Chapter 10
Thoracic Aorta

Timothy M. Maus, MD, FASE

Abstract The close proximity of the esophagus to the thoracic aorta provides an excellent imaging opportunity. Transesophageal echocardiography (TEE) serves an important role in several important pathologies including aortic dissection, aortic aneurysms, aortic atheromatous disease, and aortic trauma. A thorough understanding of the use of TEE in evaluating the thoracic aorta including normal and pathologic presentations is essential to the basic perioperative echocardiographer. However, knowledge of the limitations of the modality is also key to appropriate patient management and preventing mismanagement.

Keywords Thoracic aorta · Aortic dissection · Aortic aneurysm · Atheromatous disease · Plaque · Blunt aortic trauma

Introduction

Basic perioperative transesophageal echocardiography (TEE) guidelines suggest that knowledge of echocardiographic manifestations of lesions of the great vessels is a necessary training objective. Therefore, a thorough understanding of the use of TEE in evaluating the thoracic aorta including normal and pathologic presentations is essential to the basic perioperative echocardiographer. This chapter will review the essential views for evaluating the thoracic aorta, including potential pitfalls and

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review the major aortic pathologies of aortic dissection, aortic aneurysms, aortic atheromatous disease, and thoracic aortic trauma.

TEE: Aortic Views

The immediate proximity of the esophagus to the thoracic aorta allows excellent imaging and superior subsequent detection of disease states. The aorta is a three-layered structure, with intimal, medial, and adventitial layers, that extends from the heart immediately beyond the aortic valve through the transverse aortic arch and descends towards the lower extremity vessel branches. It is described as having five anatomical sections (Fig. 10.1):

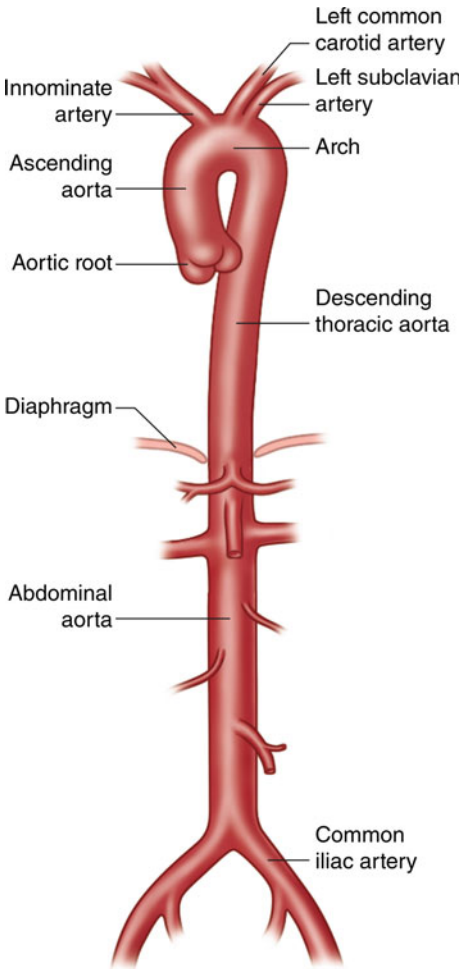


Fig. 10.1 Diagram of the entire aortic vascular system include its five anatomical components: *1* aortic root; *2* tubular ascending aorta; *3* aortic arch; *4* descending thoracic aorta; *5* abdominal thoracic aorta

1. Aortic root which extends from the aortic valve to the sinotubular junction (which connects the root to the ascending tubular aorta). The aortic root contains three sinuses of Valsalva, two of which contain the left and right coronary artery ostia.
2. Tubular ascending aorta which extends from the sinotubular junction to the aortic arch.
3. Aortic arch which is the transverse portion of the aorta with typically three great vessel branches: innominate artery, left common carotid, and left subclavian artery.
4. Descending thoracic aorta which extends from the left subclavian artery takeoff to the diaphragm.
5. Abdominal thoracic aorta which continues from the diaphragm to the lower extremity branch points.

As described above, the proximity of the esophagus to the aorta provides excellent views, however, limitations do exist. Because the trachea and left main bronchus are interposed between the esophagus and aorta creating significant air-related artifact, imaging of the distal ascending aorta and proximal arch is often challenging or impossible (Fig. 10.2). Alternative echocardiographic approaches to imaging this region include transthoracic echocardiography (TTE), epiaortic imaging intraoperatively with an opened chest or the use of saline-filled balloons placed in the trachea. However, each of these techniques is beyond the scope of this text.

The relationship of the esophagus to the aorta changes from cranial to caudal. The esophagus is posterior to the aorta at the cranial aspect near the arch, however, the structures twist on themselves such that the esophagus is anterior to the aorta at the gastroesophageal junction. This makes description and location of pathology difficult because the echocardiographic imaging demonstrates a normally circular shape throughout the entire descending thoracic aorta (Fig. 10.3). The anterior, posterior or lateral nature of a lesion thus cannot be accurately identified. Lastly, when entering the stomach, the probe enters the intraperitoneal space, while the aorta continues into the retroperitoneal space. Therefore, the probe is no longer opposing tissue near the aorta and thus, the abdominal aorta is not consistently imaged.

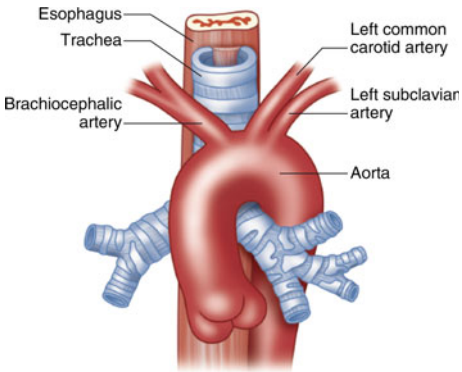


Fig. 10.2 Diagram of the distal ascending aorta, aortic arch, and proximal descending thoracic aorta including its anterior relationship to the esophagus with the trachea interposed

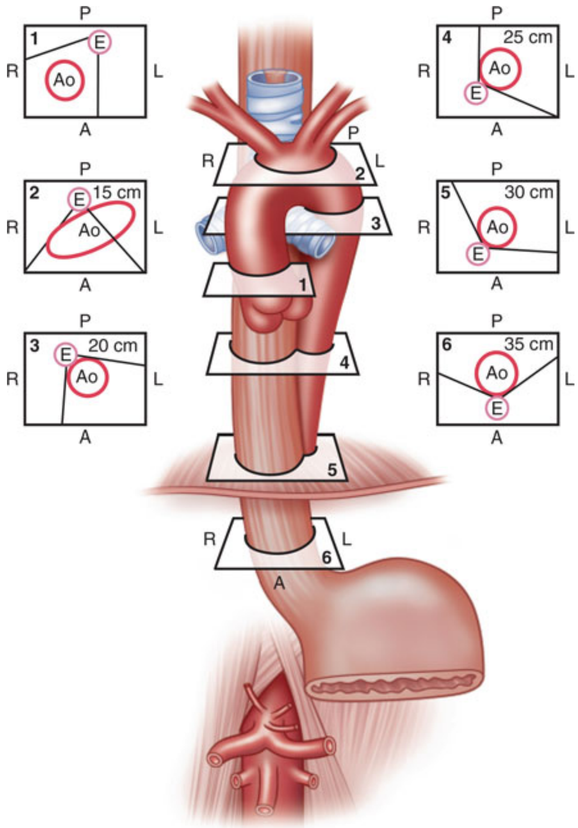
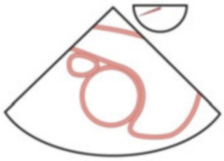
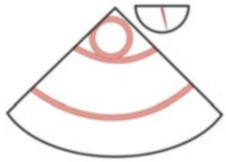
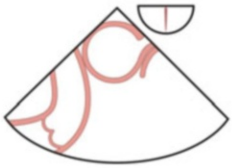
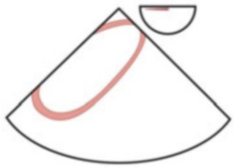
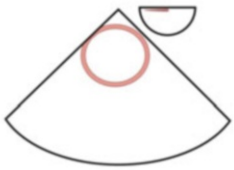
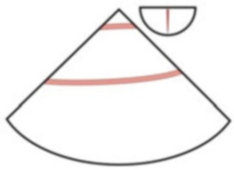


Fig. 10.3 Diagram of the thoracic aorta in relation to the esophagus. Note when cephalad the esophagus is positioned posterior to the aorta, however, when moving caudally the esophagus moves to an anterior position relative to the descending thoracic aorta. (*E*, esophagus)

The TEE views of the aorta can largely be separated into a short and long axis view of each segment of the visible thoracic aorta (Table 10.1). When discussing the thoracic aorta, one must consider the aortic valve as disease of the aorta may affect the aortic valve and vice versa. The short and long axis views of the aortic valve provide an opportunity to evaluate the number, quality, and function of leaflets, to make appropriate measurements of the left ventricular outflow tract (LVOT), aortic valve annulus, and sinotubular junction, as well as to identify pathology such as aortic dissection or aneurysmal disease. The addition of color flow Doppler aids in the detection of valvular dysfunction. Withdrawing and advancing the probe at zero and ninety degrees of multiplane allows the short and long axis examination, respectively, of the tubular ascending aorta. Obtaining the aortic arch views is easily obtained by rotating the echocardiography probe to the left until the circle shaped descending thoracic aorta is identified and subsequently withdrawing the probe until the oval shaped long axis of the aortic arch is obtained. Increasing the multiplane angle to ninety degrees develops the short axis of the aortic arch. Of note, most commonly the left subclavian artery takeoff is identified on the right side of the screen with the innominate vein noted distally. The left subclavian artery takeoff is

Table 10.1 Transesophageal aortic views

Structure	Short axis	Long axis
Aortic valve	 ME AV SAX	 ME AV LAX
Ascending aorta	 ME asc aortic SAX	 ME asc aortic LAX
Aortic arch	 UE aortic arch SAX	 UE aortic arch LAX
Descending aorta	 Desc aortic SAX	 Desc aortic LAX

an important structure for location identification in the descending aorta. As described above, determining location of pathology in the descending aorta is difficult. Therefore, most commonly the distance from the left subclavian artery takeoff to the pathology is used to communicate location. This technique is also utilized to properly place an intra-aortic balloon pump (IABP) within 1–2 cm distal to the left subclavian. Advancing the probe in both a zero and ninety degree multiplane develops the short and long axis views of the descending thoracic aorta, respectively.

Aortic Dissection

Aortic Dissection

2D	• Large undulating mobile flap within aortic lumen • Ensure flap not due to imaging artifact • Differentiate true versus false lumen • SEC or Thrombus in false lumen • Presence of Pericardial or Pleural effusion • Coronary Artery Involvement (Wall Motion Abnormality)
CFD	• Differential flow patterns (true lumen = laminar; false lumen = sluggish) • Presence of Aortic Insufficiency
Spectral	• Evaluation of Aortic Insufficiency

Aortic dissection is typified by bleeding within the medial layer of the aorta, most commonly due to intimal tearing and separation. Propagation of bleeding within this layer separates the intima from the surrounding adventitial layer, yielding the classically identified dual lumen appearance. The true lumen contains blood within the natural aortic lumen while the false lumen is created by the force of blood ejecting through the intimal tear into the medial layer and contained by the adventitial layer.

This pathology carries a significant risk of morbidity and mortality with a 1–2 % increased mortality per hour until definitive treatment [1]. Therefore, prompt diagnosis is of the utmost concern. Patients at risk for developing an aortic dissection include those with long standing hypertension, smoking, and connective tissue disorders [2]. Common associated diseases include Marfan's syndrome, Ehlers–Danlos syndrome, bicuspid aortic valve, and coarctation of the aorta.

Two classification systems exist for classifying aortic dissections, DeBakey and Stanford [1]. The Stanford system nicely separates involvement of the ascending aorta into Stanford A, which is a surgical emergency, while Stanford B involves the descending thoracic aorta below the left subclavian artery takeoff. Stanford B dissections without complicating ischemia (paralysis, mesenteric ischemia, etc.) are often treated medically. The DeBakey system divides aortic dissections into three types: Type 1 involves the ascending and descending aorta, Type 2 involves the ascending aorta only, while Type 3 involves only the descending aorta below the left subclavian artery takeoff.

As mentioned above, prompt diagnosis is of paramount importance in such patients to reduce morbidity and mortality. The historical gold standard involves angiography and the demonstration of contrast exiting the true lumen into the false lumen. The time consuming process and increased risk of contrast-induced nephropathy has reduced this practice, being replaced by Helical CT and MRI. The downside of each of these modalities is the continued need for patient transport as well as being potentially time consuming. TEE provides a mobile modality with excellent sensitivity and specificity in the detection of aortic dissections. Shiga et al.

demonstrated that TEE has a sensitivity of 98 % and specificity of 95 % and is comparable to helical CT or MRI [3]. Therefore, patients who are hemodynamically unstable and unable to be transported to an imaging suite may be evaluated by TEE in the emergency room, intensive care unit or operating room.

The echocardiographic approach to an aortic dissection involves confirming the presence and the location of a dissection flap as well as potentially identifying intimal tear entry/exit sites, differentiating true versus false lumen, and identifying complicating pathologies such as aortic insufficiency, pericardial and pleural effusions, coronary artery involvement and ventricular dysfunction. Of note, the current basic perioperative TEE consensus statement suggests that in the setting of complex pathology such as an aortic dissection, appropriate consultation with an advanced echocardiographer or other imaging modality is indicated [4].

Dissection Flap Identification

Identification of the intimal flap is the cornerstone of the aortic dissection diagnosis. Typically the intimal flap is noted as a thin, undulating mobile structure that is entirely contained within the lumen of the aorta. Multiple multiplane angles should be utilized to ensure that there is a flap present, representing the separation of the true from false lumens (Figs. 10.4 and 10.5; Videos 10.1 and 10.2). With the cardiac cycle, systolic ejection into the aortic lumen causes true lumen expansion while delayed entry of blood through the intimal tear into the false lumen causes delayed filling with sluggish or no flow. This cycling of expansion leads to the

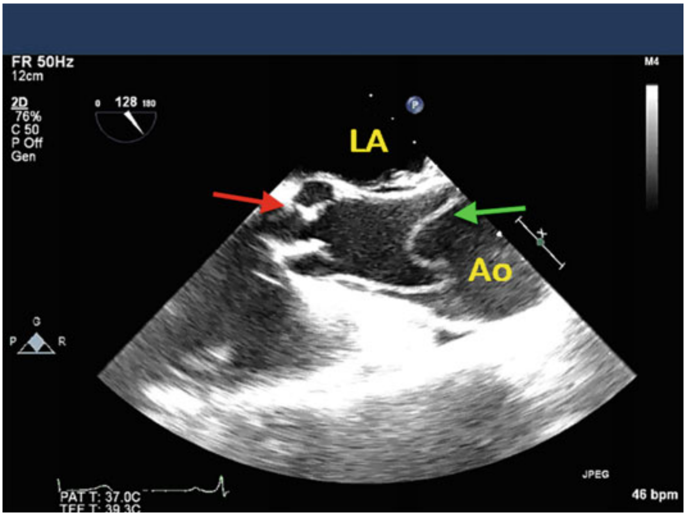


Fig. 10.4 Midesophageal aortic valve long axis view with the probe slightly withdrawn to demonstrate more of the tubular ascending aorta. The *red arrow* points to a calcified aortic valve while a *green arrow* indicates the undulating intimal flap within the aortic lumen (*LA* left atrium; *Ao* Aorta)

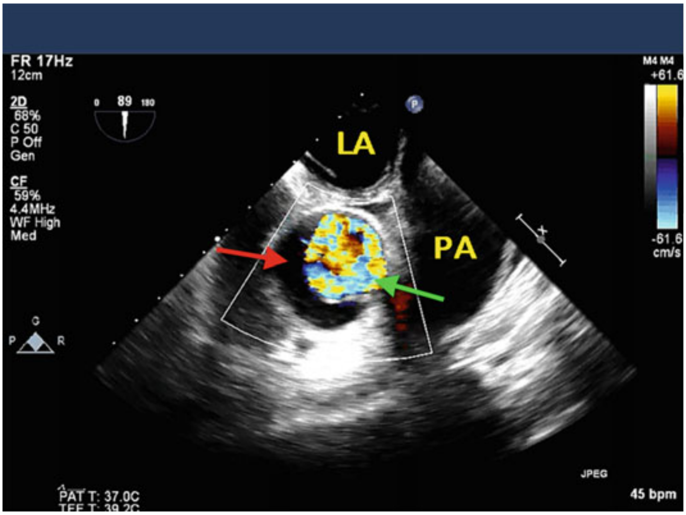


Fig. 10.5 Midesophageal view of ascending aorta developed by a slow withdrawal of the probe from an aortic valve short axis. The *green arrow* points to a true lumen with noted rapid early systolic flow while the *red arrow* indicates the false lumen with little to no flow demonstrated on color flow Doppler (*LA* left atrium; *PA* main pulmonary artery)

undulating mobile appearance of the intimal flap. Discussed below, the differential flow patterns may be detected with color flow Doppler to aid in identifying true versus false lumens. In addition to identifying the presence of the intimal flap, describing the location and extent of the dissection is important. Location within the ascending aorta or aortic arch denotes a surgical emergency. Dissection through adjacent structures or branches such as the coronary arteries, great vessels, or aortic valves may also necessitate prompt surgical repair.

Echocardiography may also identify not just the presence of an intimal flap but also the intimal tear site. The tear displays as an opening or communication in the intimal flap with color flow Doppler documenting the presence of flow from the true to the false lumen (Fig. 10.6; Video 10.3). When a tear site is identified in the ascending aorta near the aortic valve, flow may be bidirectional between the true and false lumens because of pressure differentials near the aortic valve during systole and diastole. During systolic ejection, flow and pressure is higher in the true lumen with flow from true to false lumen through the tear site. During diastole, particularly with associated aortic insufficiency, the pressure is temporarily higher in the false lumen with a return of flow to the true lumen (Fig. 10.7; Video 10.4).

Care must be taken when diagnosing intimal flaps as imaging artifacts which, particularly in the ascending aorta, may appear as linear densities within the aortic lumen. Side lobe artifacts involve weak ultrasound beams emanating off axis from the main imaging plane and returning from strong reflectors to the ultrasound probe. A not infrequent occurrence is a side lobe reflection from an off-plane central line or pulmonary artery catheter displayed as a linear echogenic density in the ascending aorta

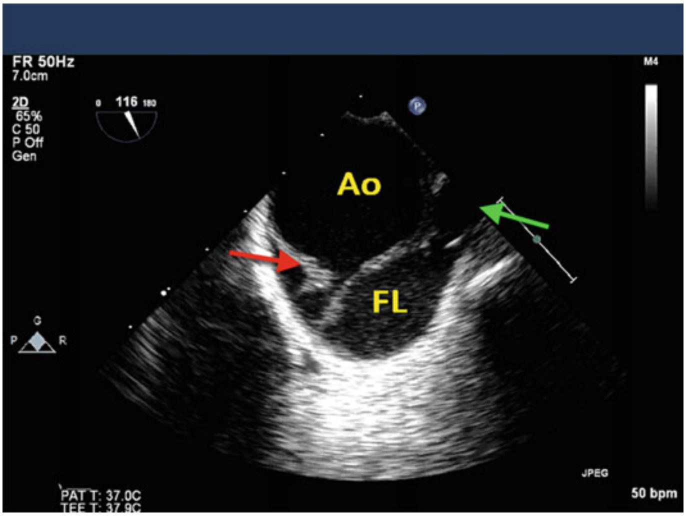


Fig. 10.6 Upper esophageal aortic arch short axis view in a patient with an acute aortic arch dissection. The *red arrow* points to the tear site in the intimal flap. The *green arrow* indicates the left subclavian artery takeoff which branches from the false lumen and has compromised flow (*Ao* Aorta; *FL* False Lumen)

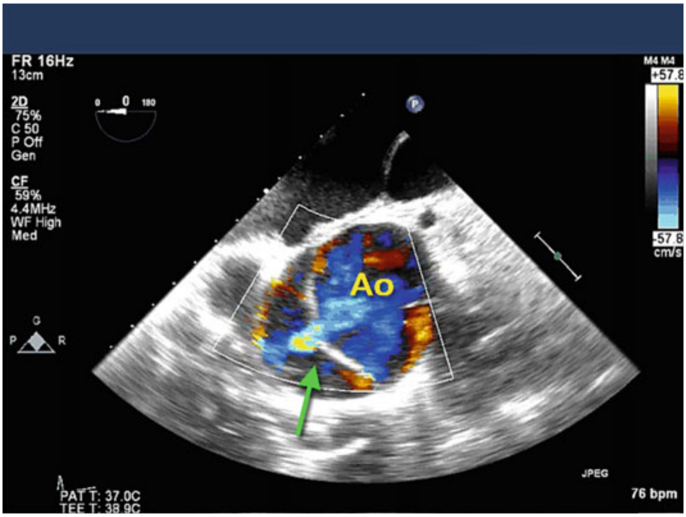


Fig. 10.7 Midesophageal ascending aortic short axis view in a patient with an ascending aortic dissection. The *green arrow* indicates the tear site in the intimal flap with systolic flow demonstrated from the true lumen into the false lumen (*Ao* ascending aorta)

in the midesophageal ascending aortic short axis view (Fig. 10.8; Video 10.5a, b). Again it is emphasized that except in emergency situations, current consensus statement suggests that a basic echocardiographer consult an advanced echocardiographer when evaluating aortic dissections to confirm the diagnosis.

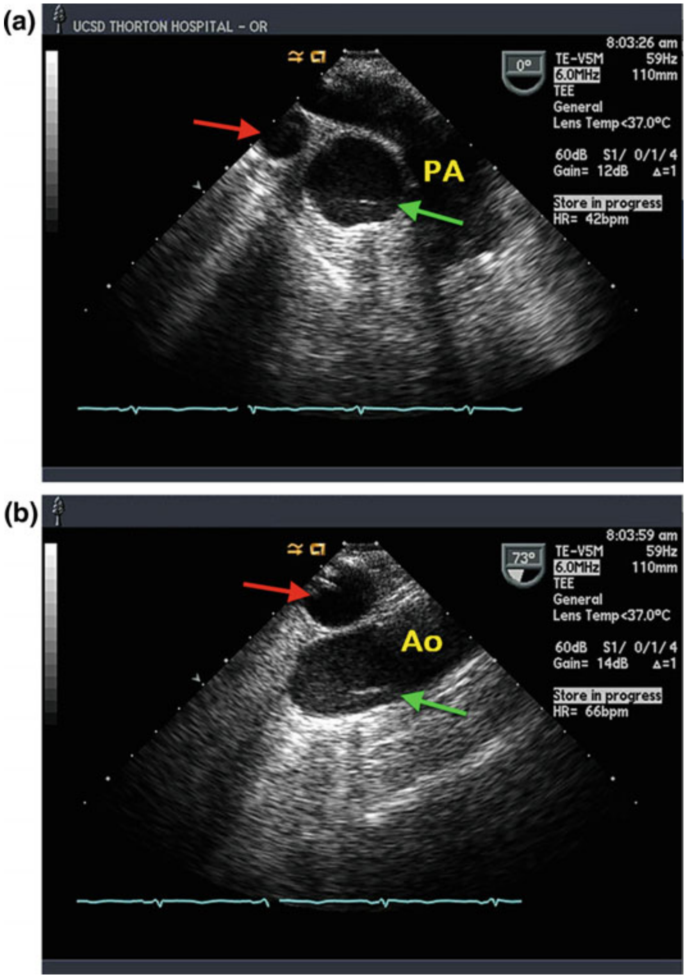


Fig. 10.8 **a** Midesophageal ascending aortic short axis view in a patient with a side lobe artifact. The *red arrow* indicates the superior vena cava. The *green arrow* indicates a side lobe artifact from a large out of plane specular reflector such as a central line or pulmonary artery catheter. This linear density in the ascending aorta may be confused with an aortic dissection. **b** Midesophageal ascending aortic long axis view of the same patient. The *red arrow* points to the right PA which contains a PA catheter. The *green arrow* points to a side lobe artifact which may be confused for an aortic dissection (*Ao* Ascending Aorta; *PA* pulmonary artery)

Differentiating True from False Lumens

The ability to differentiate true from false lumens serves to confirm the presence of an aortic dissection, rule out imaging artifacts, and in the case of aortic surgery, allow the confirmation of surgical repair (lack of false lumen flow post repair). There are several characteristics of true and false lumens that are fairly common: size, shape, systolic motion, and type or presence of flow (Table 10.2).

Table 10.2 Echocardiographic differentiation of aortic dissection true and false lumens

True lumen	False lumen
Smaller	Larger
Round	Irregular
Systolic expansion	Systolic compression
Early laminar flow	Late turbulent flow
	+/− Spontaneous contrast
	+/− Thrombus

On two-dimensional echocardiography, the true lumen is often the smaller and round shaped lumen while the false lumen tends to be the larger, irregular shaped structure. The larger false lumen is often crescentic ("moon-like") with concavity towards the true lumen. Systolic motion, as described above, involves the expansion of the true lumen with systolic ejection. Since the false lumen has delayed flow, the structure will compress during systole (Fig. 10.9; Video 10.6a, b). M-mode echocardiography may aid in identifying which structure is expanding during the systolic portion of the cardiac cycle. Lastly, color flow Doppler may be useful in differentiating where true lumens have early systolic laminar flow while false lumens have late systolic turbulent flow. False lumens contain such sluggish flow that spontaneous echo contrast or frank thrombus may be identified.

Identifying Complicating Pathologies

The proximity of the aorta to several other anatomical structures as well as the dependency of the branch vessels on an intact aorta allow an aortic dissection to wreak havoc beyond just the damaged vessel itself. An advantage of TEE over other modalities includes its ability to evaluate surrounding structures and the effect of a dissection on those structures. As described above, the aortic valve is intimately connected to the aorta such that an aortic dissection may yield significant aortic valve dysfunction (Fig. 10.10; Video 10.7a, b). There are several mechanisms by which an ascending dissection may cause aortic insufficiency, such as the mobile flap itself impeding valve closure or the large false lumen causing annular dilation or distortion and subsequent malcoaptation. A detailed evaluation of the mechanism is important in determining the need for concomitant valve replacement during aortic surgery, however this analysis is beyond the scope of this text.

In a normal state, the three layers of the aortic wall (intima, media, adventitia) contain the blood within the aortic lumen. During an aortic dissection, blood in the false lumen is now only contained by the adventitial layer, allowing a transudative process to leak into the surrounding spaces, such as the pericardial or left pleural space. An inflammatory component also appears to play a role in the development of pleural effusions [5]. Pericardial effusions are noted as an echolucent area surrounding the heart or great vessels in nearly any view but commonly the midesophageal four

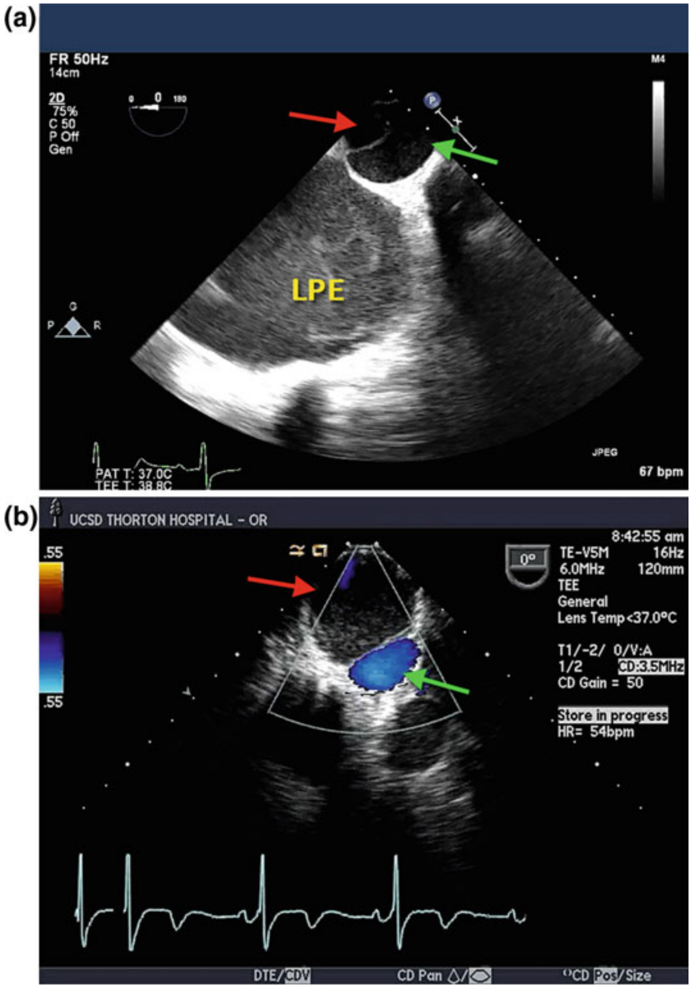


Fig. 10.9 **a** Descending thoracic aortic short axis view in a patient with an aortic dissection. The *red arrow* indicates the true lumen. The *green arrow* indicates the false lumen which contains spontaneous echo contrast. Note the large left sided pleural effusion (LPE). **b** Descending thoracic aortic short axis view with color flow Doppler in a separate patient with an aortic dissection. The *green arrow* indicates laminar flow in the true lumen and the *red arrow* indicates the false lumen with sluggish flow

chamber or transgastric short axis views. Determining tamponade physiology is discussed in the Rescue Echo chapter (See Chap. 12). Left sided pleural effusions are noted as an echolucent area anterior to the descending thoracic aorta in the descending aortic short axis view (Fig. 10.11; Video 10.8). Another process can yield pericardial or pleural effusions, however, with often more dramatic clinical presentations. Frank rupture of the dissection into the pericardial or pleural space causes a hemorrhagic effusion, rapid accumulation of blood, and significant hemodynamic deterioration.

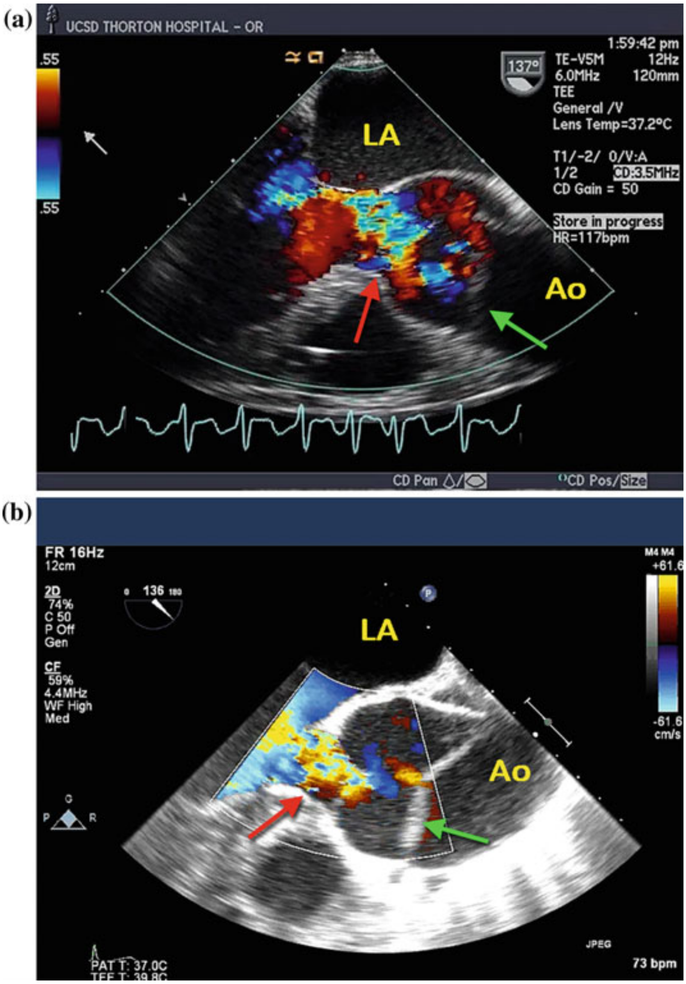


Fig. 10.10 **a** Midesophageal aortic valve long axis view in a patient with an ascending aortic dissection. The *green arrow* indicates the intimal flap located near the sinotubular junction while the *red arrow* indicates associated severe aortic insufficiency. **b** Midesophageal aortic valve long axis view in a separate patient with an ascending aortic dissection. The *green arrow* indicates the intimal flap located near the sinotubular junction. Note the intimal tear with diastolic flow reversing back into the true lumen from the false lumen. The *red arrow* indicates associated severe aortic insufficiency (*LA* left atrium; *Ao* Ascending Aorta)

During evaluation of an ascending aortic dissection that is approaching the aortic root, concern must exist for dissection through the ostium of the main coronary arteries. The aortic root contains three sinuses of Valsalva, two of which contain coronary arteries (left and right). A proximal dissection through a coronary ostium and resultant reduction of its blood supply may result in significant ischemia or frank infarction. An evaluation for wall motion abnormalities is essential if coronary involvement is suspected (Fig. 10.12; Video 10.9a, b).

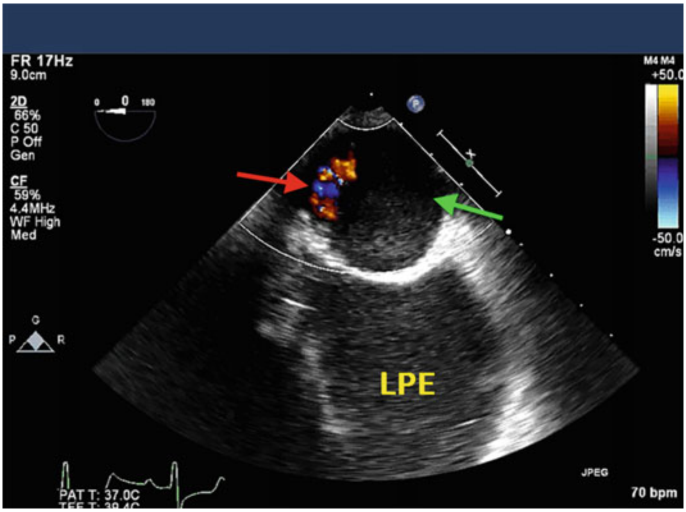


Fig. 10.11 Descending thoracic aortic short axis view with color flow Doppler in a patient with an aortic dissection and a large left pleural effusion (LPE). The *red arrow* indicates the true lumen; the *green arrow* notes the false lumen

Lastly, aortic dissections may result in ventricular dysfunction through two major mechanisms. As previously described, acute ischemia may result in wall motion abnormalities and frank RV or LV dysfunction. In another fashion, acute aortic insufficiency causes abrupt volume overload to a ventricle that has not had the time to dilate and adapt to the volume overload (as in chronic aortic insufficiency). Therefore, an evaluation of biventricular function is imperative in the setting of ascending aortic dissections.

Aortic Aneurysms

Aortic Aneurysm

2D	• Evidence of aortic dilation (linear measurements) • Branch involvement • Aortic Root Involvement (Aortic Valve malcoaptation)
CFD	• Presence of Aortic Insufficiency
Spectral	• Evaluation of Aortic Insufficiency

Aortic dilation refers to the enlargement of the aortic vessel beyond the upper limits of normal. Normal adult thoracic aortic are approximately 3.5 to 4.0 cm for the aortic root, less than 3.0 cm for the ascending aorta and descending thoracic aorta.

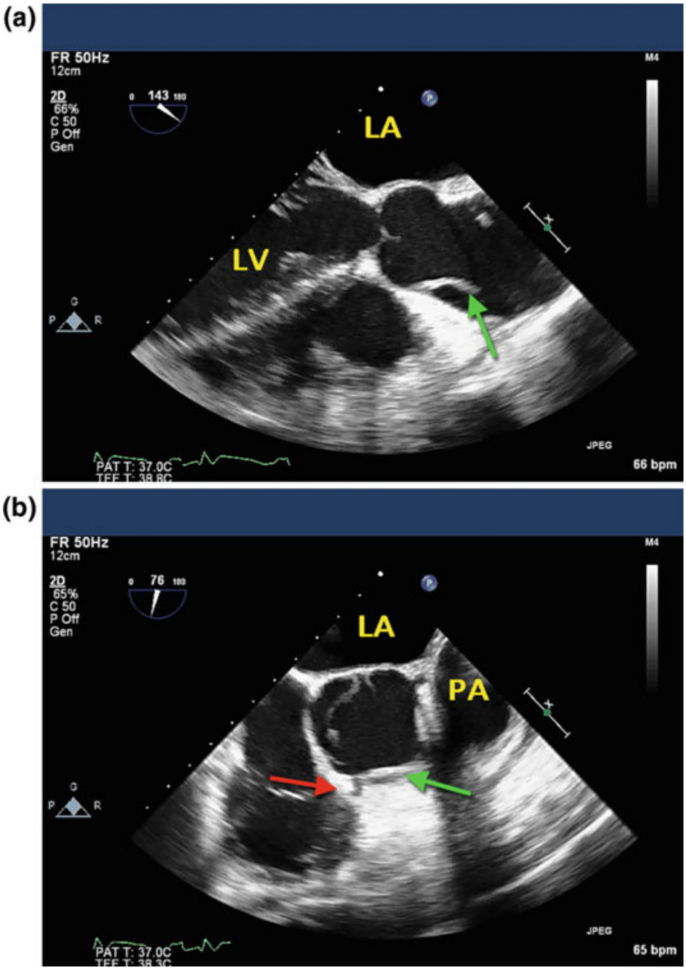


Fig. 10.12 **a** Midesophageal aortic valve long axis view in a patient with an ascending aortic dissection. The *green arrow* indicates the intimal flap located near the sinotubular junction and extending into the right sinus of Valsalva. **b** Midesophageal aortic valve short axis view in the same patient. The *green arrow* indicates the intimal flap located abutted the right coronary ostium (indicated by the *red arrow*) (*LA* left atrium; *LV* left ventricle; *PA* pulmonary artery)

Additionally, there is variability between genders at each segment (Root, Ascending, Mid-descending and Diaphragmatic) [6]. An aneurysm is classically described as a dilated segment of all three layers of an arterial wall with a vessel size that is beyond 150 % of its normal size. Surgical repair is considered as the aorta dilates beyond 4.5–5.5 cm taking into consideration the patients history and risk factors [6]. Again, the close relationship of the esophagus to the aorta allows excellent imaging of dilated and aneurysmal segments of the aorta. However, TEE does not carry the same potency in diagnosis as it does in the setting of acute aortic dissection. In the setting of aortic aneurysms, proper surgical planning is essential to successful treatment and relies on preoperative imaging. Proper identification of tortuosity, anterior spinal arteries including the artery of Adamkiewicz, and branch vessels may help guide management of cardiopulmonary bypass and neuroprotection strategies. TEE, however, still plays a role in the intraoperative management and in the setting of an unstable patient with an aneurysm rupture.

The echocardiographic approach to a patient with an aneurysm is similar to that of a dissection and includes determining the location and extent of disease as well as identifying coexisting pathologies. Measurements in multiple planes may be helpful to identify the degree and extent of the aneurysm. In the setting of ascending aortic aneurysms, measurement of the aortic valve annulus and sinotubular junction may aid in assessing both the aneurysm and potential aortic valve involvement (Fig. 10.13). Care must be undertaken as proper cross-sectional measurements may be difficult in the setting of tortuosity.

Coexisting pathology with aortic aneurysms most often relate to the aorta's intimate structural relationship to the aortic valve. In the setting of aortic stenosis, the resultant post-stenotic turbulent flow in the ascending aorta leads to altered hemodynamics and a continued outward pressure. This results in post-stenotic aortic dilatation that may halt after aortic valve replacement in calcific aortic stenosis [7]. Bicuspid aortic valve disease may also progress towards aortic stenosis with attendant aortic dilatation. However, despite aortic valve replacement, aortic dilatation may continue in these patients. In addition, another group of bicuspid aortic valve patients may present with annular dilation and aortic insufficiency without stenosis, potentially necessitating replacement of both the valve and

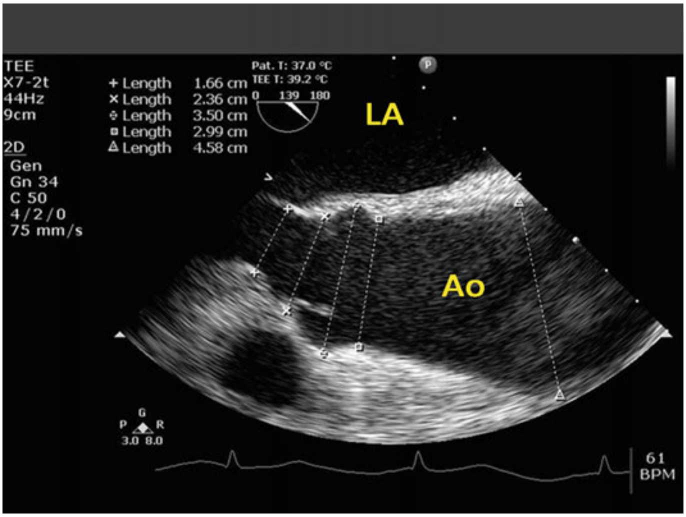


Fig. 10.13 Midesophageal aortic valve long axis view in a patient with an ascending aortic aneurysm. Measurements of the left ventricular outflow tract, aortic annulus, sinuses of Valsalva, sinotubular junction, and ascending aorta note the presence of an ascending aortic aneurysm without significant involvement of the aortic root (*LA* left atrium; *Ao* Ascending Aorta)

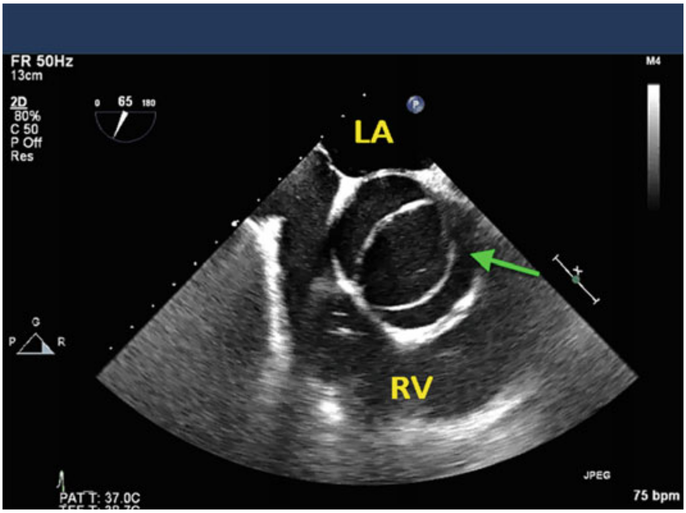


Fig. 10.14 Midesophageal aortic valve short axis view in a patient with a bicuspid valve and ascending aortic aneurysm. The *green arrow* indicates the bicuspid valve with a unified right and left coronary cusp. Note the dilated annulus (*LA* left atrium; *RV* right ventricle)

ascending aorta (Fig. 10.14; Video 10.10). Finally, the dilated aorta itself may have an impact on aortic valve function. As the aortic valve is crown shaped with attachments near the annulus at the base and the sinotubular junction at the top, dilation of the root may result in malcoaptation of the aortic valve leaflets and subsequent aortic regurgitation (Fig. 10.15; Video 10.11).

Aortic Atheroma

TEE is very sensitive to the detection of aortic atheromatous disease and the presence of such plaque carries significant patient risk. When the atheromatous disease is noted to be greater than or equal to 4 mm in thickness, it is associated with increased risk of all vascular events including stroke, myocardial infarction, peripheral embolism and death [1]. The transesophageal echocardiographic imaging approach includes noting severity, location, as well as mobility of atheromas (Figs. 10.16 and 10.17; Video 10.12a, b). The grading of atheromatous disease is displayed in Table 10.3. In the setting of interventional vascular procedures, the presence of severe atheromatous disease including plaque mobility should be communicated to the surgical team to prevent inadvertent embolization.

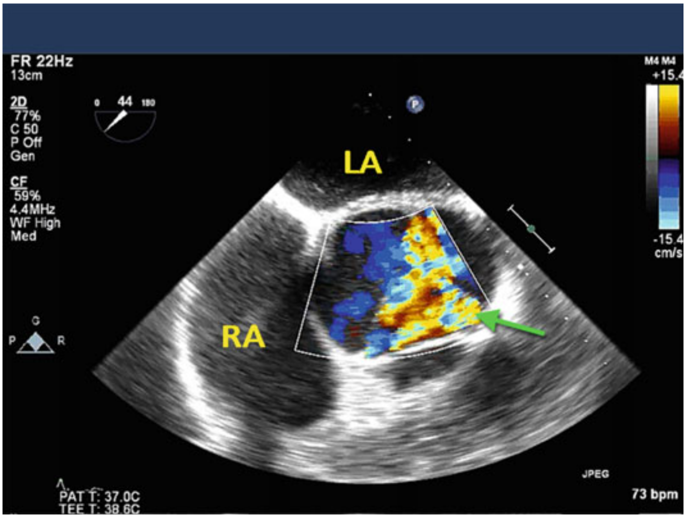


Fig. 10.15 Midesophageal aortic valve short axis view with color flow Doppler in a patient with an ascending aortic aneurysm. The *green arrow* indicates the significant aortic insufficiency from the associated dilated annulus and ascending aortic aneurysm (*LA* left atrium; *RA* right atrium)

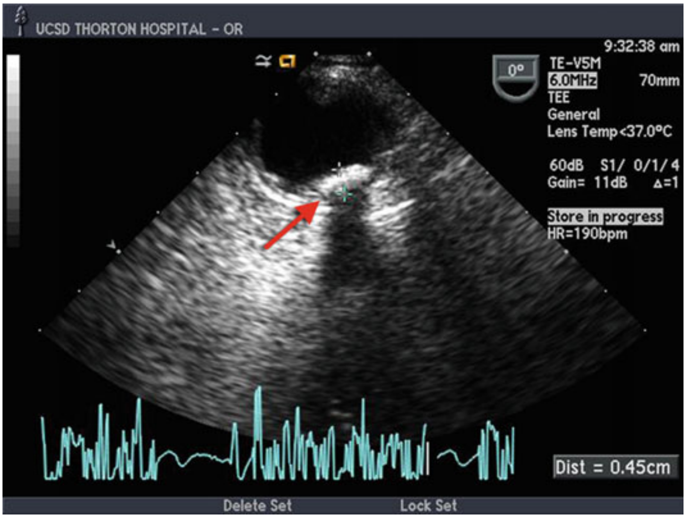


Fig. 10.16 Descending thoracic aorta short axis view demonstrating aortic atheroma measuring 4.5 cm, Grade III disease (*red arrow*)

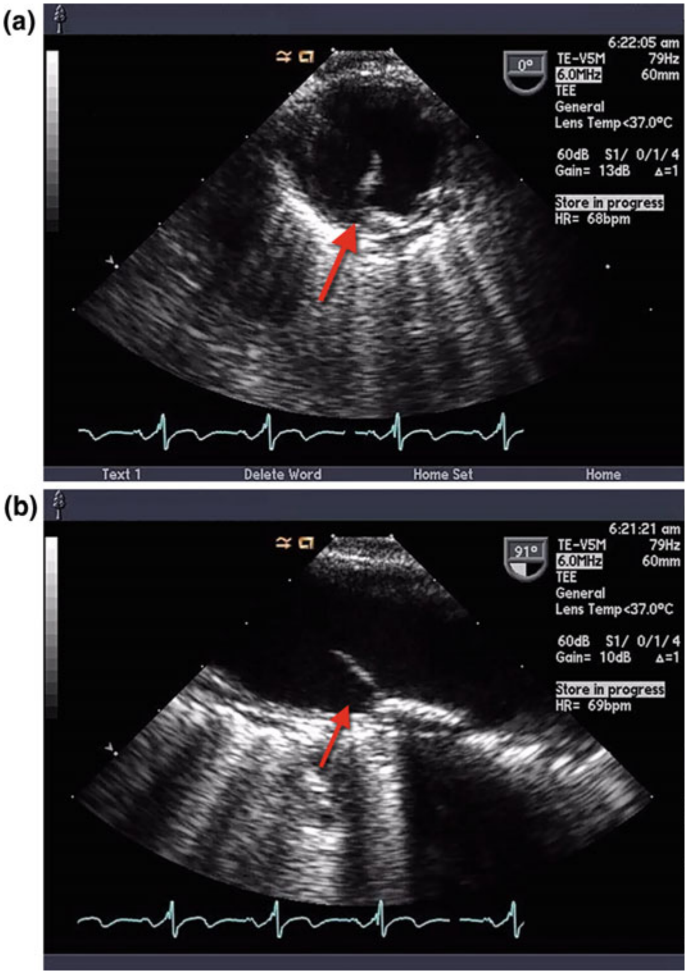


Fig. 10.17 **a** Descending thoracic aortic short axis view demonstrating complex atheromatous disease. The *red arrow* indicates a large pedunculated mobile portion extending into the aortic lumen. **b** Descending thoracic aortic long axis view of the same patient. The *red arrow* again points to the pedunculated mobile portion of the atheroma

Table 10.3 Echocardiographic grading of atheromatous disease

Grade	Description
1	Normal aorta; minimal intimal thickening
2	Extensive intimal thickening
3	Calcified aortic plaque less than 5 mm
4	Calcified aortic plaque greater than 5 mm
5	Mobile atheroma or ulcerated plaque

Thoracic Aortic Trauma

The thoracic aorta has both relatively fixed and mobile portions. The junctions of these portions are often the site of injury in blunt aortic injury, mostly commonly the aortic isthmus (immediately distal to the left subclavian artery) and the ascending aorta (immediately distal to the aortic valve). The most common mechanism for this type of injury is a rapid deceleration which transmits the sheer force between the relatively fixed and mobile portions. This usually involves damage to the aortic intima with potential damage through the media and adventitia including complete aortic transection [8].

Echocardiographically, aortic trauma may share characteristics of a spontaneous aortic dissection. However, on examination of a traumatic aortic injury, the medial flap tends to be thicker in appearance, the lesion is more often isolated without propagation, and there may be the presence of an abnormal aortic contour, an aortic pseudoaneurysm, or a crescentic shaped intramural hematoma. A complete evaluation of the thoracic aorta in this setting should be in consultation with an advanced echocardiographer or confirmed with an alternative imaging technique.

Conclusion

TEE is an excellent monitor for the diagnosis of several aortic pathologies, albeit with some limitations. Knowledge of these limitations allows this modality to be utilized in the setting of aortic dissection, aneurysm, atheroma, and trauma. The basic echocardiographer should have a sound understanding of thoracic aortic imaging with TEE.

References

1. Khalil A, Helmy T, Porembka DT. Aortic pathology: aortic trauma, debris, dissection, and aneurysm. Crit Care Med. 2007;35:S392–400.
2. Nienaber CA, Clough RE. Management of acute aortic dissection. Lancet. 2015;385:800–11.
3. 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.
4. Reeves ST, Finley AC, Skubas NJ, et al. Basic perioperative transesophageal echocardiography examination: a consensus statement of the American society of echocardiography and the society of cardiovascular anesthesiologists. J Am Soc Echocardiogr. 2013;26:443–56.
5. Hata N, Tanaka K, Imaizumi T, et al. Clinical significance of pleural effusion in acute aortic dissection. Chest. 2002;121:825–30.
6. Hiratzka LF, Bakris GL, Beckman JA, et al. ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease. A report of the American College of Cardiology Foundation/American Heart association

task force on practice guidelines, American Association for thoracic surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. J Am Coll Cardiol. 2010;55:e27–129.
7. Wilton E, Jahangiri M. Post-stenotic aortic dilatation. J Cardiothorac Surg. 2006;1:7.
8. Goldstein SA, Evangelista A, Abbara S, et al. Multimodality imaging of diseases of the thoracic aorta in adults: from the American society of echocardiography and the european association of cardiovascular imaging: endorsed by the society of cardiovascular computed tomography and society for cardiovascular magnetic resonance. J Am Soc Echocardiogr. 2015;28:119–82.