by pericardium.[54,56] Trabeculations and deep recesses in continuity with the LV cavity are features of noncompaction, a disorder of endomyocardial embryogenesis[57,58] in which intertrabecular flow can be demonstrated with color-flow Doppler and contrast-enhanced echocardiography.[59,60] In isolated LV noncompaction, thromboembolic events occurred in 24% of 34 patients.[61] A recent case report of an acute thromboembolic occlusion of the superior mesenteric artery has been described in association with isolated LV noncompaction.[62]

Patients with poor LV function are predisposed to stasis and thrombosis. After anterior myocardial infarction with severe regional wall motion abnormalities, the overall incidence of LV thrombus was 25%.[63] LV thrombus was more common in patients with larger ventricles. It occurred in 72% of 53 patients with an LV end-systolic volume index greater than 35 ml/m^2 compared with 33% in those with smaller ventricles.[64] In severe ventricular failure, LV thrombus has been reported in patients with ventricular assist devices.[65] Conversely, inferior myocardial infarction, coronary reperfusion, and preserved global LV systolic function are associated with a lower incidence of LV thrombus.[63]

Echocardiographic indices that correlate with LV thrombosis include low ejection fraction, high LV wall motion score, and high E/Em ratio. An ejection fraction less than 40% was associated with earlier LV thrombus formation, in the first week after myocardial infarction.[64] After acute anterior myocardial infarction, the mean (SD) wall motion score of 9.2 (2.8) in patients with LV thrombus was higher than that of 4.7 (2.1) in patients with no thrombus.[63] Thrombus persisting 6 weeks after myocardial infarction was associated with apical dyskinesis.[64] A high E/Em ratio (peak mitral inflow velocity [E] to peak mitral annular velocity by tissue Doppler [Em]) implies a high LV filling pressure. In 87 consecutive patients with acute myocardial infarction, the E/Em ratio (SD) was 12(5) in the group with LV thrombus compared with 7.2 (2.8) in those without thrombus. The sensitivity and specificity of E/Em greater than 9 for predicting LV thrombosis after myocardial infarction were 69% and 79%, and the positive and negative predictive values were 63% and 84%.[66]

Utility of TEE. Thrombus in the LV is associated with poor ventricular function. Patients with this condition may require coronary revascularization and insertion of a ventricular assist device:

- During coronary revascularization, TEE should be used to check for LV thrombi particularly in ventricles that are aneurysmal.[67]
- During insertion and separation from ventricular assistance, TEE is recommended to assess ventricular function,[68] in addition to examining for thrombus,[65] air, valve dysfunction,[69] and septal defects.[68]

Thrombus in the Right Heart

Right heart thrombi are most often caused by embolization from a peripheral venous source.[70] They may become entrapped in the tricuspid valve apparatus or right ventricular (RV) trabeculations. *In situ* thrombosis in the right heart is usually iatrogenic. Foreign bodies such as indwelling vascular catheters,[71] pacemaker leads,[72] and a prosthetic tricuspid valve[73,74] are predisposing factors. Thrombi in the right heart may become infected[75,76] or cause pulmonary embolism.[77,78] Unlike the LV, RV dilatation and systolic dysfunction are rare causes of thrombosis, which can occur at the site of RV infarction, or within the hypokinetic aneurysmal areas of the RV free wall in arrhythmogenic RV cardiomyopathy.[79,80]

Thrombi in the right atrium (RA) may be visualized in the mid-esophageal four-chamber or bicaval views between 0° and 90° on multiplane TEE. They should be distinguished from a Eustachian valve or a Chiari network, which are remnants of the right sinus venosus adjacent to the inferior vena cava (IVC). The former is commonly seen as a thin, mobile, linear structure, whereas a Chiari network is a large and fenestrated structure extending across the RA with additional attachments to its upper wall and the atrial septum.[81] Unlike thrombi, these structures do not cross the tricuspid valve during diastole. In the RV, thrombus may be seen in mid-esophageal four-chamber view and in the transgastric RV inflow view between 80° and 110°.

Utility of TEE. Pulmonary embolism may occur as a result of thrombi in the right heart.[77,78] These patients may present for pulmonary embolectomy and TEE may be used to assist in their diagnosis. In a study of 46 patients having pulmonary embolectomy, TEE demonstrated RV dysfunction, tricuspid regurgitation, and leftward atrial septal bowing. However, direct visualization of the emboli was not always possible. The sensitivities for detection were 0.35, 0.26, and 0.17 in the right, main, and left pulmonary arteries, respectively. Corresponding respective specificities were 0.89, 0.95, and 1.0.[82] In another study, extrapulmonary locations of thromboemboli within the right heart were detected in 26% of 50 patients who had pulmonary embolectomy. Surgical management was altered accordingly.[83]

In addition, TEE would seem to be recommended when long-term central venous catheters are removed. It is thought that TEE can monitor the procedure and provide early evidence of possible thrombus migration to the pulmonary artery.[84,85]

Similarly, it is likely that TEE would be useful in guiding placement of central venous catheter in the presence of right heart thrombus. Central venous catheter placement in the distribution of the IVC may be preferred in the presence of a superior vena cava thrombus. The converse would be appropriate if there were an IVC thrombus.

Vegetations

Vegetations are of low echo-reflectance compared with normal valve leaflets. They are independently mobile, lobulated structures that are most often identified downstream from the origin of a regurgitant jet. According to a meta-analysis of eight studies,[10] they are detected readily by TEE, which has a sensitivity of 87–100% compared with 30–63% by transthoracic echocardiography. TEE is important for determining the Duke criteria

for the clinical diagnosis of infective endocarditis,[86] and a normal TEE study has high negative predictive value. The diagnosis of prosthetic valve endocarditis is more difficult because of acoustic shadowing and reverberation artifacts.[9] In addition, vegetations should not be confused with other lesions, *e.g.*, nonbacterial thrombotic endocarditis, valve strands, or even nodules of Arantius, which are normal anatomical features of the aortic valves.

The mobility, size, and location of vegetations are important predictors of embolism. Mobile vegetations larger than 10 mm and in the mitral position[87] are associated with a high incidence of embolism.[88,89] In a prospective cohort study comparing patients with vegetations larger than 10 mm to those with smaller lesions, the relative risk (95% CI) of embolization was 2.64 (0.98–7.16).[90] Neurovascular embolization is associated with a high mortality.[91]

Utility of TEE. In the critical care setting, TEE is essential when the differential diagnosis in a patient with severe sepsis or multiple systemic embolism includes infective endocarditis.[92] The high risk of embolism in patients with large vegetations (> 10 mm), particularly in the first 2 weeks of antibiotic therapy, may be minimized by early surgery.[90] TEE should be used to plan surgery and anesthetic management.

Surgery depends on

- The site of endocarditis
- Multivalvular[93] or prosthetic valve involvement
- The extent of perivalvular infection, *e.g.*, aneurysm, fistula, chordal rupture, leaflet perforation, and abscess formation[94,95]

Patients with infective endocarditis have significant hemodynamic instability, and their anesthetic management is influenced by

- The degree of ventricular dysfunction
- The predominant pathophysiology of valvular dysfunction, *e.g.*, regurgitation or stenosis
- The magnitude of systemic sepsis[96]

Intracardiac Tumors

Cardiac tumors may be additional sources of embolism in patients receiving anesthesia and critical care. Primary tumors are rare, with an incidence ranging from 0.001% to 0.3% in unselected patients at autopsy.[11] The majority (75%), including myxoma and fibroelastoma, are benign. Malignant primary neoplasms include angiosarcoma, rhabdomyosarcoma, fibrosarcoma, leiomyosarcoma, and intracardiac lymphoma.[11] Secondary neoplasms with intracardiac extension and metastasis are more common[97] and can result in stroke and widespread systemic embolism. Examples include lung[98,99] and renal cell carcinoma,[100] which may extend into the LA *via* the pulmonary veins, and into the RA *via* the IVC, respectively.

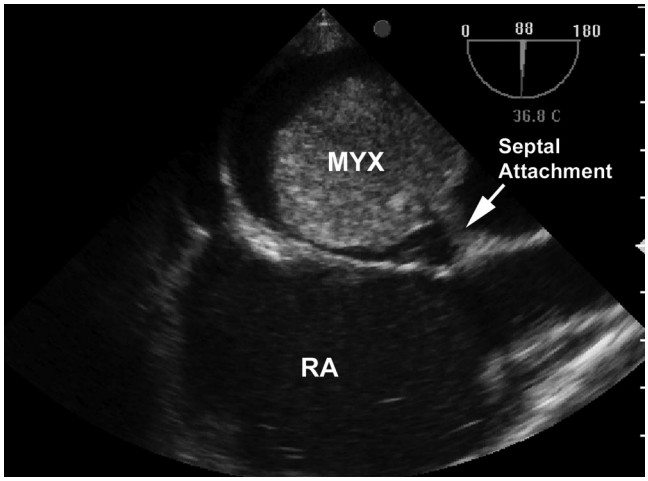

Fig. 3. Mid-esophageal bicaval view showing a left atrial myxoma (MYX) attached to the atrial septum (*arrow*). RA = right atrium.

Many intracardiac structures may mimic cardiac tumors. Vascularized thrombi,[101] giant atrial septal aneurysms[102] with thrombus,[103] or coronary artery aneurysms[104] could act as embolic sources. Other structures such as a prominent crista terminalis[105] or lipomatous hypertrophy of the atrial septum[106] are normal or incidental findings. Lipomatous hypertrophy has been reported in association with supraventricular arrhythmias[107] and probable embolic phenomena, but a causal relation has not been established. Expert echocardiography is important to obviate false-positive identification and hence unnecessary surgery.[108]

Cardiac Myxomas. Cardiac myxomas account for 30–50% of primary cardiac tumors.[109] They can be globular or irregular in shape (like a cluster of grapes) with a heterogeneous texture incorporating calcified, gelatinous, or cystic areas. Eighty percent of myxomas are located in the LA, where their most frequent attachment is to the fossa ovalis of the atrial septum (fig. 3; this TEE image is available on the ANESTHESIOLOGY Web site at http://www.anesthesiology.org). Other less typical attachments include the LAA and chordae of the mitral valve.[110] Myxomas may be found concurrently in the LV, RV, or RA.[109] Systemic embolism is thought to occur from fragmentation of tumor or dislodgement of thrombi formed on its surface.[111] Highly mobile, villous tumors with friable broad bases have much higher embolic potential than the firm, well-encapsulated variety.[112] Uncommonly, detachment and embolism of a whole cardiac myxoma may occur.[113]

Cardiac Papillary Fibroelastomas. Cardiac papillary fibroelastomas (or myxopapillary tumors) represent 8–10% of primary tumors of the heart. They occur most commonly on left heart valves, attached by a small pedicle to their endocardial surface, but they may occur at multiple other sites, including the papillary muscles, the LV free wall, the RV outflow tract,[114] and the pulmonary

valve.[115] They are usually small (< 20 mm), homogeneous, round or oval-shaped masses but may also have multiple papillary fronds appearing like a sea anemone in motion. They may mimic vegetations in their independent motion, but unlike vegetations, they are often located on the ventricular side of the mitral valve or the ascending aortic side of the aortic valve.

Presenting features of fibroelastomas include systemic embolism,[116] transient ischemic attack, stroke, blindness, syncope, myocardial infarction causing heart failure, and sudden death.[117] Fibroelastomas may undergo fragmentation or act as a nidus for platelet and fibrin aggregation, leading to thromboembolism.[118] In a summary of 725 cases reported in the literature, tumor mobility was the only independent predictor of death or nonfatal embolization.[119] Thus surgical excision has been recommended if a tumor becomes mobile during follow-up by echocardiography, even when there are no symptoms.

Utility of TEE. In the perioperative period, TEE may be used to

- Confirm location and guide manipulation.[120] Tumors may embolize, and TEE should be used to check their precise location and assist in appropriate manipulation of the heart. Tumors that embolize may cause significant hemodynamic instability requiring immediate confirmation by TEE and alteration in surgical plan.[121]
- Assist the surgeon during cannulation and cardiopulmonary bypass (CPB). Tumors that are detected in the superior vena cava or IVC may require alternative routes of cannulation.[122] Complete resection of tumors may require modification of CPB to include deep hypothermic circulatory arrest.[123]
- Assess valve function. Valvular involvement and dysfunction may occur and should be assessed perioperatively, by TEE.[124]
- Assess adequacy of tumor resection. TEE is used to check for multiple tumors[125] before surgery and to ensure complete resection.[126]

Cardiac Anomalies

Cardiac anomalies associated with systemic embolism include patent foramen ovale (PFO), atrial septal defects, and atrial septal aneurysm.[127] Anatomical variants such as Chiari remnants may also be associated with thromboembolism.[128]

Patent Foramen Ovale

Patent foramen ovale is an interatrial communication with a one-way flap that allows right-to-left shunting of blood and hence thrombi. It is a remnant of the fetal circulation and occurs when the septum primum and septum secundum fail to fuse.[129] At autopsy, the incidence of "probe-patent" PFO may be as high as 27–30%, but only 6% are wider than 5 mm.[130] PFO is more common in patients with a mobile atrial septal flap.[131]

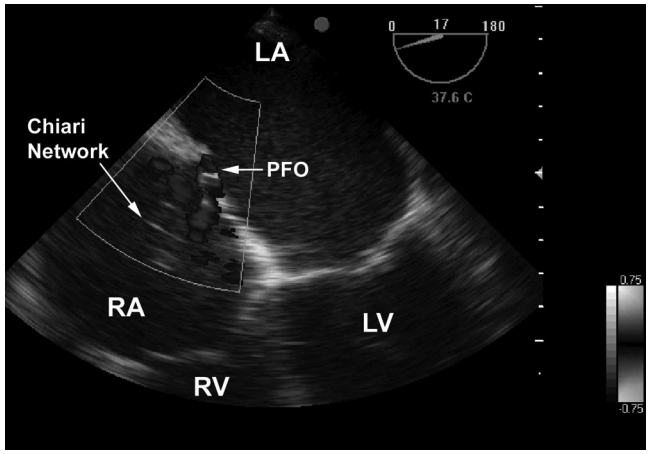

Fig. 4. Mid-esophageal four-chamber view of the heart showing a patent foramen ovale (PFO) and Chiari network (*long arrow*). The velocity of blood flow across the PFO can be demonstrated by color flow Doppler (see *Anesthesiology* website). LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

On TEE, PFO is often visualized in the mid-esophageal four-chamber (fig. 4; this TEE image is available on the ANESTHESIOLOGY Web site at http://www.anesthesiology.org) and bicaval views (fig. 1F). The diagnosis is usually made when four or more microbubbles are seen entering the LA during the first three cardiac cycles, after opacification of the RA by agitated saline.[132] Sudden release of positive airway pressure during mechanical ventilation similar to the Valsalva maneuver[133] in a conscious patient may aid the right to left shunt. In addition, saline injection into the right femoral vein seems to have a higher diagnostic yield than that into the right antecubital vein.[130] Sometimes, low-velocity flow across the defect in the atrial septum may be seen using color flow mapping at low Nyquist limits.[134]

Patent foramen ovale is associated with an increased risk of paradoxical embolism and cryptogenic stroke, which are reported to be fivefold higher in patients younger than 55 yr (OR in a meta-analysis of case–control studies, 5 [3.2–7.8]).[135] Cryptogenic stroke was associated with increased size and volume of a right-to-left shunt.[136] In addition, paradoxical air embolism has occurred during neurosurgery when patients are in the sitting position.[137] In orthopedic practice, paradoxical embolism of thrombus or fat may occur because of immobility and intramedullary reaming, respectively.[138]

Utility of TEE. In the perioperative period, TEE may be used to diagnose PFO and hence define the cause of paradoxical embolism.[139] Furthermore, unexplained hypoxemia due to right-to-left shunting of blood may be explained by TEE. This situation could occur when there is an increase in pulmonary vascular resistance, *e.g.*, intermittent positive-pressure ventilation, high positive

end-expiratory pressure, chronic lung disease, pneumonectomy, or heart transplantation.[140]

Similarly, a right-to-left shunt may occur during decompression of the left heart, *e.g.*, when a left ventricular assist device is activated. TEE has been recommended to identify the presence of a PFO and to guide surgical closure before left ventricular assist device insertion.[141]

Closure of PFO may also be done routinely by interventional cardiologists. This procedure is performed during general anesthesia and TEE guidance.[142] TEE is used to confirm the diagnosis, guide correct positioning of the occluder device, and ensure that there is no deformation of the aortic root or obstruction of venous return to the RA.[143]

Atrial Septal Aneurysm

Atrial septal aneurysm is a localized saccular deformity that bulges more than 15 mm from the plane of the atrial septum and has a width at its base greater than 10 mm.[144–146] Its prevalence is usually 2–10%[147] but may be as high as 28% on TEE during acute changes in loading conditions at cardiac surgery.[131] Atrial septal aneurysm may be visualized in the mid-esophageal four-chamber or bicaval view. Five subtypes of atrial septal aneurysm have been described.[147] Atrial septal aneurysm frequently occurs with PFO,[144,145] especially in its specific subtypes.[131]

Atrial septal aneurysm is considered a potential source and an independent predictor of cardiogenic embolism. In a prospective multicenter study of 606 patients, the prevalence of atrial septal aneurysm was 27.7% in patients who had ischemic stroke and normal carotid arteries compared with 9.9% in the control group.[144] The combination of atrial septal aneurysm with PFO was associated with an increased risk of recurrent stroke (hazard ratio, 4.17 [1.47–11.84]).[148]

The pathogenesis of thromboembolic phenomena in patients with atrial septal aneurysm is unclear.[149] A possible mechanism is paradoxical embolization of thrombus trapped from IVC inflow or formed within the aneurysm, through its multiple fenestrations.[145]

The occurrence of atrial septal aneurysm may impair the function of ventricular assist devices. In a case report, TEE was used to show that an atrial septal aneurysm impaired blood flow into the right ventricular assist device.[150]

Chiari Network

This structure is a remnant of the right sinus venosus valve that exists during fetal life. Its prevalence of 4.6% in patients undergoing TEE for unexplained arterial embolism exceeds that of 0.5% in patients evaluated for other indications.[151] A Chiari network may be visualized in the RA using the mid-esophageal four-chamber (fig. 4) or bicaval view on multiplane TEE. In contrast to the nonfenestrated flap of the Eustachian valve, a Chiari network is a fenestrated, filamentous structure with a typical undulating appearance during real-time imaging that differentiates it from RA thrombus. It usually extends from the inferolateral part of the RA onto the atrial septum, near the limbus of the fossa ovalis. It should not be mistaken for a fenestrated atrial septum.[152]

A Chiari network can be associated with atrial septal aneurysm and PFO, possibly because it facilitates blood flow from the IVC toward the atrial septum,[153] which may allow persistence of atrial septal aneurysm and prevent spontaneous closure of PFO. Because it may preferentially direct venous thrombi from the IVC toward a PFO, paradoxical embolism is possible. In a case report of a patient undergoing percutaneous closure of an atrial septal defect, entanglement of the closure device with the Chiari network has been demonstrated by TEE perioperatively.[154]

Vascular Causes of Embolism

Aortic Atheroma

Aortic atheroma is an important source of embolism and an independent risk factor for perioperative stroke.[155] In a meta-analysis of six prospective follow-up studies of 1,320 patients with severe aortic arch atheroma, the odds of stroke in patients with aortic arch atheroma was almost four times greater than in controls without atheroma (OR, 3.76 [2.57–5.51]).[156] In patients with embolic disease, the prevalence of atheroma in the aortic arch varies from 21% to 27%.[157] Atheroembolization and, more commonly, thromboembolism seem to be the mechanisms involved.[157] Aortic atheroma is common in patients older than 60 yr who require coronary revascularization; atheroma greater than 2 mm was found in 35% and mobile atheroma occurred in 3.6%.[158] During cardiac surgery with CPB, the rate of stroke in patients with aortic arch atheroma is high (12%) and six times higher than the rate in controls undergoing noncardiac surgery.[156]

Atherosclerotic lesions in the descending thoracic aorta may be visualized in short and long axis views; the distance of the tip of the probe from the teeth should be noted to document the level of an abnormality. At the upper esophageal level, approximately 20 cm from the incisors, the distal aortic arch may be visualized between 0° and 90° (fig. 5; this TEE image is available on the ANESTHESIOLOGY Web site at http://www.anesthesiology.org). The aortic root and proximal half of the ascending aorta can be seen in mid-esophageal or basal views between 20° and 130°, and some of the mid–ascending aorta can be imaged through the right pulmonary artery (fig. 1G). However, there are blind spots on TEE for imaging the distal ascending aorta and proximal aortic arch because of air in the bronchus between the upper esophagus and these vessels. A recent case report de-

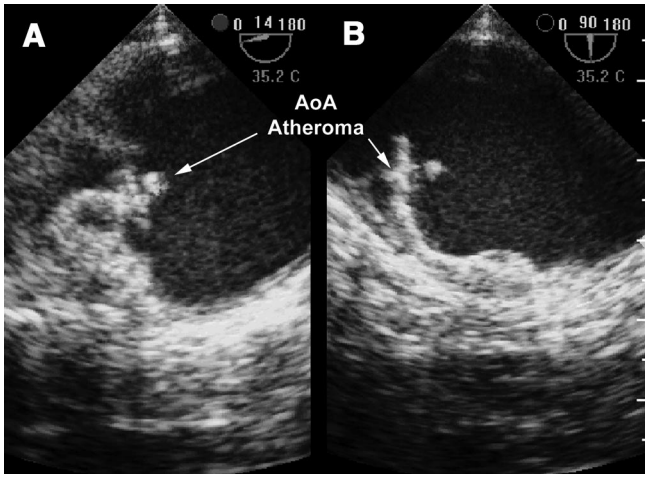

Fig. 5. Upper esophageal long (*A*) and short axis views (*B*) of the distal aortic arch (AoA) during routine coronary revascularization surgery. Grade V, complex atheroma detected (*arrows*).

scribed the use of a saline-filled balloon cuff of an endotracheal tube (inserted only when ventilation was terminated during CPB) to improve visualization of the proximal arch.[159]

Epiaortic echocardiography has been shown to provide additional visualization of the aorta.[160,161] In a study that enrolled 100 patients for cardiac surgery, the frequency for detection of mild, distal ascending aortic atheroma during epiaortic imaging was significantly higher than during TEE or digital palpation.[162] During an epiaortic scan, mapping of distribution of aortic atheroma is required. The ascending aorta may be divided into proximal, middle, and distal thirds. Each segment may have atheroma in its anterior, posterior, lateral, and medial walls.[163]

The severity of atheroma may be graded according to its morphology and appearance on echocardiography.[164] The modified Montgomery scale is as follows:

- Grade I: normal
- Grade II: mild intimal thickening greater than 2 mm
- Grade III: moderate, atheromatous plaque less than 4 mm
- Grade IV: severe, protruding atheromas 4 mm or greater
- Grade V: complex, ulcerated, and mobile atheroma of any size

It has high sensitivity and specificity and excellent reproducibility.[165] Evaluation of the thickness of atherosclerotic plaques in the aorta compares well with magnetic resonance imaging.[166]

The risk of stroke correlates with the size and complexity of atheroma:

Plaque Thickness. Increasing plaque thickness is associated with higher risk of stroke. Compared with controls without atheroma, the OR (95% CI) of stroke was 4.4 (2.8–6.8) in patients with plaques between 1 and 3.9 mm, and 13.8 (5.2–36.1) for plaques 4 mm or greater.[167] Similarly, Cohen *et al.* [168] reported an OR (95% CI) of 12.6 (7.7–17.6) for stroke in patients with plaques 4 mm or greater in the aortic arch. From analysis of receiver operator characteristics, plaque thickness greater than 3.5 mm was reported as the best predictor of cardiovascular events.[169]

Complex Plaque. Protruding plaques with ulceration, superimposed mobile thrombi, or noncalcified (fat-laden) areas confer additional independent risks for stroke and peripheral embolism. Compared with controls, the OR (95% CI) of embolism in patients with complex plaques was 17.1 (5.1–57.3).[170]

SEC in the Aorta. The presence of SEC in the thoracic aorta was associated with recent stroke in a prospective study of 224 patients and 85 controls undergoing TEE.[171] Compared with controls without aortic SEC, the OR (95% CI) of stroke in patients with SEC in the aorta was 2.8 (1.65–4.46; $P < 0.001$).

Surgical Manipulations of the Proximal Aorta. Aortic clamping[172] or cannulation[173] and aortic arch endarterectomy are associated with a threefold increase in perioperative stroke.[174]

Utility of Echocardiography. Detection of ascending aortic atheroma has been shown to influence clinical decision making during cardiac surgery.[175–177] Examples include modifications in

- Aortic cannulation
- Aortic cross clamping
- Proximal aortic vein anastomosis
- Cannulation for cardioplegia
- Deep hypothermic circulatory arrest for partial or complete ascending aortic replacement[178]

Furthermore, off-pump coronary artery bypass grafting[179] with minimal aortic manipulation[172] may be performed. Stroke rate was reduced from 2.2% in patients with severe atheroma and partial aortic cross clamp to 0.2% using the "no-touch" technique.[180] It is envisaged that these alterations may minimize the risk of atherothromboembolism and hence the occurrence of perioperative stroke. Off-pump coronary artery bypass grafting in high-risk patients with atheromatous aorta has been associated with lower risk of stroke and death.[181]

Other Sources of Embolism

Less common sources of embolism or features that have been associated with embolism and that may be detected by TEE include air embolism, mitral annular calcification, and valve strands.

Intracardiac Air Embolism

Air or microbubbles may be seen readily within the cardiac chambers during intraoperative TEE. They may

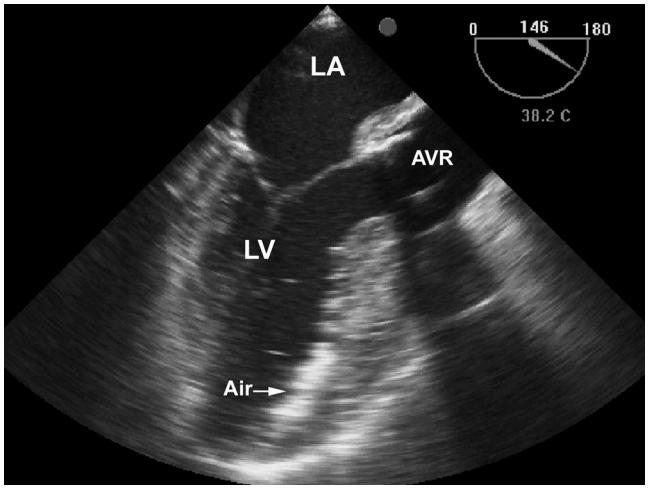

Fig. 6. Mid-esophageal long axis view of the left ventricle (LV) after aortic valve replacement (AVR) showing intracoronary air embolism. Note the presence of bright echogenic microbubbles (air) within the LV anteroseptal wall. LA = left atrium.

occur during neurosurgical procedures performed with the patient in a sitting position,[182] or during surgery involving any sites above the level of the heart. The incidence of venous air embolism during neurosurgical procedures has been reported to be high at 43%,[183] and venous air embolism with paradoxical air embolism has also been documented.[182]

After open heart surgery, clusters of air or numerous microbubbles are invariably seen within the cardiac chambers. Despite careful standard mechanical deairing, delayed release of air trapped in the pulmonary vessels is common. Numerous highly echo-reflective particles can be seen entering the LA from the pulmonary veins, in the mid-esophageal four-chamber view.[184] These echo-reflections by bubbles are relatively large and sparsely distributed, unlike the pattern seen with SEC. Bright, echogenic structures within the wall of myocardium often indicate intracoronary air embolism (fig. 6; this TEE image is available on the ANESTHESIOLOGY Web site at http://www.anesthesiology.org). This complication has been implicated in cardiac events after CPB.[185] Myocardial injury with transient ST elevation on electrocardiogram, conduction disturbances, and regional wall motion abnormalities, are more frequent in patients with intracavitary pooled air than in those with minimal appearance of microbubbles.[186]

During coronary revascularization with cardiopulmonary bypass, cerebral microembolization during the administration of drugs or blood[187] may be detected by transcranial Doppler ultrasound. The number of perfusion interventions and hence episodes of microembolization may contribute toward long-term cognitive dysfunction, *e.g.*, reduction in learning, memory, attention, and concentration.[188]

Mitral Annular Calcification

Mitral annular calcification (MAC) is a chronic, noninflammatory, degenerative process typically affecting the posterior annulus and appearing as an echo-dense, semilunar mass. It may extend to involve the intervalvular fibrosa, mitral leaflets, aortic annulus, and papillary muscle.[189] MAC occurs in 10% of patients older than 50 yr.[190] It is associated with aortic sclerosis and aortic annular calcification,[191] coronary artery disease,[192] atherosclerosis,[193] and end-stage renal disease.[194]

Mitral annular calcification seems to be an independent risk factor for cardiovascular disease and embolic stroke even in patients without AF, congestive heart failure, or coronary artery disease. The relative risk (95% CI) of stroke in individuals with MAC was 3.12 (1.77–5.25).[195] In the Framingham Heart Study, MAC was independently associated with an increased risk of stroke, with a hazard ratio (95% CI) of 1.5 (1.1–2.0) and a 10% increase in cardiovascular morbidity and mortality for each 1-mm increase in MAC.[196] Mobile elements associated with MAC that have been demonstrated in patients with end-stage renal disease[197] include thrombi,[198] vegetations,[199] and caseous abscess.[200] Therefore, emboli may be thrombotic or calcific.

Valve Strands

Valve strands are thin (< 1 mm), long (< 10 mm) filiform projections from heart valves that have undulating independent motion. Typically, they are observed near the closure line of the mitral valve. It was first suggested that they might be composed of fibrin, but morphologic analysis of strands recovered from native and prosthetic valves has revealed collagen.[201]

Valve strands were reported in 39% of elderly patients who had TEE for suspected cardioembolic stroke, giving an OR (95% CI) of ischemic stroke in patients with mitral valve strands of 2.2 (1.4–3.6; $P < 0.005$).[202] In other studies, however, no relation with brain infarction or clinical embolic events was demonstrated.[203]

Conclusion

We have shown that TEE is invaluable in patients who are at risk of cardioembolic events. It enables risk stratification, influences medical therapy, and refines clinical decision making in patients with thrombus, vegetations, tumor, aortic atheroma, and other sources of embolism.[204] It is envisaged that its routine use may improve patient outcome in the perioperative period.

References

1. Arrowsmith JE, Grocott HP, Reves JG, Newman MF: Central nervous system complications of cardiac surgery. Br J Anaesth 2000; 84:378–93

2. Murdock DK, Rengel LR, Schlund A, Olson KJ, Kaliebe JW, Johnkoski JA, Riveron FA: Stroke and atrial fibrillation following cardiac surgery. Wisc Med J 2003; 102:26–30

3. Di Tullio M, Homma S: Mechanisms of cardioembolic stroke. Curr Cardiol Rep 2002; 4:141–8
4. Lerakis S, Nichoholson W: Part I: Use of echocardiography in the evaluation of patients with suspected cardioembolic stroke. Am J Med Sci 2005; 329:310–16
5. Vitebskiy S, Fox K, Hoit BD: Routine transesophageal echocardiography for the evaluation of cerebral emboli in elderly patients. Echocardiography 2005; 22:770–4
6. Conture P, Denault AY, McKenty S, Boudreault D, Plante F, Perron Babin D, Normandin L, Poirier N: Impact of routine use of intraoperative TEE during cardiac surgery. Can J Anaesth 2000; 47:20–6
7. De Bruijn SF, Agema WR, Lammers GJ, van der Wall EE, Wolterbeek R, Holman ER, Bollen EL, Bax JJ: Transesophageal echocardiography is superior to transthoracic echocardiography in management of patients of any age with transient ischemic attack or stroke. Stroke 2006; 37:2531–4
8. Lengyel M, Horstkotte D, Voller H, Mistiaen WP, Working Group Infection, Thrombosis, Embolism and Bleeding of the Society for Heart Valve Disease: Recommendations for the management of prosthetic valve thrombosis. J Heart Valve Dis 2005; 14:567–75
9. Bach DS: Transesophageal echocardiography evaluation of prosthetic valves. Cardiol Clin 2000; 18:751–71
10. Jacob S, Tong AT: Role of echocardiography in the diagnosis and management of infective endocarditis. Curr Opin Cardiol 2002; 17:478–85
11. Peters PJ, Reinhardt S: The echocardiographic evaluation of intracardiac masses: A review. J Am Soc Echocardiogr 2006; 19:230–40
12. Shanewise JS, Cheung AT, Aronson S, Stewart WJ, Weiss RL, Mark JB, Savage RM, Sears-Rogan P, Mathew JP, Quinones MA, Cahalan MK, Savino JS: ASE/SCA guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography examination: Recommendations of the American Society of Echocardiography Council for Intraoperative Echocardiography and the Society of Cardiovascular Anesthesiologists Task Force for Certification in Perioperative Transesophageal Echocardiography. J Am Soc Echocardiogr 1999; 12:884–900
13. Shiga T, Wajima Z, Apfel CC, Inoue T, Ohe Y: Diagnostic accuracy of transesophageal echocardiography, helical computed tomography, and magnetic resonance imaging for suspected thoracic aortic dissection: Systematic review and meta-analysis. Arch Intern Med 2006; 166:1350–6
14. Min JK, Spencer KT, Furlong KT, DeCara JM, Sugeng L, Ward RP, Lang RM: Clinical features of complications from transesophageal echocardiography: A single-center case series of 10,000 consecutive examinations. J Am Soc Echocardiogr 2005; 18:925–9
15. Kallmeyer IJ, Collard CD, Fox JA, Body SC, Shernan SK: The safety of intraoperative transesophageal echocardiography: A case series of 7200 cardiac surgical patients. Anesth Analg 2001; 92:1126–30
16. Yamaji K, Fujimoto S, Yutani C, Hashimoto T, Nakamura S: Is the site of thrombus formation in the LAA associated with the risk of cerebral embolism? Cardiology 2002; 97:104–10
17. Von der Recke G, Schmidt H, Illien S, Luderitz B, Omran H: Use of transesophageal contrast echocardiography for excluding LAA thrombi in patients with atrial fibrillation before cardioversion. J Am Soc Echocardiogr 2002; 15:1256–61
18. Ha JW, Lee BK, Kim HJ, Pyun WB, Byun KH, Rim SJ, Chung N: Assessment of left atrial appendage filling pattern by using intravenous administration of microbubbles: Comparison between mitral stenosis and mitral regurgitation. J Am Soc Echocardiogr 2001; 14:1100–6
19. Kaymaz C, Ozdemir N, Kirma C, Sismanoglu M, Daglar B, Ozkan M: Location, size, and morphological characteristics of LA thrombi as assessed by echocardiography in patients with rheumatic mitral valve disease. Eur J Echocardiogr 2001; 2:270–6
20. Stoddard MF, Singh P, Dawn B, Longaker RA: Left atrial thrombus predicts transient ischemic attack in patients with atrial fibrillation. Am Heart J 2003; 145:676–82
21. Abe Y, Asakura T, Gotou J, Iwai M, Wantanabe Y, Sando M, Ishikawa S, Nagata K, Saito T, Maehara K, Maruyama Y: Prediction of embolism in atrial fibrillation: Classification of left atrial thrombi by transesophageal echocardiography. Jpn Circ J 2000; 64:411–5
22. Vincelj J, Sookol I, Jaksic O: Prevalence and significance of left atrial SEC detected by transesophageal echocardiography. Echocardiography 2002; 19: 319–24
23. Maltagliati A, Pepi M, Tamborini G, Muratori M, Celeste F, Doria E, Galli C: Usefulness of multiplane TEE in the recognition of artifacts and normal variants that may mimic LA thrombi in patients with atrial fibrillation. Ital Heart J 2003; 4:797–802
24. Stollberger C, Chnupa P, Kronik G, Brainin M, Finsterer J, Schneider B, Slany J: Transesophageal echocardiography to assess embolic risk in patients with atrial fibrillation. ELAT Study Group. Embolism in Left Atrial Thrombi. Ann Intern Med 1998; 128:630–8
25. Srimannarayana J, Varma RS, Satheesh S, Anilkumar R, Balachander J: Prevalence of left atrial thrombus in rheumatic mitral stenosis with atrial fibrillation and its response to anticoagulation: A transesophageal echocardiographic study. Indian Heart J 2003; 55:358–61
26. Gonzalez-Torrecilla E, Garcia-Fernandez MA, Perez-David E, Bermejo J, Moreno J, Delcan JL: Predictors of LA spontaneous echo contrast and thrombi in patients with mitral stenosis and atrial fibrillation. Am J Cardiol 2000; 86:529–34
27. Lopez-Candales A, Edelman K: Large left atrial appendage thrombus in the presence of severe mitral regurgitation: Contradictory hemodynamics or expected findings. Echocardiography 2004; 2:625–9
28. Laplace G, Lafitte S, Labeque JN, Perron JM, Baudet E, Deville C, Roques X, Roudaut R: Clinical significance of early thrombosis after prosthetic mitral valve replacement: A postoperative monocentric study of 680 patients. J Am Coll Cardiol 2004; 43:1283–90
29. Panagiotopoulos K, Toumanidis S, Vemmos K, Saridakis N, Stamatelopoulous S: Secondary prognosis after cardioembolic stroke of atrial origin: The role of left atrial and left atrial appendage dysfunction. Clin Cardiol 2003; 26:269–74
30. Sadanandans S, Sherrid MV: Clinical and echocardiographic characteristics of left atrial spontaneous echo contrast in sinus rhythm. J Am Coll Cardiol 2000; 35:1932–38
31. Ito T, Suwa M, Kobashi A, Yagi H, Nakamura T, Miyazaki S, Kitaura Y: Integrated backscatter assessment of left atrial SEC in chronic nonvalvular AF: Relation with clinical and echocardiographic parameters. J Am Soc Echocardiogr 2000; 13:666–73
32. Bashir M, Asher CR, Schaffer K, Murray RD, Apperson-Hansen C, Jasper S, Thomas JD: Left atrial appendage SEC in patients with arrhythmia using integrated backscatter and transesophageal echocardiography. Am J Cardiol 2001; 88:923–5
33. Bernhardt P, Schmidt H, Hammersting C, Luderitz B, Omran H: Patients with AF and dense SEC at high risk: A prospective and serial follow-up over 12 months with TEE and cerebral MRI. J Am Coll Cardiol 2005; 45:1807–12
34. Handke M, Harloff A, Hetzel A, Olschewski M, Bode C, Geibel A: Left atrial appendage flow velocity as a quantitative surrogate parameter for thromboembolic risk: Determinants and relationship to spontaneous echocontrast and thrombus formation- a transesophageal echocardiographic study in 500 patients with cerebral ischemia. J Am Soc Echocardiogr 2005; 18:1366–72
35. Bashir M, Asher CR, Garcia MJ, Abdalla I, Jasper SE, Murray RD, Grimm RA, Thomas JD, Klein AL: Atrial spontaneous echo contrast and thrombi in atrial fibrillation: A transesophageal echocardiography study. J Am Soc Echocardiogr 2001; 14:122–7
36. Thumala A, Parra C, Maragano P, Puelma A, Florenzano F: Thromboembolic risk factors in atrial flutter: A transesophageal echocardiographic study. Rev Med Chil 2000; 128:1327–34
37. Shaw TR, Northridge DB, Francis CM: Left atrial standstill in a patient with mitral stenosis and sinus rhythm: A risk of thrombus hidden by left and right atrial electrical dissociation. Heart 2003; 89:1173
38. Corrado G, Beretta S, Sormani L, Tadeo G, Foglia-Manzillo G, Tagliagambe LM, Santarone M: Prevalence of atrial thrombi in patients with atrial fibrillation and subtherapeutic anticoagulant prior to cardioversion. Eur J Echocardiogr 2004; 5:257–61
39. Wang YC, Lin JL, Hwang JJ, Lin MS, Tseng CD, Huang SK, Lai LP, Wang YS: Left atrial dysfunction in patients with atrial fibrillation after successful rhythm control for more than 3 months. Chest 2005; 128:2551–6
40. Ren JF, Marchlinski FE, Callans DJ: Left atrial thrombus associated with ablation for atrial fibrillation: Identification with intracardiac echocardiography. J Am Coll Cardiol 2004; 43:1861–7
41. Khan IA: Atrial stunning: Basic and clinical considerations. Int J Cardiol 2003; 92:113–28
42. Klein AL, Grimm RA, Jasper SE, Murray RD, Apperson-Hansen C, Lieber EA, Black IW, Davidoff R, Erbel R, Halperin JL, Orsinelli DA, Porter TR, Stoddard MF: ACUTE Steering and Publication committee for the ACUTE investigators: Efficacy of transesophageal echocardiography-guided cardioversion of patients with atrial fibrillation at 6 months: A randomized controlled trial. Am Heart J 2006; 151:380–9
43. Thambidorai SK, Murray RD, Parakh K, Shah TK, Black IW, Jasper SE, Li J, Appleson-Hansen C, Asher CR, Grimm RA, Klein AL: Utility of transesophageal echocardiography in identification of thrombogenic milieu in patients with atrial fibrillation (an ACUTE ancillary study). Am J Cardiol 2005; 96:935–41
44. Jaber WA, Prior DL, Thamilarsasan M, Grimma RA, Thomas JD, Klein AL, Asher CR: Efficacy of anticoagulation in resolving left atrium and left atrial appendage thrombi: A transesophageal echocardiographic study. Am Heart J 2000; 140:150–6
45. Tansuphaswadikul S, Hengrussamee K, Chantadansuwan T, Silaruks S, Kehasukcharoen W, Saejueng B: Transesophageal echocardiography during percutaneous mitral commissurotomy in patients with left atrial thrombus. J Med Assoc Thai 2001; 84:1534–40
46. Ostermayer SH, Reisman M, Kramer PH, Mathew RV, Gray WA, Block PC, Omran H, Bartorelli AL, Della BP, Di Mario C, Pappone C, Casale PN, Moses JW, Poppas A, Williams DO, Meier B, Skanes A, Teirstein PS, Lesh MD, Nakai T, Bayard Y, Billinger K, Trepels T, Krumsdorf U, Sievert H: Percutaneous left atrial appendage transcatheter occlusion (PLAATO) system to prevent stroke in high risk patients with non-rheumatic atrial fibrillation: Results from the international multicenter feasibility trials. J Am Coll Cardiol 2005; 46:9–14
47. Ha JW, Chung N, Hong YW, Kwak YR, Chang BC, Cho SY: Alteration of surgical management following intraoperative transesophageal echocardiography in a patient with mobile left atrial thrombi embolized during anesthesia. Echocardiography 2003; 20:291–2
48. Garcia-Fernandez MA, Perez-David E, Quiles J, Peralta J, Garcia-Rojas I, Bermejo J, Moreno M, Silva J: Role of left atrial appendage obliteration in stroke

reduction in patients with mitral valve prosthesis: A transesophageal echocardiographic study. J Am Coll Cardiol 2003; 42:1253–8

49. Schneider B, Stollberger C, Sievers HH: Surgical closure of the left atrial appendage: A beneficial procedure? Cardiology 2005; 104:127–32

50. Barkhausen J, Hunold P, Eggebrecht H, Schuler WO, Sabin GV, Erbel R, Debatin JF: Detection and characterization of intracardiac thrombi on MRI. Am J Roentgenol 2002; 179:1539–44

51. Moreno R, Zamorano JL, Almeria C, Rodrigo JL, Villate A, Serra V, Alvarez L, Aubele A, Sanchez-Harguindey L: Usefulness of contrast agents in the diagnosis of LV aneurysm after acute myocardial infarction. Eur J Echocardiogr 2003; 3:111–6

52. Bednarz JE, Spencer KT, Weinert L, Sugeng L, Mor-Avi V, Lang RM: Identification of cardiac masses and abnormal blood flow patterns with harmonic power Doppler contrast echocardiography. J Am Soc Echocardiogr 1999; 12: 871–5

53. Heatlie GJ, Mohiaddin R: Left ventricular aneurysm: Comprehensive assessment of morphology and thrombus using cardiovascular MRI. Clin Radiol 2005; 60:687–92

54. Butany J, Dias B, Grapa J, Mickleborough L, Siu S: Left ventricular pseudoaneurysm. Can J Cardiol 2002; 18:1122–3

55. Moreno R, Gordillo E, Zamorano J, Almeria C, Garcia-Rubira JC, Fernandez-Ortiz A, Macaya C: Long term outcome of patients with post-infarction LV pseudoaneurysm. Heart 2003; 89:1144–6

56. Makaryus AN, Manetta F, Goldner B, Stephen B, Rosen SE, Park CH: Large left ventricular pseudoaneurysm presenting 25 years after penetrating chest trauma. J Interv Cardiol 2005; 18:193–200

57. Stollberger C, Finsterer J: Left ventricular hypertrabeculation/non-compaction. J Am Soc Echocardiogr 2004; 17:91–100

58. Aragona P, Badano LP, Pacileo G, Pino GP, Sinagra G, Zachara E: Isolated left ventricular non-compaction. Ital Heart J 2005; 6:649–59

59. Lowery MH, Martel JA, Zambrano JP, Ferreira A, Eco L, Gallagher A: Non-compaction of the ventricular myocardium: The use of contrast-enhanced echocardiography in diagnosis. J Am Soc Echocardiogr 2003; 16:94–6

60. Koo BK, Choi D, Ha JW, Kang SM, Chung N, Cho SY: Isolated non-compaction of the ventricular myocardium: Contrast echocardiographic findings and review of the literature. Echocardiography 2002; 19:153–6

61. Oechslin EN, Attenhofer Jost CH, Rojas JR, Kaufmann PA, Jenni R: Long-term follow-up of 34 adults with isolated left ventricular non-compaction: A distinct cardiomyopathy with poor prognosis. J Am Coll Cardiol 2000; 19:153–6

62. Blessing E, Rottbauer W, Mereles D, Hosch W, Benz A, Friess H, Autschbach F, Muller M, Stremmel W, Katu H: Isolated Left ventricular noncompaction of the myocardium as a cause of embolic SMA occlusion. J Am Soc Echocardiogr 2005; 6:693

63. Ileri M, Tandogan I, Kosar F, Yetkin E, Buyukasik Y, Kutuk E: Influence of thrombolytic therapy on the incidence of LV thrombi after acute anterior myocardial infarction: Role of successful reperfusion. Clin Cardiol 1999; 22:477–80

64. Neskovic AN, Marinkovic J, Bojic M, Popovic AD: Predictors of Left ventricular thrombus formation and disappearance after anterior wall myocardial infarction. Eur Heart J 1998; 19:908–16

65. Miyake Y, Sugioka K, Bussey CD, Di Tullio M, Homma S: Left ventricular mobile thrombus associated with ventricular assist device: Diagnosis by transesophageal echocardiography. Circ J 2004; 68:383–4

66. Yilmaz R, Celik S, Baykan M, Kasap H, Kaplan S, Kucukosmanoglu M, Erdol C: Assessment of mitral annular velocities by Doppler tissue imaging in predicting LV thrombus formation after first acute anterior myocardial infarction. J Am Soc Echocardiogr 2005; 18:632–37

67. Sharma S, Ehsan A, Couper GS, Shernan SK, Wholey RM, Aranki SF: Unrecognised LV thrombus during reoperative CABG. Ann Thorac Surg 2004; 78:79–80

68. Scalia GM, McCarthy PM, Savage RM, Smedira NG, Thomas JD: Clinical utility of echocardiography in the management of implantable ventricular assist devices. J Am Soc Echocardiogr 2000; 13:754–63

69. Banchs JE, Dawn B, Abdel-Latif A, Qureshi A, Agrawal N, Bouvette M, Stoddard MF: Acquired aortic cusp fusion after chronic left ventricular assist device support. J Am Soc Echocardiogr 2006; 19:1401.e1–3

70. Ogren M, Bregqvist D, Eriksson H, Lindblad B, Sternby NH: Prevalence and risk of pulmonary embolism in patients with intracardiac thrombosis: A population-based study of 23 769 consecutive autopsies. Eur Heart J 2005; 26:1108–14

71. Shapiro MA, Johnson M, Feintein SB: A retrospective experience of right atrium and superior vena cava thrombi diagnosed by transesophageal echocardiography. J Am Soc Echocardiogr 2002; 15:76–9

72. Khairy P, Landzberg MJ, Gatzoulis MA, Mercier LA, Fernandes SM, Cote JM, Lavoie JP, Fournier A, Guerra PG, Frogoudaki A, Walsh EP, Dore A, Epicardial *versus* Endocardial Pacing and Thromboembolic Events Investigators: Transvenous pacing leads and systemic thromboemboli in patients with intracardiac shunts: A multicenter study. Circulation 2006; 113:2391–7

73. Kang CH, Ahn H, Kim KH, Kim KB: Long-term result of 1144 CarboMedics mechanical valve implantations. Ann Thorac Surg 2005; 79:1939–44

74. Shapira Y, Nill M, Hirsch R, Vaturi M, Vidne B, Sagie A: Mid-term clinical and echocardiographic follow-up of patients with CarboMedic valves in the tricuspid position. J Heart Valve Dis 2000; 9:396–402

75. Wenke K, Goppl J, Kronski D, Nowak L, Kemkes B: Endocarditis resulting from thrombotic vegetations on a right ventricular pacing lead. Herz 2005; 30:668–74

76. Ruiz M, Anguita M, Castillo JC, Delgado M, Romo E, Torres F, Mesa D, Franco M, Valles F: Pacemaker-related endocarditis: Clinical features and treatment. J Heart Valve Dis 2006; 15:122–4

77. Mansencal N, Dubourg O: Free-floating thrombus in the right heart and pulmonary embolism. Int J Cardiol 2006; 112:33–4

78. Basarici I, Yilmaz H, Demir I, Yalcinkaya S: Imminent pulmonary embolism: A fatal mobile right atrial thrombus. Int J Cardiovasc Imag 2006; 22:55–8

79. Antonini-Canterin F, Sandrini R, Pavan D, Zardo F, Nicolosi GL: Right ventricular thrombosis in arrhythmogenic cardiomyopathy. Ital Heart J 2000; 1:415–8

80. Wlodarska EK, Wozniak O, Konka M, Rydlewska-Sadowska W, Biederman A, Hoffman P: Thromboembolic complications in patients with arrhythmogenic right ventricular dysplasia/cardiomyopathy. Europace 2006; 8:596–600

81. Pellett AA, Kerut EK: The Chiari network in an echocardiography student. Echocardiography 2004; 21:91–3

82. Rosenberger P, Shernan SK, Body SC, Eltzschig HK: Utility of intraoperative transesophageal echocardiography for diagnosis of pulmonary embolism. Anesth Analg 2004; 99:12–6

83. Rosenberger P, Shernan SK, Mihaljevic T, Eltzschig HK: Transesophageal echocardiography for detecting extrapulmonary thrombi during pulmonary embolectomy. Ann Thorac Surg 2004; 78:862–6

84. Sivaram CA, Craven P, Chandrasekaran K: Transesophageal echocardiography during removal of central venous catheter associated with thrombus in superior vena cava. Am J Card Imaging 1996; 10:266–9

85. Weber T, Huemer G, Tschernich H, Kranz A, Imhof M, Sladen RN: Catheter-induced thrombus in the superior vena cava diagnosed by transesophageal echocardiography. Acta Anaesthesiol Scand 1998; 42:1227–30

86. Roe MT, Abramson MA, Li J, Heinle SK, Kisslo J, Corey GR, Sexton DJ: Clinical information determines the impact of transesophageal echocardiography on the diagnosis of infective endocarditis by the Duke criteria. Am Heart J 2000; 139:945–51

87. Cabell CH, Pond KK, Peterson GE, Durack DT, Corey GR, Anderson DJ, Ryan T, Lukes AS, Sexton DJ: The risk of stroke and death in patients with aortic and mitral valve endocarditis. Am Heart J 2001; 142:75–80

88. Di Salvo G, Habib G, Pergola V, Avierinos JF, Philip E, Casalta JP, Vailloud JM, Derumeaux G, Gouvernet J, Ambrosi P, Lambert M, Ferracci A, Raoult D, Luccioni R: Echocardiography predicts embolic events in infective endocarditis. J Am Coll Cardiol 2001; 37:1069–76

89. Deprele C, Berthelot P, Lemetayer F, Corntet C, Fresard A, Cazoria C, Fascia P, Cathebras P, Chaumentin G, Convert G, Isaaz K, Barral X, Lucht F: Risk factors for systemic emboli in infective endocarditis. Clin Microbiol infect 2004; 10:46–53

90. Vilacosta I, Graupner C, San Roman JA, Sarria C, Ronderos R, Fernandez C, Mancini L, Sanz O, Sanmartin JV, Steermann W: Risk of embolization after institution of antibiotics therapy for infective endocarditis. J Am Coll Cardiol 2002; 39:1489–95

91. Heiro M, Nikoskelainen J, Engblom E, Kotilainen E, Marttila R, Kotilainen P: Neurologic manifestations of infective endocarditis: A 17 year experience in a teaching hospital in Finland. Arch Intern Med 2000; 160:2781–7

92. Karski JM: Transesophageal echocardiography in the intensive care unit. J Cardiothorac Vasc Anesth 2006; 10:162–70

93. Haldar SM, O'Gara PT: Infective endocarditis: Diagnosis and management. Nat Clin Pract Cardiovasc Med 2006; 3:310–7

94. Anguera I, Miro JM, Cabell CH, Abrutyn E, Fowler VG Jr, Hoen B, Olaison L, Pappas PA, de Lazzari E, Eykyn S, Habib G, Pare C, Wang A, Corey R, ICE-MD Investigators: Clinical characteristics and outcome of aortic endocarditis with periannular abscess in the International Collaboration on Endocarditis Merged Database. Am J Cardiol 2005; 96:976–81

95. Anguera I, Miro JM, Vilacosta I, Almirante B, Anguita M, Roman JA, De-Alarcon A, Ripoll T, Navas E, Gonzalez-Juanatey C, Cabell CH, Sarria C, Garcia-Bolao I, Farinas MC, Leta R, Rufi G, Miralles F, Pare C, Evangelista A, Fowler VG, Mestres CA, De-Lazzari E, Guma JR, Aorto-cavitary Fistula in Endocarditis Working Group: Aorto-cavitary fistulous tract formation in infective endocarditis: Clinical and echo features of 76 cases and risk factors for mortality. Eur Heart J 2005; 26:288–97

96. Mathew J, Anand A, Addai T, Freels S: Value of echocardiographic findings in predicting cardiovascular complications in infective endocarditis. Angiology 2001; 52:801–9

97. Ragland MM, Tak T: The role of echocardiography in diagnosing space-occupying lesions of the heart. Clin Med Res 2006; 4:22–32

98. Collins NJ, Barlow MA, Woodford PA, Hayes PC: Intracardiac extension of metastatic pulmonary leiomyosarcoma. Heart Lung Circ 2005; 14:121–2

99. Gates GF, Aronsky A, Ozgur H: Intracardiac extension of lung cancer demonstrated on PET scanning. Clin Nucl Med 2006; 31:68–70

100. Parekh DJ, Cookson MS, Chapman W, Harrell JR, Wells N, Chang MS, Smith JA: Renal cell carcinoma with renal vein and inferior vena cava involvement: Clinicopathological features, surgical techniques and outcomes. J Urology 2005; 173:1897–902

101. Reck R, Speth M, Kirch Stanek M, Hedemann A, Von Mengden HJ: Vascularized atrial thrombus: A rare differential atrial myxoma diagnosis. Med Clin North Am 1996; 91:413–6

102. Pappas KD, Arnaoutoglou E, Papadopoulos G: Giant atrial septal aneurysm simulating a right atrial tumor. Heart 2004; 90:493
103. Ginon I, Mestrallet C, Barthelet M, Robin J, Andre Fouel X: A closed interatrial septum aneurysm filled with blood mimicking a tumor in the right atrium. Eur J Echocardiogr 2000; 1:289–90
104. Anfinsen OG, Aaberge L, Geiran O, Smith HJ, Aakhus S: Coronary artery aneurysms mimicking cardiac tumor. Eur J Echocardiogr 2004; 5:308–12
105. Gaudio C, Di Michele, S Cera M, Nguyen BL, Pannarale G, Alessandri N: Prominent crista terminalis mimicking a RA myxoma: Cardiac magnetic resonance aspects. Eur Rev Med Pharmacol Sci 2005; 8:165–8
106. Moinuddeen K, Marica S, Clausi RL, Zama N: Lipomatous interatrial septal hypertrophy: An unusual cause of intracardiac mass. J Thorac Cardiovasc Surg 2002; 22:468–9
107. O' Connor S, Recavarren R, Nichols LC, Parwani AV: Lipomatous hypertrophy of the interatrial septum: An overview. Arch Pathol Lab Med 2006; 130:397–9
108. Nadra I, Dawson D, Schmitz SA, Punjabi PP, Nihoyannopoulos P: Lipomatous hypertrophy of the interatrial septum: A commonly misdiagnosed mass leading to unnecessary cardiac surgery. Heart 2004; 90:66
109. Mittle S, Makaryus AN, Boutis L, Hartman A, Rosman D, Kort S: Right-sided myxomas. J Am Soc Echocardiogr 2005; 18:695
110. Sugeng L, Lang RM: Atypical cardiac myxomas. Echocardiography 2004; 21:43–7
111. Idir M, Oysel N, Guibaud JP, Labouyrie E, Roudaut R: Fragmentation of a RA myxoma presenting as a pulmonary embolism. J Am Soc Echocardiogr 2000; 13:61–3
112. Braun S, Schrotter H, Reynen K, Schwencke C, Strasser RH: Myocardial infarction as a complication of LA myxoma. Int J Cardiol 2005; 101:115–21
113. Fang BR, Chang CP, Cheng CW, Yang NI, Shieh MC, Lee N: Total detachment of cardiac myxoma causing saddle embolization and mimicking aortic dissection. Jpn Heart J 2004; 45:359–63
114. Chang YS, Chu PH, Jung SM, Lim KE, Chu JJ, Hsueh C, Lee YS: Unusual cardiac papillary fibroelastoma in the right ventricular outflow tract. Cardiovasc Pathol 2005; 14:104–6
115. Costa MJ, Makaryus AN, Rosman DR: A rare case of a cardiac papillary fibroelastoma of the pulmonary valve diagnosed by echocardiography. Int J Cardiovasc Imaging 2006; 22:199–203
116. Misawa Y, Kaminishi Y, Taguchi M: Cardiac papillary fibroelastoma. Ann Thorac Surg 2006; 82:381
117. Sun JP, Asher CR, Yang XS, Cheng GG, Scalia GM, Massed AG, Griffin BP, Ratliff NB, Stewart WJ, Thomas JD: Clinical and echocardiographic characteristics of papillary fibroelastomas: A retrospective and prospective study in 162 patients. Circulation 2001; 103:2687–93
118. Basso C, Bottio T, Valente M, Bonato R, Casarotto D, Thiene G: Primary cardiac valve tumours. Heart 2003; 89:1259–60
119. Gowda RM, Khan IA, Nair CK, Mehta NJ, Vasavada BC, Sacchi TJ: Cardiac papillary fibroelastoma: A comprehensive analysis of 725 cases. Am Heart J 2003; 146:404–10
120. Cheitlin MD, Armstrong WF, Aurigemma GP, Beller GA, Bierman FZ, Davis JL, Douglas PS, Faxon DP, Gillam LD, Kimball TR, Kussmaul WG, Pearlman AS, Philbrick JT, Rakowski H, Thys DM, Antman EM, Smith SC Jr, Alpert JS, Gregoratos G, Anderson JL, Hiratzka LF, Faxon DP, Hunt SA, Fuster V, Jacobs AK, Gibbons RJ, Russell RO, ACC, AHA, ASE: ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography: Summary article. A report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography). J Am Soc Echocardiogr 2003; 16:1091–110
121. Chen H, Ng V, Kane CJ, Russell IA: The role of transesophageal echocardiography in rapid diagnosis and treatment of migratory tumor embolus. Anesth Analg 2004; 99:357–9
122. Komanapalli CB, Tripathy U, Sokoloff M, Daneshmand S, Das A, Slater MS: Intraoperative renal cell carcinoma tumor embolization to the right atrium: Incidental diagnosis by transesophageal echocardiography. Anesth Analg 2006; 102:378–9
123. Kaplan S, Ekici S, Dogan R, Demircin M, Ozen H, Pasaoglu I: Surgical management of renal cell carcinoma with inferior vena cava tumor thrombus. Am J Surg 2002; 183:292–9
124. Nakamura K, Asai T, Murakami M, Saito Y, Yoshimoto A, Yamaguchi H: Giant right atrial myxoma associated with tricuspid regurgitation. Jpn J Thorac Cardiovasc Surg 2006; 54:332–4
125. Eslami-Varaneh F, Brun EA, Sears-Rogan P: An unusual case of multiple fibroelastoma: Review of literature. Cardiovasc Pathol 2003; 12:170–3
126. Sadeghi N, Sadeghi S, Karimi A: Mitral valve recurrence of a left atrial myxoma. Eur J Cardiothorac Surg 2002; 21:568–73
127. Serafini O, Misuraca G, Greco F, Bisignani G, Manes MT, Venneri N: Prevalence of structural abnormalities of the atrial septum and their association with recent ischemic stroke or transient ischemic attack: Echocardiographic evaluation in 18631 patients. Ital Heart J 2003; 4:39–45
128. Payne DM, Baskett RJ, Hirsch GM: Infectious endocarditis of a Chiari network. Ann Thorac Surg 2003; 76:1303–5
129. Kizer JR, Devereux RB: Patent foramen ovale in young adults with unexplained stroke. N Engl J Med 2005; 353:2361–72
130. Kerut EK, Norfleet WT, Plotnick GD, Giles TD: Patent foramen ovale: A review of associated conditions and the impact of physiological size. J Am Coll Cardiol 2001; 38:613–23
131. Augoustides JG, Weiss SJ, Ochroch AE, Weiner J, Mancini J, Savino JS, Cheung AT: Analysis of the interatrial septum by transesophageal echocardiography in adult cardiac surgical patients: Anatomic variants and correlation with patent foramen ovale. J Cardiothorac Vasc Anesth 2005; 19:146–9
132. Woods TD, Patel A: A critical review of patent foramen ovale detection using saline contrast echocardiography: When bubbles lie. J Am Soc Echocardiogr 2006; 19:215–22
133. Yoshida M, Goto S, Aikawa M, Oguma T, Nakajima T, Abe S, Kumagai A, Hoshiba Y, Tanabe T, Handa S, Yamamoto M: Detection of right to left shunting through a patent foramen ovale in Japanese patients with ischemic stroke by transesophageal echocardiography using a standardized Valsalva maneuver. Tokai J Exp Clin Med 2005; 30:211–6
134. Feigenbaum H, Armstrong WF, Ryan T: Masses, tumors, and source of embolus, Feigenbaum's Echocardiography, 6th edition. Edited by Feigenbaum H. Philadelphia: Williams & Wilkins, 2005, pp 701–34
135. Overell JR, Bone I, Less KR: Interatrial septal abnormalities and stroke: A meta-analysis of case control studies. Neurology 2000; 55:1172–9
136. Natanzon A, Goldman ME: Patent foramen ovale: Anatomy *versus* pathophysiology which determines stroke risk? J Am Soc Echocardiogr 2003; 16:71–6
137. Schmitt HJ, Hemmerling TM: Venous air emboli occur during release of positive end-expiratory pressure and repositioning after sitting position surgery. Anesth Analg 2002; 94:400–3
138. Coles RE, Clements FM, Lardenoye JW, Wermeskerken GV, Hey LA, Nunley JA, Levin LS, Pearsall AW IV: Transesophageal echocardiography in quantification of emboli during femoral nailing: Reamed *versus* unreamed techniques. J South Orthop Assoc 2000; 9:98–104
139. Yasaka M, Otsubo R, Minematsu K: Is stroke a paradoxical embolism in patients with patent foramen ovale? Intern Med 2005; 44:434–8
140. Sukernik MR, Mets B, Bennett-Guerrero E: Patent foramen ovale and its significance in the perioperative period. Anesth Analg 2001; 93:1137–46
141. Mets B: Anesthesia for left ventricular assist device placement: J Cardiothorac Vasc Anesth 2000; 14:316–26
142. Wahl A, Krumsdorf U, Meier B, Sievert H, Ostermayer S, Billinger K, Schwerzmann M, Becker U, Seiler C, Arnold M, Mattle HP, Windecker S: Transcatheter treatment of atrial septal aneurysm associated with patent foramen ovale for prevention of recurrent paradoxical embolism in high-risk patients. J Am Coll Cardiol 2005; 45:377–80
143. Wang JK, Tsai SK, Wu MH, Lin MT, Lue HC: Short and intermediate term results of transcatheter closure of atrial septal defect with the Amplatzer septal occluder. Am Heart J 2004; 148:511–7
144. Mattioli AV, Aquilina M, Oldani A, Longhini C, Mattioli G: Atrial septal aneurysm as a cardioembolic source in adult patients with stroke and normal carotid arteries: A multicentre study. Eur Heart J 2001; 22:261–8
145. Mattioli AV, Bonetti L, Aquilina M, Oldani A, Longhini C, Mattioli G: Association between ASA and PFO in young patients with recent stroke and normal carotid arteries. Cerebrovasc Dis 2003; 15:4–10
146. Schneider B, Hanrath P, Vogel P, Meinertz T: Improved morphologic characterization of atrial septal aneurysm by transesophageal echocardiography: Relation to cerebrovascular events. J Am Coll Cardiol 1990; 16:1000–9
147. Olivares-Reyes A, Chan S, Lazar EJ, Bandlamudi K, Naria V, Ong K: Atrial Septal Aneurysm: A new classification in 205 adults. J Am Soc Echocardiogr 1997; 10:644–56
148. Mas JL, Arquizan C, Lamy C, Zuber M, Cabanes L, Derumeaux G, Coste J: Recurrent cerebrovascular events associated with PFO, ASA, or both. N Engl J Med 2001; 345:1740–6
149. Burger AJ, Sherman HB, Charlamb MJ: Low incidence of embolic strokes with ASA: A prospective, long-term study. Am Heart J 2000; 139:149–52
150. Augoustides J, Mancini DJ, Horak J, Pochettino A, Dupont F, Dowling RD: Case 1: The use of intraoperative echocardiography during insertion of ventricular assist devices. J Cardiothorac Vasc Anesth 2003; 17:113–20
151. Schneider B, Hofmann T, Justen MH, Meinertz T: Chiari Network: Normal anatomic variant or risk factor for arterial embolic events? J Am Coll Cardiol 1995; 26:203–10
152. Becker A, Buss M, Sebening W, Meisner H, Dohlemann C: Acute inferior cardiac inflow obstruction resulting from inadvertent surgical closure of a prominent Eustachian valve. Pediatr Cardiol 1999; 20:155–7
153. Schuchlenz HW, Saurer G, Weihs W, Rehak P: Persisting Eustachian valve in adults: Relation to PFO and cerebrovascular events. J Am Soc Echocardiogr 2004; 17:231–3
154. Cooke JC, Gelman JS, Harper RW: Chiari network entanglement and herniation into the left atrium by an atrial septal defect occluder device. J Am Soc Echocardiogr 1999; 12:601–3
155. Djaiani G, Fedorko L, Borger M, Mikulis D, Carroll J, Cheng D, Karkouti K, Beattie S, Karski J: Mild to moderate atheromatous disease of the thoracic aorta and new ischemic brain lesion after conventional coronary artery bypass graft surgery. Stroke 2004; 35:356–8
156. Macleod MR, Amarenco P, Davies SM, Donnan GA: Atheroma of the aortic arch: An important and poorly recognized factor in the aetiology of stroke. Lancet Neurol 2004; 3:408–14

157. Tunick PA, Kronzon I: Atheroma of the thoracic aorta: Clinical and therapeutic update. J Am Coll Cardiol 2000; 35:545–54
158. Djaiani G: Aortic arch atheroma: Stroke reduction in cardiac surgical patients. J Cardiothorac Vasc Anesth 2006; 10:143–57
159. Li YL, Wong DT, Wei W, Liu J: A new method for detecting the proximal aortic arch and innominate artery by transesophageal echocardiography. ANESTHESIOLOGY 2006; 105:226–7
160. Eltzschig HK, Kallmeyer IJ, Mihaljevic T, Alapati S, Shernan SK: A practical approach to a comprehensive epicardial and epiaortic echocardiographic examination. J Cardiothorac Vasc Anesth 2003; 17:422–9
161. Zingone B, Rauber E, Gatti G, Pappalardo A, Benussi B, Dreas L, Lattuada L: The impact of epiaortic ultrasonographic scanning on the risk of perioperative stroke. Eur J Cardiothorac Surg 2006; 29:720–8
162. Sylivris S, Calafiore P, Matalanis G, Rosalion A, Yuen HP, Buxton BF, Tonkin AM: The intraoperative assessment of ascending aortic atheroma: Epiaortic imaging is superior to both transesophageal echocardiography and direct palpation. J Cardiothorac Vasc Anesth 1997; 11:704–7
163. Van der Linden J, Hadjinikolaou L, Bergman P, Lindblom D: Postoperative stroke in cardiac surgery is related to the location and extent of atherosclerotic disease in the ascending aorta. J Am Coll Cardiol 2001; 38:131–5
164. Wilson MJ, Boyd SY, Lisagor PG, Rubal BJ, Cohen DJ: Ascending aortic atheroma assessed intraoperatively by epiaortic and transesophageal echocardiography. Ann Thorac Surg 2000; 70:25–30
165. Zaidat OO, Suarez JI, Hedrick D, Redline S, Schiuchter M, Landis DM, Hoit B: Reproducibility of transeosophageal echocardiography in evaluating aortic atheroma in stroke patients. Echocardiography 2005; 22:326–30
166. Fayad ZA, Nahar T, Fallon JT, Goldman M, Aguinaldo JG, Badimon JJ, Shinnar M, Chesebro JH, Fuster V: *In vivo* MRI evaluation of atherosclerotic plagues in the human thoracic aorta: A comparison with TEE. Circulation 2000; 101:2503–9
167. Donnan GA, Davis SM, Jones EF, Amarenco P: Aortic source of brain embolism. Curr Treat Options Cardiovasc Med 2003; 5:211–19
168. Cohen A, Amarenco P: Atherosclerosis of the thoracic aorta: From risk stratification to treatment. Am J Cardiol 2002; 90:1333–5
169. Tanaka M, Yasaka M, Nagano K, Otsubo R, Oe H, Naritomi H: Moderate atheroma of the aortic arch and the risk of stroke. Cerebrovas Dis 2006; 21:26–31
170. Di Tullio MR, Sacco RL, Savoia MT, Sciacca RR, Homma S: Aortic atheroma morphology and the risk of ischemic stroke in a multiethnic population. Am Heart J 2000; 139:329–36
171. Velho FJ, Dotta F, Scherer L, Bartholomay E, Da Silva, DA Fernandes JG, Torres MA: Association between the effect of spontaneous contrast in the thoracic aorta and recent ischemic stroke determined by transesophageal echocardiography. Ar Qbras Cardiol 2004; 82:52–6
172. Kapetanakis EI, Stamou SC, Dullum MK, Hill PC, Haile E, Boyce SW, Bafi AS, Petro KR, Corso PJ: The impact of aortic manipulation on neurologic outcomes after coronary artery bypass surgery: A risk-adjusted study. Ann Thorac Surg 2004; 78:1564–71
173. Ura M, Sakata R, Nakayama Y, Miyamoto TA, Goto T: Ultrasound demonstration of manipulation related aortic injuries after cardiac surgery. J Am Coll Cardiol 2000; 35:1303–10
174. Stern A, Tunick PA, Culliford AT, Lachmann J, Baumann FG, Kanchuger MS, Marshall K, Shah A, Grossi E, Kronzon I: Protruding aortic arch atheroma: Risk of stroke during heart surgery with and without aortic arch endarterectomy. Am Heart J 1999; 138:746–52
175. Gold JP, Torres KE, Maldarelli W, Zhuravlev I, Condit D, Wasnick J: Improving outcomes in coronary surgery: The impact of echo-directed aortic cannulation and perioperative hemodynamic management in 500 patients. Ann Thorac Surg 2004; 78:1579–85
176. Hangler HB, Nagele G, Danzmayr M, Mueller L, Ruttmann E, Laufer G, Bonatti J: Modification of surgical technique for ascending aortic atherosclerosis: Impact on stroke reduction in coronary artery bypass grafting. J Thorac Cardiovasc Surg 2003; 126:391–400
177. Bolotin G, Domany Y, de Perini L, Frolkis I, Lev-Ran O, Nesher N, Uretzky G: Use of intraoperative epiaortic ultrasonography to delineate aortic atheroma. Chest 2005; 127:60–5
178. Rokkas CK, Kouchoukos NT: Surgical management of the severely atherosclerotic ascending aorta during cardiac operations. Semin Thorac Cardiovasc Surg 1998; 10:240–6
179. Sharony R, Bizekis CS, Kanchuger M, Galloway AC, Saunders PC, Applebaum R, Schwartz CF, Ribakove GH, Culliford AT, Baumann FG, Kronzon I, Colvin SB, Grossi EA: Off-pump coronary artery bypass grafting reduces mortality and stroke in patients with atheromatous aortas: A case control study. Circulation 2003; 108:15–20
180. Lev Ran O, Braunstein R, Sharony R, Kramer A, Paz Y, Mohr R, Uretzky G: No touch aorta off pump coronary surgery: The effect on stroke. J Thorac Cardiovasc Surg 2005; 129:307–13
181. Mishra M, Malhotra R, Karlekar A, Mishra Y, Trehan N: Propensity case-matched analysis of off-pump *versus* on-pump coronary artery bypass grafting in patients with atheromatous aorta. Ann Thorac Surg 2006; 82:608–14
182. Engelhardt M, Folkers W, Brenke C, Harders A, Fidorra H, Schmieder K: Neurosurgical operations with the patient in sitting position: Analysis of risk factors using transcranial Doppler sonography. Br J Anaesth 2006; 96:467–72
183. Domaingue CM: Neurosurgery in the sitting position: A case series. Anaesth Intensive Care 2005; 33:332–5
184. Tingleff J, Joyce FS, Pettersson G: Intraoperative echocardiographic study of air embolism during cardiac operations. Ann Thorac Surg 1995; 60:673–7
185. Chandraratna A, Ashmeg A, Pasha HC: Detection of intracoronary air embolism by echocardiography. J Am Soc Echocardiogr 2002; 15:1015–7
186. Orihashi K, Matsuura Y, Sueda T, Shikata H, Mitsui N, Sueshiro M: Pooled air in open heart operations examined by transesophageal echocardiography. Ann Thorac Surg 1996; 61:1377–80
187. Schoenburg M, Kraus B, Muehling A, Taborski U, Hoffmann H, Erhardt H, Hein S, Roth M, Vogt PR, Karliczek GF, Kloevekorn WP: The dynamic air bubble trap reduces cerebral microembolism during cardiopulmonary bypass. J Thorac Cardiovasc Surg 2003; 126:1455–60
188. Borger MA, Peniston CM, Weisel RD, Vasiliou M, Green RE, Feindel CM: Neuropsychologic impairment after coronary bypass surgery: Effect of gaseous microemboli during perfusionist interventions. J Thorac Cardiovasc Surg 2001; 121:743–9
189. Madu EC, D'Cruz IA, Wall BM, Mansour N, Shearin S: Transesophageal echocardiographic spectrum of calcific mitral abnormalities in patients with end stage renal disease. Echocardiography 2000; 17:29–35
190. Marcu CB, Ghantous AE, Prokop EK: Caseous calcification of the mitral valve ring. Heart Lung Circ 2006; 15:187–8
191. Barasch E, Gottdiener JS, Larsen EK, Chaves PH, Newman AB, Manolio TA: Clinical significance of calcification of the fibrous skeleton of the heart and aortosclerosis in community dwelling elderly: The Cardiovascular Heath Study (CHS). Am Heart J 2006; 151:39–47
192. Kim HK, Park SJ, Suh JW, Kim YJ, Kim HS, Sohn DW, Oh BH, Lee MM, Park YB, Choi YS: Association between cardiac valvular calcification and coronary artery in a low-risk population. Coron Artery Dis 2004; 15:1–6
193. Allison MA, Cheung P, Criqui MH, Langer RD, Wright CM: Mitral and aortic annular calcifications are highly associated with systemic calcified atherosclerosis. Circulation 2006; 113:861–6
194. Bittrick J, D'Cruz IA, Wall BM, Mansour N, Mangold T: Differences and similarities between patients with and without end-stage renal disease, with regard to location of intracardiac calcification. Echocardiography 2002; 19:1–6
195. Kizer JR, Wiebers DO, Whisnant JP, Galloway JM, Welty TK, Lee ET, Best LG, Resnick HE, Roman MJ, Devereux RB: Mitral annular calcification, aortic valve sclerosis, and incident stroke in adults free of clinical cardiovascular disease: The Strong Heart Study. Stroke 2005; 36:2533–7
196. Fox CS, Vasan RS, Parise H, Levy D, O'Donnell CJ, d'Agostino RB, Benjamin EJ: Framingham Heart Study: Mitral annular calcification predicts cardiovascular morbidity and mortality: The Framingham Heart Study. Circulation 2003; 107:1492–6
197. Willens HJ, Ferreira AC, Gallagher AJ, Morytko JA: Mobile components associated with rapidly developing mitral annulus calcification in patients with chronic renal failure: Review of mobile elements associated with mitral annulus calcification. Echocardiography 2003; 20:363–7
198. Eicher JC, Soto FX, deNadai L, Ressencourt O, Falcon-Eicher S, Giroud M, Louis P, Wolf JE: Possible association of thrombotic, nonbacterial vegetations of the mitral ring-mitral annular calcium and stroke. Am J Cardiol 1997; 79:1712–5
199. Eicher JC, DeNadal L, Soto FX, Falcon-Eicher S, Dobsak P, Zanetta G, Petit JM, Portier H, Louis P, Wolf E: Bacterial endocarditis complication mitral annular calcification: A clinical and echocardiographic study. J Heart Valve Dis 2004; 13:217–27
200. Harpaz D, Auerbach I, Vered Z, Motro M, Tobar A, Rosenblatt S: Caseous calcification of the mitral annulus: A neglected, unrecognized diagnosis. J Am Soc Echocardiogr 2001; 14:825–31
201. Ionescu AA, Newman GR, Butchart EG, Fraser AG: Morphologic analysis of a strand recovered from a prosthetic mitral valve: No evidence of fibrin. J Am Soc Echocardiogr 1999; 12:766–8
202. Homma S, Di Tullio, MR Sciacca RR, Sacco RL, Mohr JP: Effect of aspirin and warfarin therapy in stroke patients with valvular strands. Stroke 2004; 35:1436–42
203. Roldan CA, Shively BK, Crawford MH: Valve excrescences: Prevalence, evolution and risk for cardioembolism. J Am Coll Cardiol 1997; 30:1308–14
204. Dawn B, Hasnie AM, Calzada N, Longaker RA, Stoddard MF: Transesophageal echocardiography impacts management and evaluation of patients with stroke, transient ischemic attack, or peripheral embolism. Echocardiography 2006; 23:202–7