

**FATİH YALÇIN, MD***

Cardiovascular Imaging Section, Department of Cardiology, Cleveland Clinic

DAVID HOMA, BS

Cardiovascular Imaging Section, Department of Cardiology, Cleveland Clinic

JAMES D. THOMAS, MD*

Director, Cardiovascular Imaging Center, Department of Cardiology, Cleveland Clinic

FRANK A. FLACHSKAMPF, MD

Cardiovascular Imaging Section, Department of Cardiology, Cleveland Clinic

Transesophageal echocardiography: First-line imaging for aortic diseases

■ ABSTRACT

Transesophageal echocardiography (TEE) is now commonly used to evaluate the thoracic aorta, because it is widely available and provides high-resolution images and flow information by Doppler. This article reviews the essential features on TEE of acute and chronic aortic diseases, such as aortic dissection, aneurysm, and atherosclerosis, and discusses its strengths, weaknesses, and indications.

■ KEY POINTS

The advantage of TEE over computed tomography and magnetic resonance imaging is that it can be done quickly at the bedside in the emergency department, operating room, or intensive care unit.

The main disadvantage of TEE is that it cannot show the top of the aortic arch or the abdominal aorta.

Speed and accuracy are crucial in diagnosing acute aortic dissection, as the mortality rate is exceptionally high.

TRANSESOPHAGEAL ECHOCARDIOGRAPHY (TEE) has vastly improved our ability to diagnose diseases of the thoracic aorta such as aortic dissection and aneurysms. Twenty-five years ago, the only imaging studies for these potentially catastrophic diseases were chest roentgenography and angiography; today, they are ancillary, having given way to TEE, magnetic resonance imaging (MRI), and computed tomography (CT). Of these, TEE is often the first-line study, as it is widely available and relatively inexpensive.

This review discusses the most important aortic diseases and how they appear on TEE.

■ HOW WE USE TEE TO IMAGE THE AORTA

Because the esophagus runs parallel and close to the thoracic aorta, TEE gives high-resolution cross-section images (ie, short-axis views, using a horizontal imaging plane) of nearly the entire thoracic aorta,^{1,2} except for the upper part of the ascending aorta and the proximal part of the arch, which are obscured because the trachea and left bronchus lie between the aorta and esophagus in this area (FIGURE 1).³

To obtain a short-axis view of the ascending aorta, we gradually withdraw the TEE probe from the level of the aortic valve up to the level of the right and left pulmonary arteries.

A long-axis (ie, lengthwise) view of this area will reveal the aortic valve as well; to do this we rotate the imaging plane, to 120° to 150°.

Rotating the probe counterclockwise brings into view the descending aorta, which can be followed in short-axis and long-axis

*Supported in part by Grant NCC 9-60, National Aeronautics and Space Administration, Houston, TX.

TABLE 1

Normal and abnormal findings on transesophageal echocardiography of the aorta

AREA	NORMAL FINDINGS	ABNORMAL FINDINGS
Aortic lumen	Diameter < 4 cm Echo-free One lumen	Diameter 4–5 cm: dilatation Diameter > 5 cm: aneurysm (if localized), ectasia (if diffuse) Thrombus (in aneurysm or false lumen) Intimal flap and two lumina in dissection In dissection, on color Doppler the true lumen displays higher flow velocities than the false lumen; also, entry and reentry sites display localized higher velocity jets
Aortic wall	Homogeneous texture Ascending aorta ≤ 3 mm thick Descending aorta ≤ 5 mm thick	Thickening, calcification, ulcer with atherosclerosis Atheroma with or without attached mobile thrombus Intramural hematoma Rupture
Periaortic space	No fluid	Effusion, hematoma

views from the arch to the level of the diaphragm. By slowly withdrawing the probe and turning clockwise at the distal end of the arch, the inner curvature of the arch and sometimes the supraaortic arteries can also be seen.

TABLE 1 describes the key points of the TEE examination of the aorta.

■ AORTIC DISSECTION

Acute aortic dissection is dramatic and life-threatening. Weakened by degenerative changes in its smooth muscle and elastic tissue (usually owing to hypertension, atherosclerosis, or congenital abnormalities of collagen and elastin), the aortic media loses its hold on the intima. The intima abruptly begins to tear away from the media, and blood surges in, creating a false lumen. Under the pounding force of the heartbeat, blow by blow the advancing column of blood rips the intima away from the media down the length of the aorta. The pain is intense and the mortality rate is high if the condition is untreated: approximately 2% per hour in the first 48 hours if the ascending aorta is involved. Death is from rupture of the aorta into the pericardial or pleural space or from occlusion of major arteries. With emergency surgery to

stabilize the ascending aorta, mortality can be less than 20%.

Speed and accuracy are critical in diagnosing this disease. Moreover, the differential diagnosis includes several other high-risk conditions, such as myocardial infarction and pulmonary embolism, for which prompt treatment is also urgent.

Classification of aortic dissections

Aortic dissections are classified according to their location, ie, in the ascending aorta, descending aorta, or both. Two classification systems are used:

The DeBakey classification

- Type I—Originating in the ascending aorta and extending into the descending aorta
- Type II—Limited to the ascending aorta
- Type III—Limited to the descending aorta

The Stanford classification

- Type A—Involving the ascending aorta (and possibly the descending aorta)
- Type B—Limited to the descending aorta

TEE in diagnosing aortic dissection

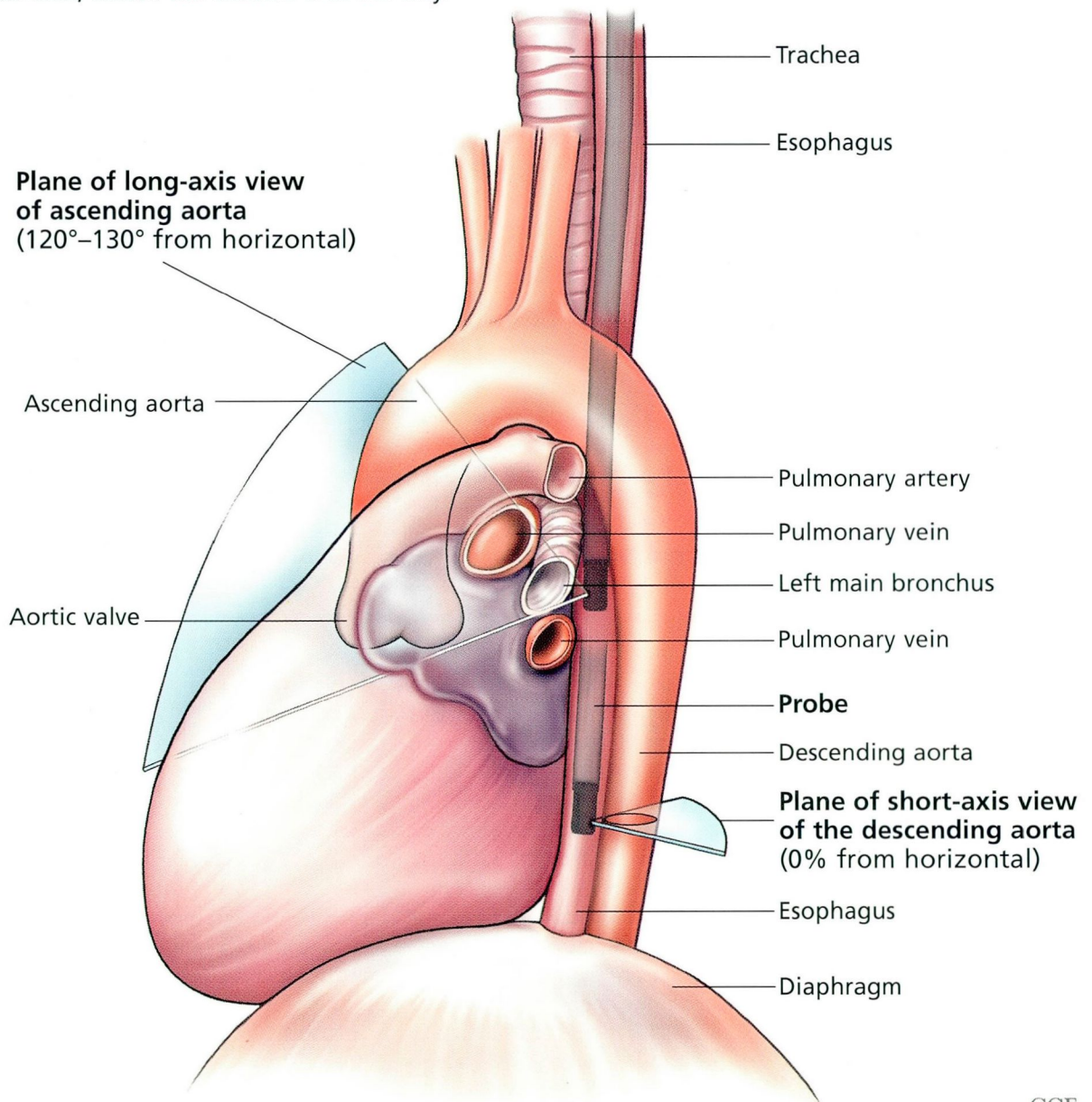
On TEE, the typical features of aortic dissection (FIGURES 2 AND 3) include a double lumen separated by a mobile membrane (ie, that can

In acute ascending aortic dissection, mortality is 2% per hour



■ Transesophageal echocardiography of the aorta

Using transesophageal echocardiography (TEE), one can obtain short-axis (cross-sectional) and long-axis (longitudinal) images of nearly the entire thoracic aorta, except for the top of the aortic arch, where the trachea is in the way.



CCF
©2000

FIGURE 1

be seen to move back and forth with the heartbeat), different flow patterns within the true and false lumens, and intimal tears (entry or reentry sites).⁴ TEE also provides information about the functional status of the aortic

valve, pericardial effusions, blood flow, and thrombus burden in the false lumen.

In a study of 199 patients with aortic disease,⁵ TEE was as accurate as CT in identifying the type and extent of thoracic aortic dis-

Acute dissection of the ascending aorta

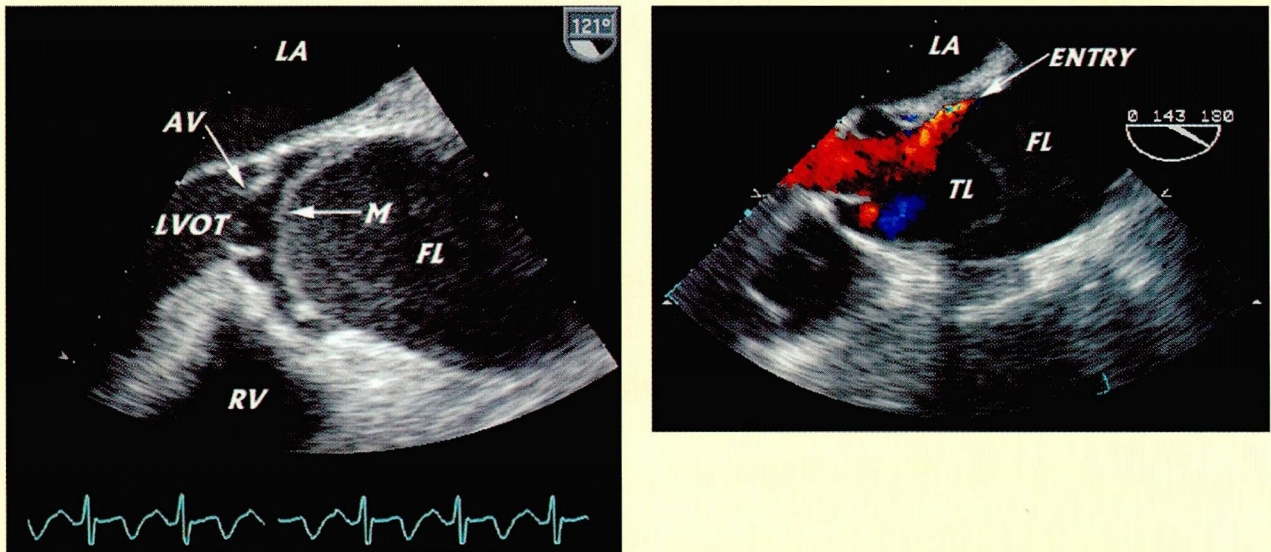


FIGURE 2. Acute dissection of the ascending aorta (DeBakey type I, Stanford type A), long-axis views. **Left**, the dissection membrane (M) is seen in the aortic root, and most of the ascending aorta is occupied by the false lumen (FL). The ascending aorta is dilated (diameter, 5 cm). AV—aortic valve, LA—left atrium, LVOT—left ventricular outflow tract, RV—right ventricle. **Right**, color Doppler flow mapping in a different patient during systole. Note the open aortic valve. TL—true lumen.

section. Nienaber et al⁶ reported that TEE had a sensitivity of 97.7% and a specificity of 76.9%, while Erbel et al⁷ reported that the sensitivity of echocardiography, including TEE, was 99% and the specificity was 98%. The higher specificity in the latter study probably reflects increasing familiarity with the technique and its pitfalls.

TEE is also suitable, safe, and accurate for serial follow-up of patients with aortic dissection.^{8,9} However, MRI or CT are necessary if the findings on TEE are ambiguous, and to evaluate the supraaortic vessels (in particular the carotid arteries) and the abdominal aorta.

Thrombus formation

In aortic dissection, a thrombus often forms in the false lumen. TEE can detect a thrombus by visualizing it directly (it often has a crescent shape),¹⁰ or indirectly by showing lack of flow in the false lumen^{11,12} or central displacement of intimal calcification.^{13,14}

Thrombosis is more common in chronic aortic dissections than in the acute phase, and

sometimes the false lumen can completely fill with thrombus.¹⁰ In the acute phase, thrombosis appears to occur more often in dissections in the descending aorta (ie, DeBakey type III or Stanford type B) than in those in the ascending aorta (ie, DeBakey type I or II or Stanford type A).^{10,13,15}

A problem is that thrombosis also can occur in aneurysms and in intramural hematomas, complicating the diagnosis of aortic dissection by TEE.

Thrombosis of a false lumen must be distinguished from mural thrombosis of an aortic aneurysm because the conditions carry different prognoses and require different surgical techniques.¹⁶

In addition, approximately 10% of patients with typical aortic dissection have segments with purely intramural hematomas.^{17–20} Aortic intramural hematoma without an intimal tear was first described almost 80 years ago in autopsy studies.²¹ This picture of extensive segments of dissected aorta without entry and without detectable flow has been termed “noncommu-



Acute dissection of the descending aorta

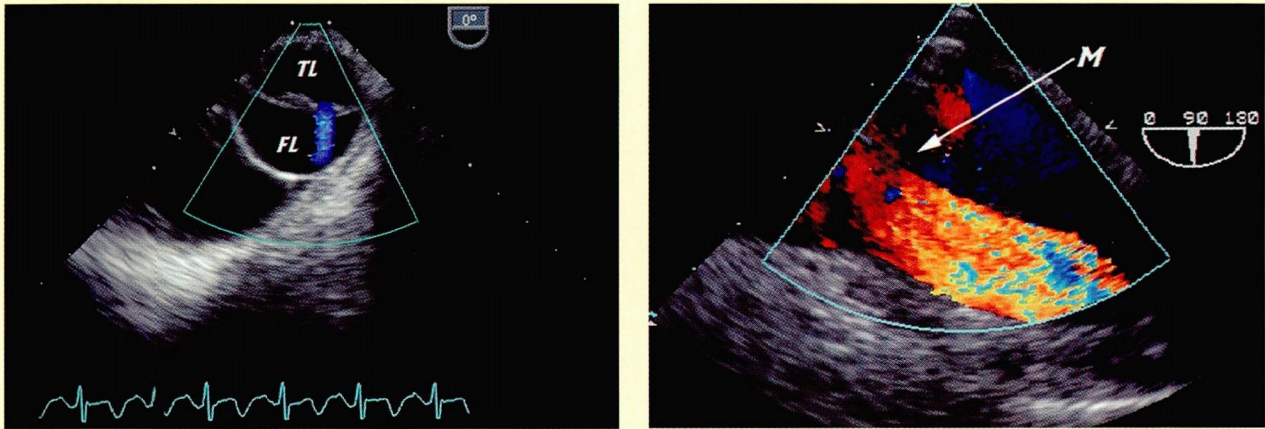


FIGURE 3. Dissection of the descending aorta. **Left**, short-axis view. The dissection membrane is seen, separating the true lumen (TL) on top from the false lumen (FL) at the bottom. Flow across an entry site (in blue, indicating flow from top to bottom) is seen on color Doppler flow mapping. **Right**, long-axis view in a different patient. The true lumen is at the bottom and the false lumen is on top, separated by the dissection membrane (M). On color Doppler, higher flow velocities are seen in the true lumen than in the false lumen, which shows almost stagnant blood.

nicating dissection,” and it implies a better prognosis than chronically persisting flow in the false lumen (“communicating dissection”).^{22–24} Possible mechanisms of pathogenesis include hemorrhage from rupture of the vasa vasorum in the aortic wall, atherosclerotic plaque rupture followed by bleeding into an atheroma,²¹ or penetrating aortic wall ulcer.^{17–21,25} It is frequently associated with pericardial or pleural effusion.

On TEE, an extensive intramural hematoma can be impossible to differentiate from complete thrombosis of the false lumen. Both appear as crescentic or circular wall thickening without a primary intimal tear, sometimes with an intramural echo-free space and no or slow flow by color Doppler.

■ AORTIC ANEURYSM

An aneurysm can occur in any segment of the aorta. Predisposing factors include atherosclerosis, hypertension, aortic valve disease (for the aortic root), and Marfan syndrome.

TEE not only detects aneurysms but also measures their luminal diameters (FIGURE 4), which helps determine the treatment.

Elective surgery is recommended for aortic aneurysms 5 cm or larger in diameter regardless of location. If the diameter is between 4 cm and 5 cm, close follow-up (usually in half-yearly intervals) is advised. Surgery should be considered even for diameters less than 5 cm if the aneurysm increases in size, causes symptoms by displacing other structures, or causes localized pain.

As noted above, a thrombus frequently forms in aortic aneurysms, because a larger aortic diameter leads to lower shear rates and predisposes to blood stasis.

On TEE, a sign of blood stasis is spontaneous echo contrast,²⁶ thought to be caused by rouleaux formation of red blood cells,²⁷ which occurs when shear rate is reduced.²⁸ As noted above, TEE is also highly accurate in identifying thrombus.

A thrombus should be observed carefully even after it covers the inner wall of the aneurysm completely. Findings indicating that the thrombus is being destroyed by exfoliation and tearing of the surface should be considered a signal of imminent rupture of the aneurysm. Another potential risk of aneurysms is systemic embolism of fresh thrombi.

Consider surgery if an aortic aneurysm is ≥ 5 cm

Aortic aneurysms with thrombus

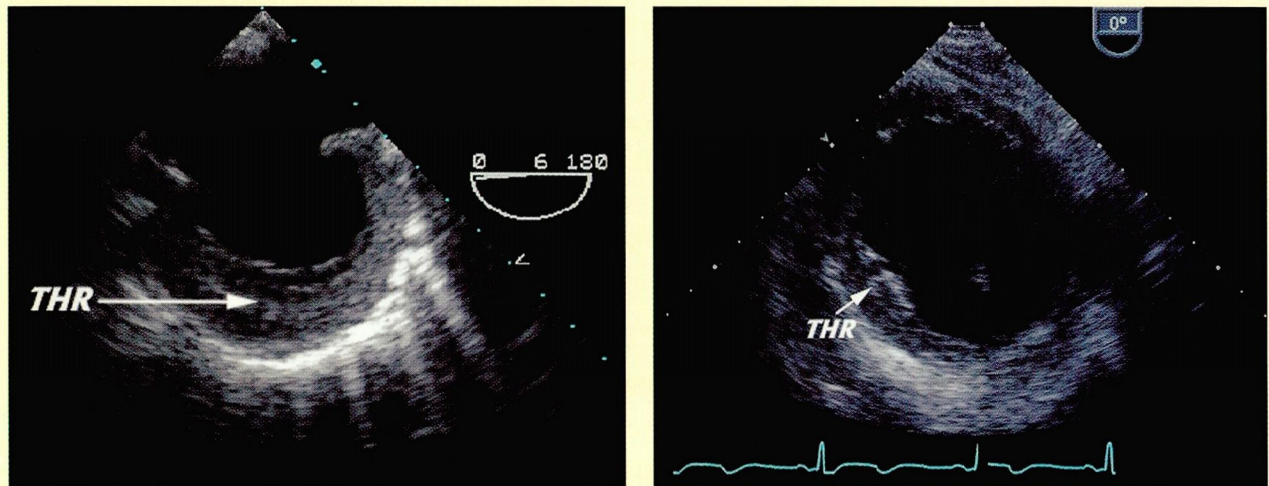


FIGURE 4. Two cases of aneurysm with thrombus (THR) in the descending aorta, in short-axis views. The maximal diameters are 7.5 cm (left) and 6.5 cm (right).

**We now know
that aortic
atheromas
often embolize**

■ AORTIC ATHEROSCLEROSIS

Severe atherosclerosis of the descending aorta is common, found on 7% to 17% of routine TEE studies,²⁹ more often in the descending than in the ascending aorta.

During the last decade we have begun to recognize that this condition poses a risk of cardiovascular mortality and vascular events due to systemic embolism.^{29–32} Therefore, if a patient suffers a systemic embolism, one should evaluate the aorta for mobile aortic atheromas by TEE as part of the search for the source.

On TEE, atherosclerosis is first evident by wall thickening greater than 5 mm. At the lumen surface, it can produce well-defined protruding atheromas, which sometimes have portions that are mobile, ie, that move back and forth during systole and diastole (FIGURE 5).

Semi-detached material such as this in the aorta, termed “debris,” can consist of either thrombi or complex elements of ulcerated plaque. In mobile lesions treated with warfarin, serial TEE examinations showed that the lesions decreased in size, indirectly supporting their mostly thrombotic nature. On the other hand, severe, long-standing atherosclerosis can lead to large plaque ulcerations, in particular in the descending aorta.

■ MARFAN SYNDROME

Marfan syndrome, an autosomal-dominant disorder of connective tissue with variable expression, can cause a number of possible ocular, skeletal, and cardiovascular abnormalities.

Although several mutations that affect the formation of collagen or elastin have been described in this disease, the diagnosis is still best made on the basis of clinical criteria: ocular manifestations, characteristic skeletal deformities, cardiovascular manifestations, and family history.^{33–35}

Cardiovascular manifestations of Marfan syndrome

The cardiovascular problems in Marfan syndrome result from disruption and loss of elastic fibers in the media and an increase in mucopolysaccharides. The main manifestations are:

- **Aortic root dilatation** with the potential for dissection or rupture of the thoracic aorta. From 5% to 10% of patients with aortic dissection have Marfan syndrome.
- **Cardiac failure** due to progressive aortic regurgitation. Dilatation of the aortic root is observed in 70% to 85% of patients with Marfan syndrome, with concomitant aortic



Atherosclerosis of the descending aorta

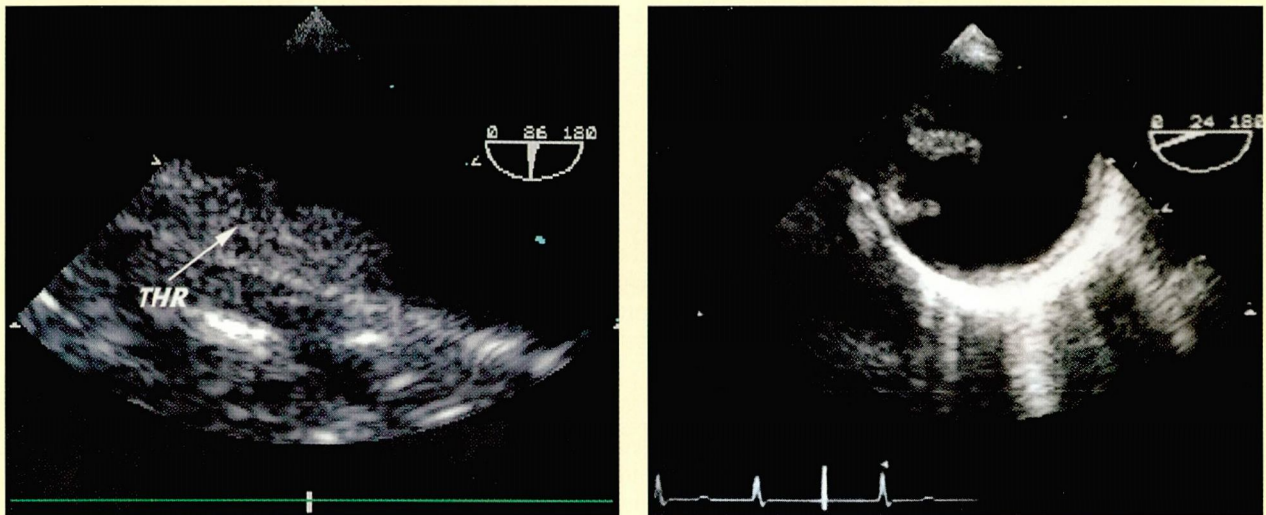


FIGURE 5. Two cases of atherosclerosis of the descending aorta. **Left,** long-axis view of severe atherosclerosis with a thrombus (THR) that is sessile (ie, fixed). **Right,** short-axis view of the descending aorta with mild circumferential atherosclerosis and a thrombus attached to the wall between 9 and 11 o'clock that is mobile (ie, semi-detached, moving with systole and diastole).

regurgitation in 20% to 23% of cases.³⁶

The morphologic basis of aortic insufficiency in Marfan syndrome is different from that of degenerative annuloaortic ectasia, the most common cause of aortic insufficiency and ascending aortic aneurysm. In degenerative annuloaortic ectasia, each aortic leaflet is cylindrical and is attached to the aortic sinus along a crescentic line. The length of the base of the aortic leaflet at its insertion line is approximately 50% longer than the length of its free margins. In some of these patients, surgical repair of the aortic valve is feasible as long as the leaflets are not grossly distorted.³⁷

In Marfan syndrome, in contrast, the aortic root becomes nonelastic and the sinuses of Valsalva are dilated and thin. This dilatation of the aortic root pulls the commissures of the aortic valve outward, causing lack of central coaptation of the leaflets with consequent aortic insufficiency. In these patients, the lengths of the free margins of the leaflets approximate the lengths of their bases. The leaflets become overstretched and thinned, and display histologic changes similar to those seen in the arterial wall. The sinotubular junctions become effaced. The subaortic fibrous tissue is often

involved in this degenerative process and also becomes dilated.

- Myxomatous mitral valve changes and prolapse with mitral regurgitation.

TEE in Marfan syndrome

TEE provides rapid diagnostic information in patients with Marfan syndrome with suspected aortic dissection and enhances the assessment of other cardiovascular manifestations of this condition.^{37,38} The two most important factors bearing on the patient's risk for aortic dissection are the maximal aortic root dimension and a family history of aortic dissection. Bicuspid aortic valve, also readily diagnosed by TEE, can be associated with dissection of the aortic aneurysm in Marfan syndrome.

■ AORTIC TRAUMA

Rapid deceleration, such as in a car accident, can cause traumatic aortic rupture, most often in the descending aorta immediately distal to the takeoff of the left subclavian artery. The hallmarks of traumatic rupture are laceration of the wall and periaortic hematoma.

TEE has been reported to be sensitive and

5% to 10% of patients with aortic dissection have Marfan syndrome

TABLE 2

Advantages and disadvantages of transesophageal echocardiography (TEE), computed tomography (CT), and magnetic resonance imaging (MRI) in diagnosing aortic disease

TEST	ADVANTAGES	DISADVANTAGES
TEE	Rapid (5–10 minutes) Widely available Can be done at bedside (maximal treatment possible at the same time, including surgery) Provides simultaneous anatomic and functional cardiac evaluation Relatively inexpensive	Has a “blind spot” in the ascending aorta Does not show abdominal aorta Operator-dependent Uncomfortable for patient Patient may need sedation
CT or MRI	Evaluates entire aorta Little operator or reader dependency Higher accuracy in some indications (coarctation, intramural hematoma) No or very little patient discomfort	Approximately 20-minute time to diagnosis Not universally available Limited accessibility of patient during exam Needs contrast agent Depending on hardware, resolution may be inferior to that of TEE Costly Limited simultaneous cardiac exam

Only TEE can diagnose aortic dissection within minutes in the ER

specific enough to be used as the primary imaging method in patients with suspected traumatic rupture of the thoracic aorta, comparing favorably with arch aortography. One study in 160 consecutive patients suspected of having blunt thoracic aortic injury found TEE to be 100% sensitive and specific.^{39–41}

■ AORTIC COARCTATION

Coarctation (localized narrowing) of the aorta most often occurs immediately distal to the left subclavian artery takeoff and at the level of the attachment of the ductus arteriosus. It is often associated with a bicuspid aortic valve. Rarer forms may involve longer segments of the thoracic aorta and are associated with complex congenital heart disease.

Although TEE can detect the narrowing, the morphology is often complex (especially after repair or valvuloplasty), and more definitive evaluation often requires CT or MRI. Moreover, to minimize the angle between the ultrasound beam and the blood flow, Doppler assessment of the gradient is best performed transthoracically, from the suprasternal window.

■ TEE IN OTHER DISEASES

TEE has also been used to diagnose other conditions accompanying aortic dissection and aneurysm, including bicuspid aortic valve,³⁶ quadricuspid aortic valve,⁴² rupture of the aortic wall due to an interventricular septal hydatid cyst,⁴³ and others. In addition, it can detect aortic root abscess and other complications of infective endocarditis,⁴⁴ the increased wall thickness of giant cell and Takayasu arteritis (which are due to inflammatory processes and are sometimes called “pulseless disease”), and other abnormalities.

■ WHEN IS TEE APPROPRIATE?

In diagnosing aortic diseases, the main alternatives to TEE are CT (including spiral CT) and MRI. TABLE 2 summarizes the relative advantages and disadvantages of these methods. MRI and CT both provide reliable, excellent images and can show the abdominal aorta and the segments of the thoracic aorta that are difficult or impossible to visualize by TEE.

Hence, the critical issues in the choice are not diagnostic accuracy but availability, speed,



and cost. Only TEE can provide a firm diagnosis of aortic dissection within minutes at the bedside in the intensive care unit, emergency room, or operating room. It is therefore the approach of choice in acutely ill, unstable patients, such as in aortic dissection or aortic trauma.

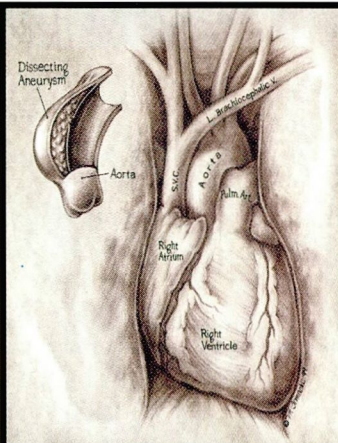
On the other hand, TEE has some limitations, such as in detecting intramural hematoma⁴⁵ or delineating the exact mor-

phology of aortic coarctation. It is operator-dependent and occasionally has problems with image quality. Therefore, in cases of doubt or ambiguity, one would be wise to obtain a CT or MRI image. Furthermore, in the elective setting, such as the evaluation of a chronic aortic aneurysm or following-up a chronic dissection, speed and availability are less important, and the advantages of CT or MRI outlined in TABLE 2 may outweigh those of TEE. ■

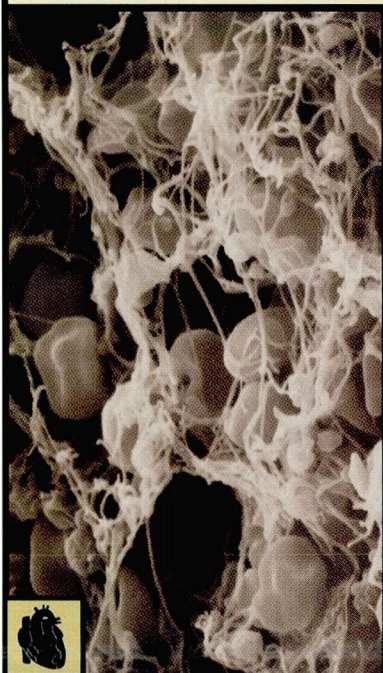
■ REFERENCES

1. Hanrath P, Kremer P, Langenstein BA, Matsumoto M, Bleifeld W. Transösophageale Echokardiographie. Ein neues Verfahren zur dynamischen Ventrikelfunktionsanalyse. *Dtsch Med Wochenschr* 1981; 156:523–525.
2. Borner N, Erbel R, Braun B, Henkel B, Meyer J, Rumpelt J. Diagnosis of aortic dissection by transesophageal echocardiography. *Am J Cardiol* 1984; 54:1157–1158.
3. Erbel R. Role of transesophageal echocardiography in dissection of the aorta and evaluation of degenerative aortic disease. *Cardiology Clinics* 1993; 11:461–473.
4. Nienaber CA, von Kodolitsch Y, Nicolas V. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. *N Engl J Med* 1993; 328:1–9.
5. Wiet SP, Pearce WH, McCarthy WJ, Joob AW, Yao JS, McPherson DD. Utility of transesophageal echocardiography in the diagnosis of disease of the thoracic aorta. *J Vasc Surg* 1994; 20:613–620.
6. Nienaber CA, Spielmann RP, Kodolitsch YV, et al. Diagnosis of thoracic aortic dissection: magnetic resonance imaging versus transesophageal echocardiography. *Circulation* 1992; 85:434–447.
7. 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.
8. Masani ND, Banning AP, Jones RA, Ruttley MS, Fraser AG. Follow-up of chronic thoracic aortic dissection: comparison of transesophageal echocardiography and magnetic resonance imaging. *Am Heart J* 1996; 131:1156–1163.
9. Maffei S, Baroni M, Terrazzi M, et al. Ambulatory follow-up of aortic dissection: comparison between computed tomography and biplane transesophageal echocardiography. *Intern J Cardiac Imag* 1996; 12:105–111.
10. Nishino M, Tanouchi J, Tanaka K, et al. Transesophageal echocardiographic diagnosis of thoracic aortic dissection with the completely thrombosed false lumen: Differentiation from true aortic aneurysm with mural thrombus. *J Am Soc Echocardiogr* 1996; 9:79–85.
11. Mohr-Kahaly S, Erbel R, Renollet H, et al. Ambulatory follow-up of aortic dissection by transesophageal, two-dimensional and color-coded Doppler echocardiography. *Circulation* 1989; 80:24–33.
12. Hoshimo T, Ohmae M, Sakai A. Spontaneous resolution of a dissection of the descending aorta after medical treatment with an alpha-blocker and a calcium antagonist. *Br Heart J* 1987; 58:82–84.
13. Erbel R, Engberding G, Daniel W, Roelandt J, Visser C, Renollet H. Echocardiography in diagnosis of aortic dissection. *Lancet* 1989; 1:457–461.
14. Erbel R, Mohr-Kahaly S, Oelert H, Renollet A, Thelen M, Meyer J. Diagnostic strategies in suspected aortic dissection: comparison of computed tomography, aortography, and transesophageal echocardiography. *Am J Cardiac Imaging* 1990; 4:157–172.
15. Yamaguchi T, Naito H, Ohta M, et al. False lumen in type III aortic dissections: progress CT study. *Radiology* 1985; 156:757–760.
16. Gandjbakhch I, Jault F, Vaissier E, et al. Surgical treatment of chronic aortic dissections. *Eur J Cardiothorac Surg* 1990; 4:466–471.
17. Hirst AE, Johns VJ, Kime SW. Dissecting aneurysms of the aorta: a review of 505 cases. *Medicine (Baltimore)* 1958; 37:217–219.
18. Gore S. Pathogenesis of dissecting aneurysm of the aorta. *Arch Pathol* 1952; 53:142–153.
19. Stanson AW, Welch TJ, Ehman RL, Sheedy SS. A variant of aortic dissection: computer tomography and magnetic resonance findings. *Cardiovasc Imaging* 1989; 1:55–59.
20. Mohr-Kahaly S, Erbel R, Kearney P, Puth M, Meyer J. Aortic intramural hemorrhage visualized by transesophageal echocardiography: findings and prognostic implications. *J Am Coll Cardiol* 1994; 23:658–664.
21. Krukenberg E. Beiträge zur Frage des Aneurysma dissecans. *Beitr Pathol Anat Allg Pathol* 1920; 67:329–351.
22. Stanson AW, Welch TJ, Ehman RL, Sheedy PF 2nd. A variant of aortic dissection: computer tomography and magnetic resonance findings. *Cardiovasc Imaging* 1989; 1:55–59.
23. Yamada T, Tada S, Harada J. Aortic dissection without intimal rupture: diagnosis with MR imaging and CT. *Radiology* 1988; 168:347–352.
24. Dinsmore RE, Willerson JT, Buckley MJ. Dissecting aneurysm of the aorta: angiographic features affecting prognosis. *Radiology* 1972; 105:567–572.
25. Hirst AE, Johns VJ, Kime SW. Dissecting aneurysm of the aorta: a review of 505 cases. *Medicine (Baltimore)* 1958; 37:217–279.
26. Finkelhor R, Lamont W, Ramanavarapu SK, Bahler R. Spontaneous echocardiographic contrast in the thoracic aorta: Factors associated with its occurrence and its association with embolic events. *Am Heart J* 1995; 130:1254–1258.
27. Sigel B, Coelho JCU, Schade SG, Justin J, Spigos DG. Effect of plasma proteins and temperature on echogenicity of blood. *Invest Radiol* 1982; 17:29–33.
28. Beppu S, Nimura Y, Sakakibara H. Smoke-like echo in the left atrial cavity in mitral valve disease: its fea-

THE CLEVELAND
CLINIC
FOUNDATION
DEPARTMENT of
CARDIOLOGY
PRESENTS



An Intensive Review of
CARDIOLOGY



A COMPREHENSIVE

5½-DAY SYMPOSIUM

...

SEPTEMBER 24 - 29,

2000

MARRIOTT KEY

CENTER HOTEL,

CLEVELAND, OH



**Co-Directors: Eric Topol, MD, Brian Griffin, MD,
and Curtis Rimmerman, MD**

For more information, please contact the
Cleveland Clinic Center for Continuing
Education at

1-800-762-8173 or 216-444-5695

or visit our website:

www.clevelandclinicmeded.com

YALÇIN AND COLLEAGUES



- tures and significance. *J Am Coll Cardiol* 1985; 6:744-749.
29. Karalis DG, Chandrasekaran K, Victor M, Ross JJ Jr, Mintz GS. Recognition and embolic potential of intraaortic atherosclerotic debris. *J Am Coll Cardiol* 1991; 17:73-78.
 30. Tunick PA, Perez JL, Kronzon I. Protruding atheromas in the thoracic aorta and systemic embolization. *Ann Intern Med* 1991; 115:423-427.
 31. Dressler FA, Craig WR, Castello R, Labovitz AJ. Mobile aortic atheroma and systemic efficacy of anticoagulation and influence of plaque morphology on recurrent stroke. *J Am Coll Cardiol* 1998; 31:134-138.
 32. Amarenco P, Duyckaerts C, Tzourio C, Henin D, Bousser MG, Hauw JJ. The prevalence of ulcerated plaques in the aortic arch in patients with stroke. *N Engl J Med* 1992; 326:221-225.
 33. Pyeritz RE, MacKusick VA. The Marfan syndrome: diagnosis and management. *N Engl J Med* 1979; 300:772-777.
 34. Moodie DS. Diagnosing Marfan syndrome is still based on clinical characteristics. *Cleve Clin J Med* 1998; 65:176-181.
 35. Simpson IA, de Belder MA, Treasure T. Cardiovascular manifestation of Marfan's syndrome: improved evaluation by transesophageal echocardiography. *Br Heart J* 1993; 69:104-108.
 36. Epperlein S, Mohr-Kahaly S, Erbel R, Kearney P, Meyer J. Aorta and aortic valve morphologies predisposing to aortic dissection. An in vivo assessment with transesophageal echocardiography. *Eur Heart J* 1994; 15:1520-1527.
 37. Davies MJ. *Pathology of Cardiac Valves*. Toronto: Butterworth Publishers, 1980:37-61.
 38. David TE. Aortic valve repair in patients with Marfan syndrome and ascending aorta aneurysms due to degenerative disease. *J Card Surg* 1994; 9:182-187.
 39. Smith MD, Cassidy JM, Souther S, et al. Transesophageal echocardiography in the diagnosis of traumatic rupture of the aorta. *N Engl J Med* 1995; 332:356-362.
 40. Buckmaster MJ, Keaney PA, Johnson SB, Smith MD, Sapin PM. Further experience with transesophageal echocardiography in the evaluation of thoracic aortic injury. *J Trauma* 1994; 37:989-995.
 41. Brathwaite CE, Cilly JM, O'Connor WH, Ross SE, Weiss RL. The pivotal role of transesophageal echocardiography in the management of traumatic thoracic aortic rupture with associated intra-abdominal haemorrhage. *Chest* 1994; 105:1899-1901.
 42. Erena C, Breithardt OA, Franke A, Flachskampf FA. Quadricuspid aortic valve: diagnosis by multiple transesophageal echocardiography. *Z Kardiol* 1996; 85:889-892.
 43. Sabah I, Yalçin F, Okay T. Rupture of a presumed hydatid cyst of the interventricular septum diagnosed by transesophageal echocardiography. *Heart* 1998; 79:420-421.
 44. Leung DY, Ranney GB, Hopkins AP, Walsh WF. Role of transesophageal echocardiography in the diagnosis and management of aortic root abscess. *Br Heart J* 1994; 72:175-181.
 45. Flachskampf FA, Banbury M, Smedira N, Thomas JD, Garcia, M. TEE diagnosis of intramural hematoma of the ascending aorta: a word of caution. *J Am Soc Echocardiogr* 1999; 12:866-870.

ADDRESS: James D. Thomas, MD, Department of Cardiology, Desk F15, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, OH 44195; e-mail thomasj@ccf.org.