Chapter

4 Transesophageal Echocardiography for Diagnosis and Treatment of Aortic Diseases

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Contents

4.1 Introduction

Transesophageal echocardiography (TEE) provides optimal imaging of the whole aorta and sometimes visualizes the upper part of the abdominal aorta. Use of a multiplane probe reduces the classic blind zone, due to the trachea, at the junction of the ascending aorta and the proximal part of the arch. The origin of the left subclavian artery is easily observed. But, emergences of innominate and left carotid arteries remain inconsistently detected. In that case, the transthoracic echocardiographic (TTE) suprasternal view appears very useful. TEE is a semi-invasive procedure. Patient information, screening of contraindications and preparation (local anesthesia, intravenous access, material and structures for resuscitation) are especially important in cases of aortic pathologies. Patients with acute dissection or traumatic rupture of the isthmus need to be explored in an intensive care unit by a trained physician. TEE can be performed quickly, at the bedside and without nephrotoxic contrast agent.

The thoracic aorta is divided into three segments: ascending, horizontal (arch) and descending. TEE allows measurements of each segment, which is determinant for diagnosis, management and follow-up. The ascending aorta presents a particular aspect; four diameters need to be measured: annulus, sinus of Valsalva, sinotubular junction and ascending segment. Age, sex, height, weight and body surface area are independent determinant factors of these diameters. The aortic annulus and the sinus of Valsalva are correlated with body surface area; the sinotubular junction and the ascending segment are more related to age [19].

4.2 Transesophageal Echocardiography for Diagnosis of Aortic Diseases

4.2.1 Aortic Dissection

Aortic dissection occurs generally in patients with risk factors: age, hypertension, disease of fibroelastic tissue (Marfan syndrome), congenital disease (bicuspid valve, coarctation) or pregnancy. Iatrogenic or traumatic causes are less frequent. The dissecting process has generally an anterograde longitudinal extension, but retrograde dissection is possible. Circular extension involves partial or complete wall circumference. Two classifications may be used. De Bakey classification presents three types. In type I, the ascending segment is at least dissected with variable extension to horizontal and descending parts. In type II, dissection is only localized in the ascending aorta. Type III consists in an involvement of the descending thoracic aorta (IIIa) with possible extension to the abdominal aorta (IIIb). Stanford classification is now currently used. Type A defines dissections with, at least, the involvement of the ascending aorta, whatever the extension or the site of intimal tear. Type B dissection respects the ascending aorta. It concerns essentially the descending aorta with possible anterograde extension to the abdominal segment or retrograde to the horizontal aorta [5, 7].

4.2.1.1 Intimal Flap

Dissection consists in a cleavage of the medial layer of the aortic wall. It leads to formation of the intimal flap, which really corresponds to the association of the intimal layer and two thirds of media. The intimal flap represents the first echocardiographic sign of aortic dissection (Fig. 4.1) [7]. On TEE it appears as a linear intraluminal, thin and mobile echo that divides the aortic lumen in two parts: true and false lumens. There is a correlation between flap mobility and mortality.

4.2.1.2 Intimal Tear

The intimal tear corresponds to the beginning of the dissecting process. Its detection is determinant to choose the time and type of treatment. From this point, dissection has generally an anterograde progression but retrograde extension is possible. The tear appears as a discontinuity of the intimal flap. A multiplane TEE probe provides a direct image of the tear and allows its measurement (Fig. 4.2). Color Doppler imaging enhances detection of the tear and can reveal the presence of other multiple small communications between the two lumens, especially in the descending aorta. They appear as thin color flows without a direct image of the tear (Figs. 4.3, 4.4). Anatomical controls showed that these images might correspond to the origin of intercostal arteries [30]. Intravenous infusion of an echographic contrast agent can detect them with good accuracy (Fig. 4.5). There is a positive relation between flap mobility and the site of the tear. Increased mobility is observed around the tear and helps to localize it. Pulsed Doppler imaging flow velocities through the intimal tear reflect the pressure gradient between the two lumens (Fig. 4.6). During a cardiac cycle, the velocity profile may have a mono-, bi-, tri- or quadriphasic aspect [7].

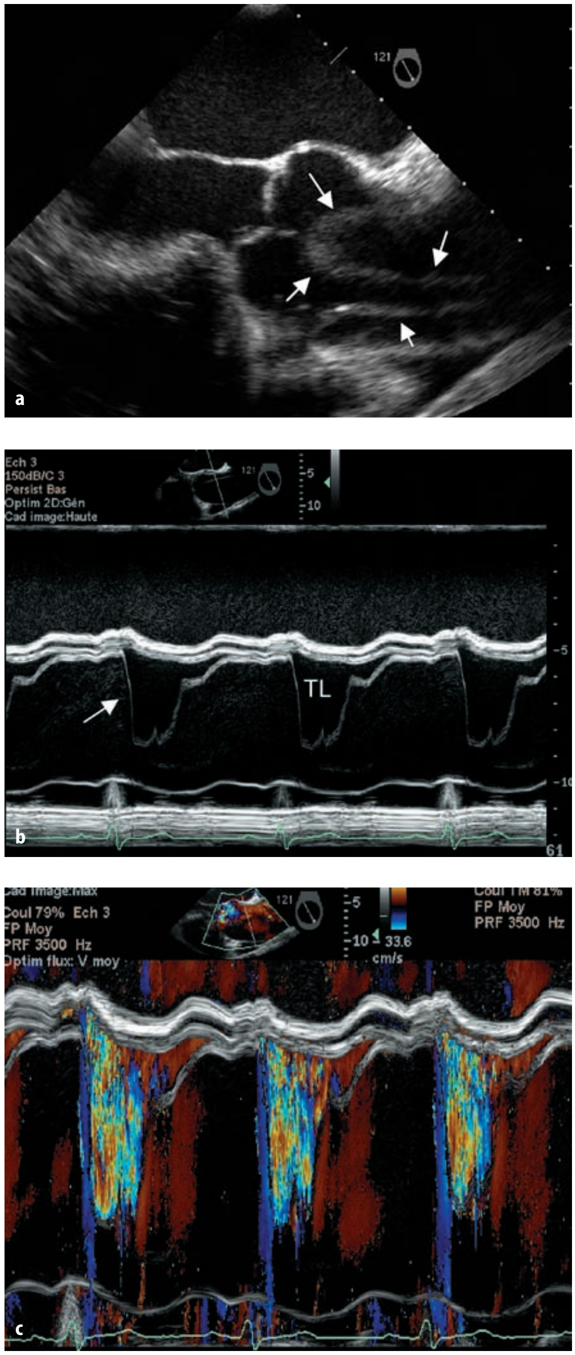

Fig. 4.1. Dissection of the ascending aorta. **a** Longitudinal view with sinuous intimal flap (*arrows*). **b** M mode showing amplitude of displacement of the intimal flap. **c** M mode color Doppler image showing systolic flow into the true lumen (*TL*)

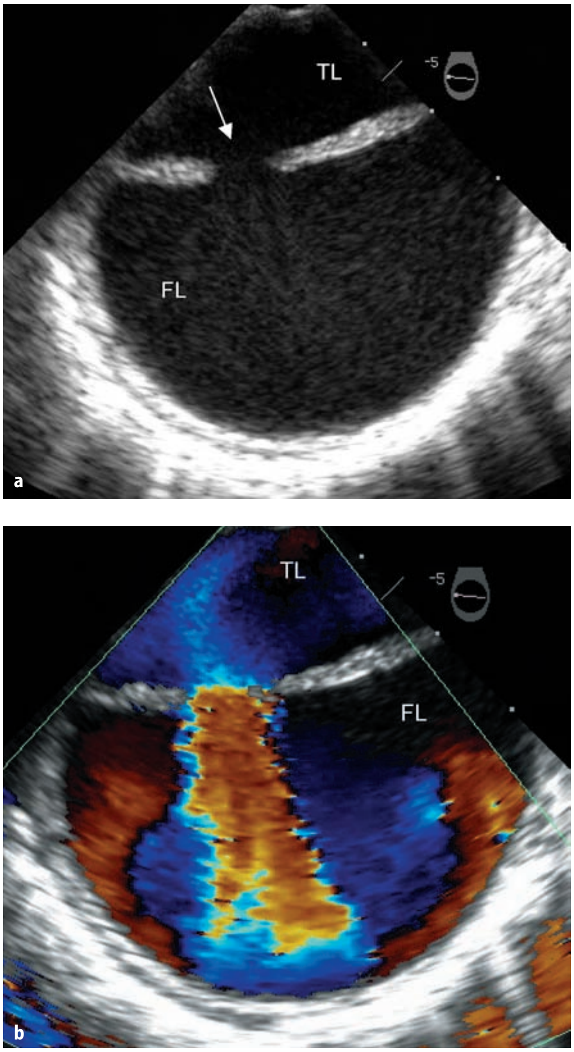

Fig. 4.2. Dissection of the descending aorta. **a** Intimal tear (*arrow*). **b** Color flow through the intimal tear, from the true lumen (*TL*) towards the false lumen (*FL*)

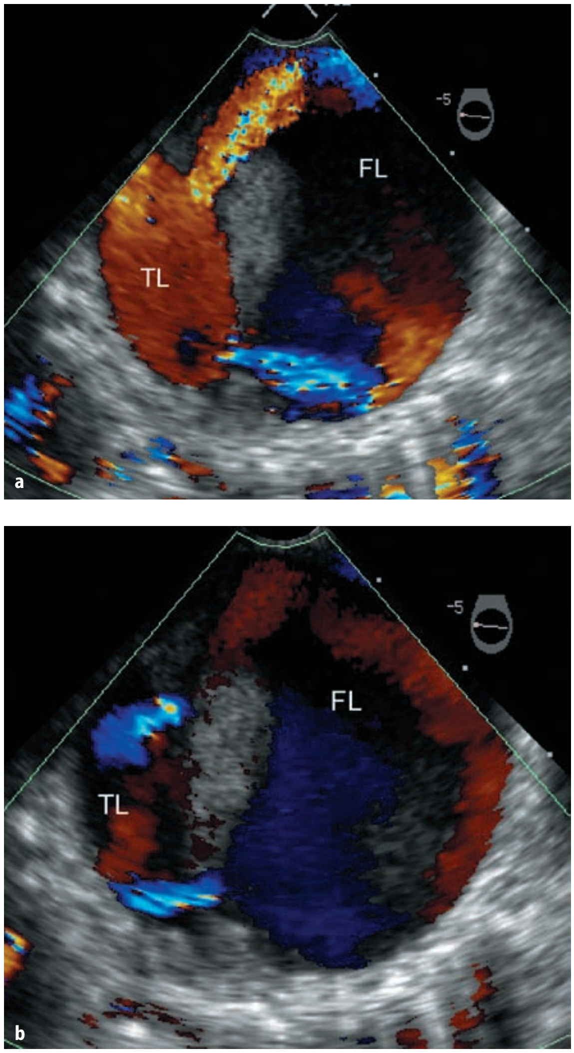

Fig. 4.3. Dissection of the descending aorta. Color Doppler flow through two intimal tears. **a** Towards the false lumen (*FL*) in the systole. **b** Towards the true lumen (*TL*) in the diastole

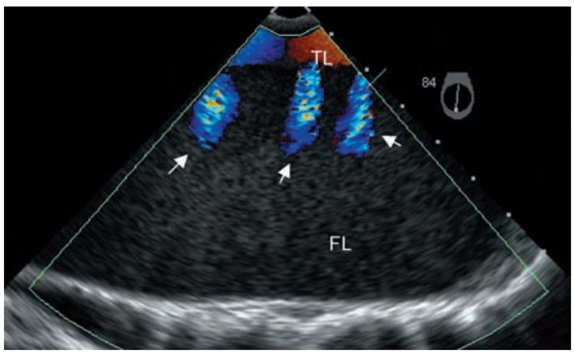

Fig. 4.4. Dissection of the descending aorta. Color Doppler flow of small communications (*arrows*) between the true lumen (*TL*) and the false iumen (*FL*) that might correspond to ostia of intercostal arteries

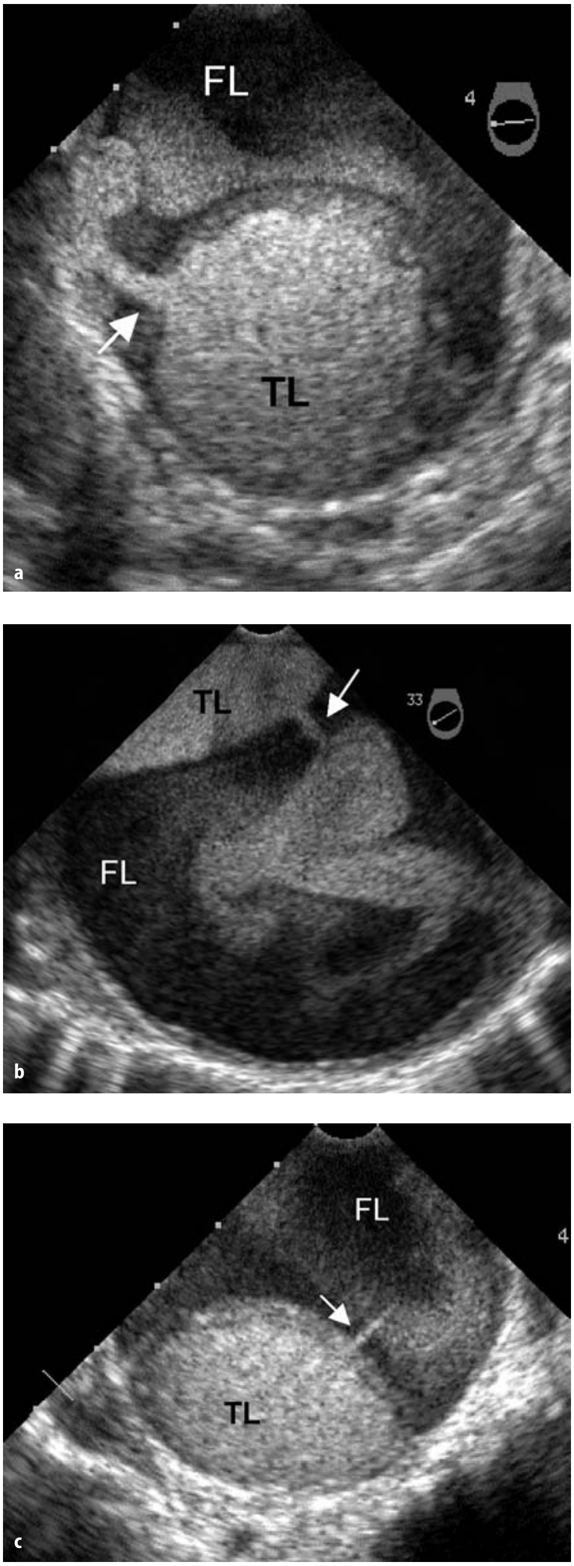

Fig. 4.5. Dissection of the descending aorta. Contrast echographic agent for detection of small intimal tears (*arrows*), true lumen (*TL*) and false lumen (*FL*)

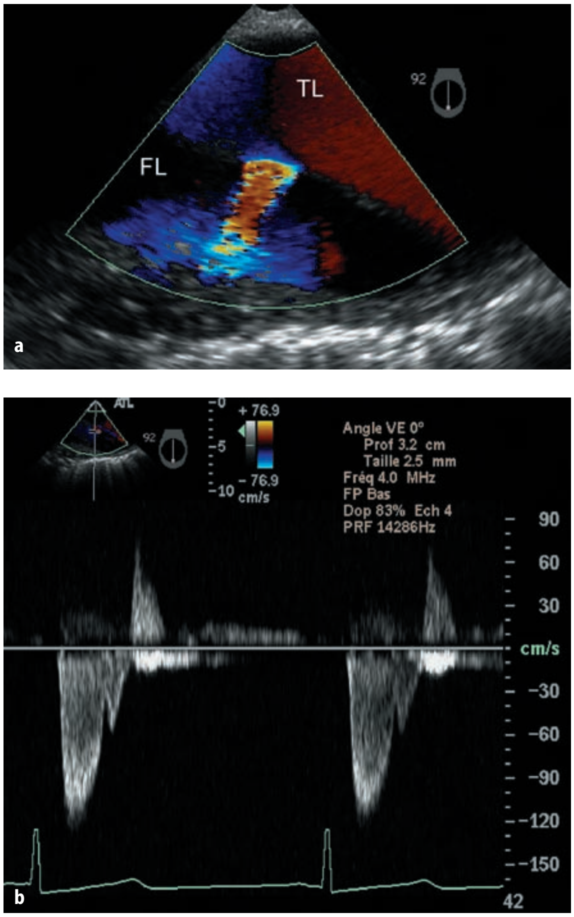

Fig. 4.6. Dissection of the descending aorta. **a** Color flow through the intimal tear, from the true lumen (*TL*) towards the false lumen (*FL*). **b** Pulsed Doppler image through the intimal tear

4.2.1.3 True and False Lumens

The true lumen is generally smaller than the false one but presents a systolic expansion [7]. Color Doppler imaging presents an aspect of aliasing owing to the high velocity of systolic anterograde flow (Fig. 4.7). The false lumen is larger and presents a systolic compression. Blood velocity is lower and, sometimes, results in a constitution of the spontaneous echo contrast effect. It appears as dynamic smokelike echos with slow swirling movements that are markers of blood stasis. So, a thrombus is frequently observed in the false lumen. The importance of thrombosis (partial or complete) seems to be negatively related with the site and the size of the tear. A thrombus must be distinguished from thin cobweblike echos joining the intimal flap to the outer wall of the false lumen. These residual strands of media, essentially observed in the descending aorta, may be helpful to identify the false lumen [29]. Dissection is frequently associated with and complicated by valvular regurgitation, involvement of collateral arteries, and blood extravasation to periaortic structures.

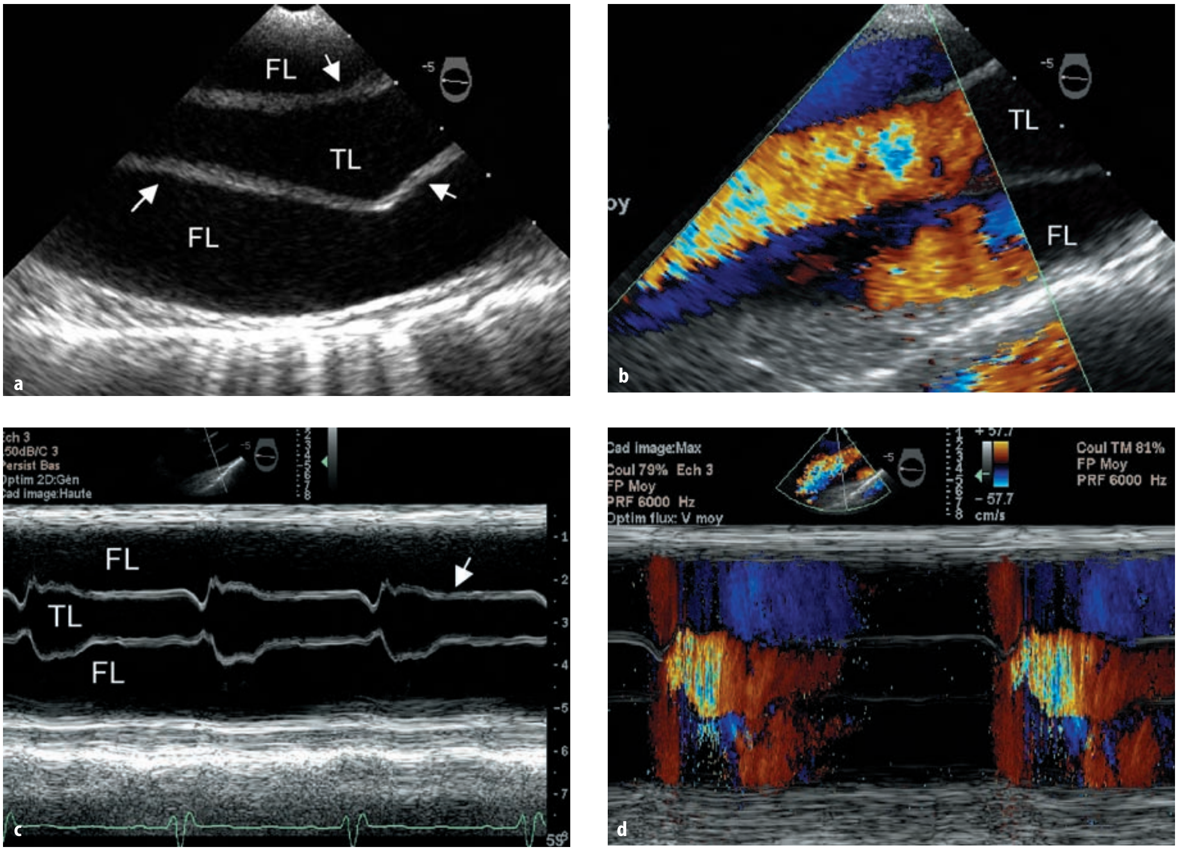

Fig. 4.7. Dissection of the aortic arch. **a** Two-dimensional view of the horizontal aorta showing the intimal flap (*arrows*) separating the true lumen (*TL*) and the false lumen (*FL*). **b** Color Doppler flow within the true lumen (*TL*). **c** M mode at the same level. **d** M mode color Doppler image into the true lumen

4.2.1.4 Aortic Regurgitation

This complication is frequent. It occurs in about 40–76% of patients [15]. Some cases require surgical treatment. TTE is used to evaluate the grade of regurgitation and its consequences on left ventricular size and function. TEE is determinant to analyze the mechanism of regurgitation and the possibility of valve repair. Five mechanisms are described: incomplete valve closure due to dilatation of the sinotubular junction, leaflet prolapse by extension of dissection to the valve attachment, intimal flap prolapse into aortic annulus [15] and bicuspid and degenerative valves. The first three mechanisms, characterized by a normal leaflet structure, may benefit from a conservative treatment by surgical valve repair. The last two mechanisms are independent of dissection and must be treated by valve replacement if needed.

4.2.1.5 Aortic Branches

Mechanisms of the involvement of aortic branches are multiple: dissection, disinsertion or obstruction of the artery ostium, compression by the false lumen. Except for the left subclavian artery, TEE fails to analyze these complications (Fig. 4.8). However, dissection of the initial part of the coronary artery has been detected by TEE. Computer tomography (CT) and MRI are more efficient in recognizing them [16]. Extension of dissection to the innominate and left carotid arteries can be detected by TTE in suprasternal incidence.

4.2.1.6 Periaortic Structures

Acute or subacute bleeding from the aorta can involve periaortic structures with constitution of pericardial, mediastinal or pleural effusions (Fig. 4.9). Mediastinal hematoma is characterized by an increased distance between the esophagus and the aorta or the left posterior atrial wall [12].

4.2.1.7 Pitfalls

TEE evaluation of aortic dissection includes some pitfalls that can impair its accuracy: intimal flap artefacts, localized or retrograde dissections, aneurysm or false aneurysm with a thrombus, complex plaque of athero-

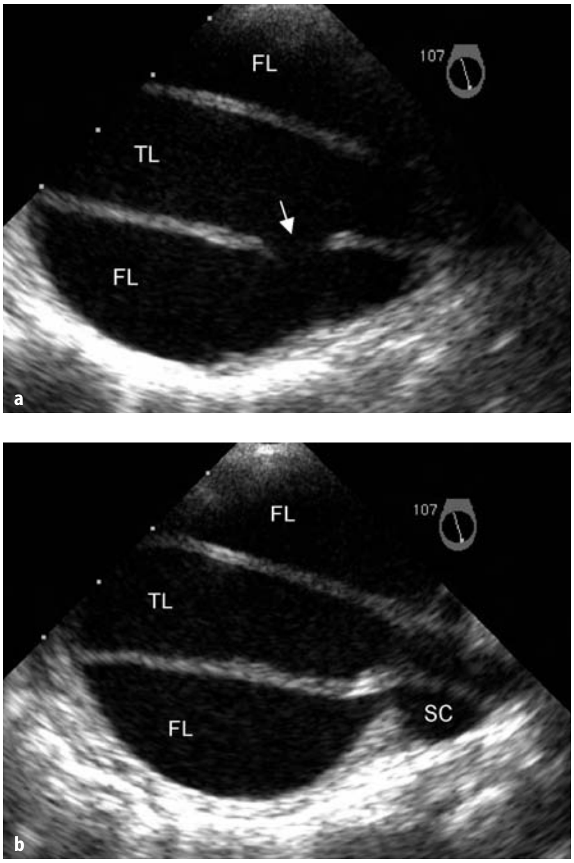

Fig. 4.8. Dissection of the aortic arch. **a** Intimal tear (*arrow*). **b** Extension of the intimal flap into the subclavian artery (*SC*). *TL* true lumen, *FL* false lumen

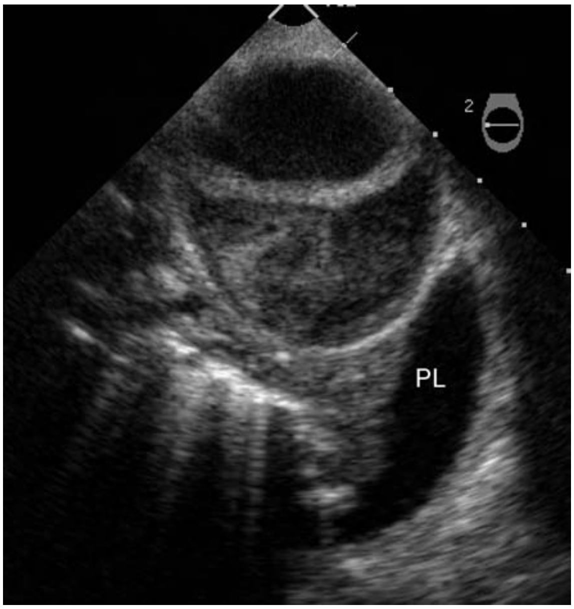

Fig. 4.9. Dissection of the descending aorta complicated by pleural effusion (*PL*)

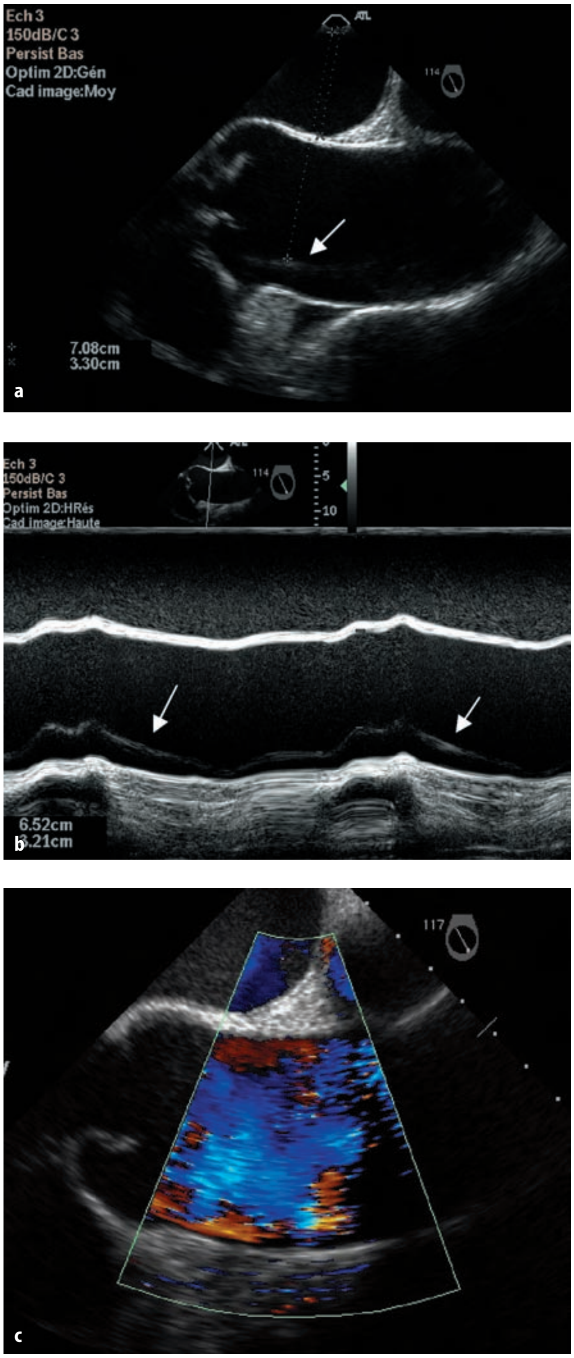

Fig. 4.10. Aortic dissection. Linear artefacts into ascending aorta. **a** Two-dimensional long-axis view. **b** M mode. **c** Color Doppler image showing homogeneous flow

ma with a mobile component, tumor, innominate or azygos veins, periaortic fat tissue or abscess. Linear artefacts mimicking the intimal flap are frequent in the ascending aorta and are infrequent in the horizontal arch (Fig 4.10). This is due to echographic reverberation of the left atrial or right pulmonary artery walls.

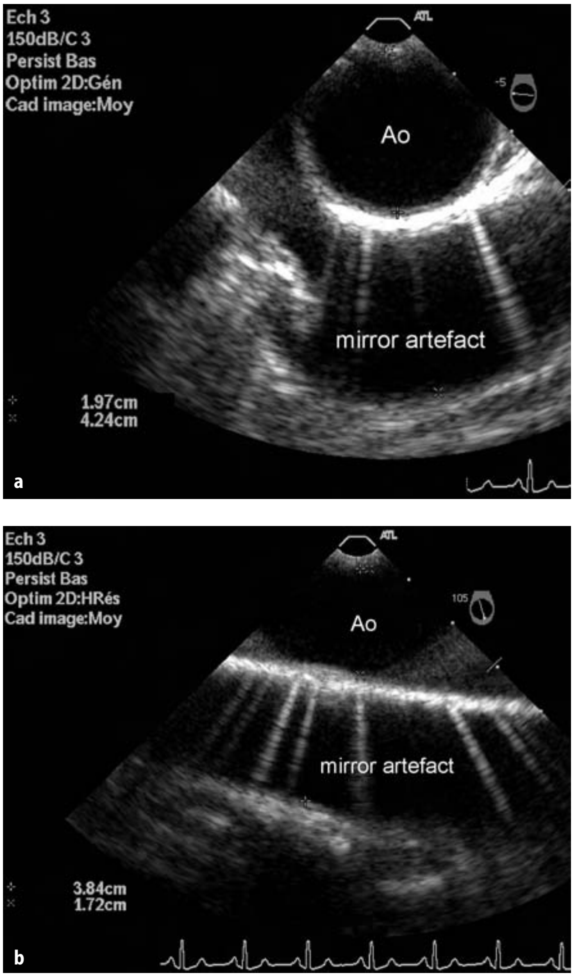

Fig. 4.11. Transesophageal artefacts of aortic dissection. Mirror artefact in descending aorta mimicking two channels. **a** Transverse view. **b** Longitudinal view. *Ao* aorta

In M mode, artefact echo is generally located at twice the distance from the transducer and structure that produces the artefact and presents a double-amplitude displacement. The color flow aspect is similar on both sides of the artefact [1, 6]. Pulsed Doppler imaging contributes by positioning the sample volume at the suspected image level. The intimal flap produces high-intensity signals with clicks that are absent in the case of an artefact [21]. A mirror artefact is frequent in the descending aorta. A typical aspect consists in an image of two parallel channels with the same diameter (Fig. 4.11) [1]. Despite these limits, TEE sensitivity, specificity, positive and negative predictive accuracies are greater than 90% and similar to those of CT. However, MRI offers the best accuracy (near 100%) [16].

New cardiovascular imaging tools and improvements of treatments and follow-up periods lead to a new concept of aortic diseases. A new classification recently proposed [27] includes five classes: class 1, classic aortic dissection; class 2, intramural hematoma or hemorrhage; class 3, small dissection with an eccentric bulge at the tear site; class 4, penetrating aortic ulcer; class 5, iatrogenic and traumatic dissections. Each class 2, 3 and 4 lesion could be a precursor of classic aortic dissection.

4.2.2 Intramural Hematoma

Intramural hematoma consists in bleeding into the medial layer of the aortic wall. A cystic medial degeneration is often present and leads to a rupture of the vasa vasorum. Intramural bleeding induces circular and longitudinal cleavage of the aortic wall. Hematoma can involve the whole aorta. It occurs typically in elderly patients (mean 65–70 years) with hypertension. Symptoms mimic aortic dissection. Complications are severe and frequent, especially if the ascending aorta is involved. Classification of hematoma is similar to that of dissection. Type A involves at least the ascending aorta; type B, the descending part. TEE findings are associated with the following: a circular or crescentic thickening (more than 5 mm) of the aortic wall (more than 7 mm in the initial description); an increased thrombuslike homogeneous echodensity; an absence of the intimal flap, intimal tear and flow; a longitudinal mean extension of 1–20 cm [13, 17]. The aortic diameter is generally increased. In the case of intimal calcification or atheroma, they are displaced towards the center of the aortic lumen. Hemomediastinum, pericardial or pleural effusions may be associated and represent signs of complications. Compared with this classic form, other echographic aspects could be observed. They are related to the importance and the onset of the intramural bleeding. The crescentic image can be heterogeneous with echo-free space or completely nonechogen. These forms correspond to recent bleeding or to liquefaction of hematoma (Fig. 4.12). In such cases, CT or MRI (by distinction between oxyhemoglobin and methemoglobin) are very helpful in evaluating the age of lesions [7, 17]. If important, intramural hematoma presents as a classic noncommunicating dissection with an intimal flap but no tear. However, increase of the bleeding and intramural pressure can induce an intimal tear. Several types of evolution have been described [13, 17]: normalization; decrease; stabilization or increase of wall thickness; circular and/or longitudinal extension; aortic dissection or rupture. TEE control within 3 months of acute syndrome helps to evaluate the trend of evolution. Aortic dilatation or aneurysm occur later. TEE signs of hematoma must be distinguished: aortic dissection and thrombosed false lumen; aneurysm with a thrombus; plaque of atheroma; penetrating aortic ulcer.

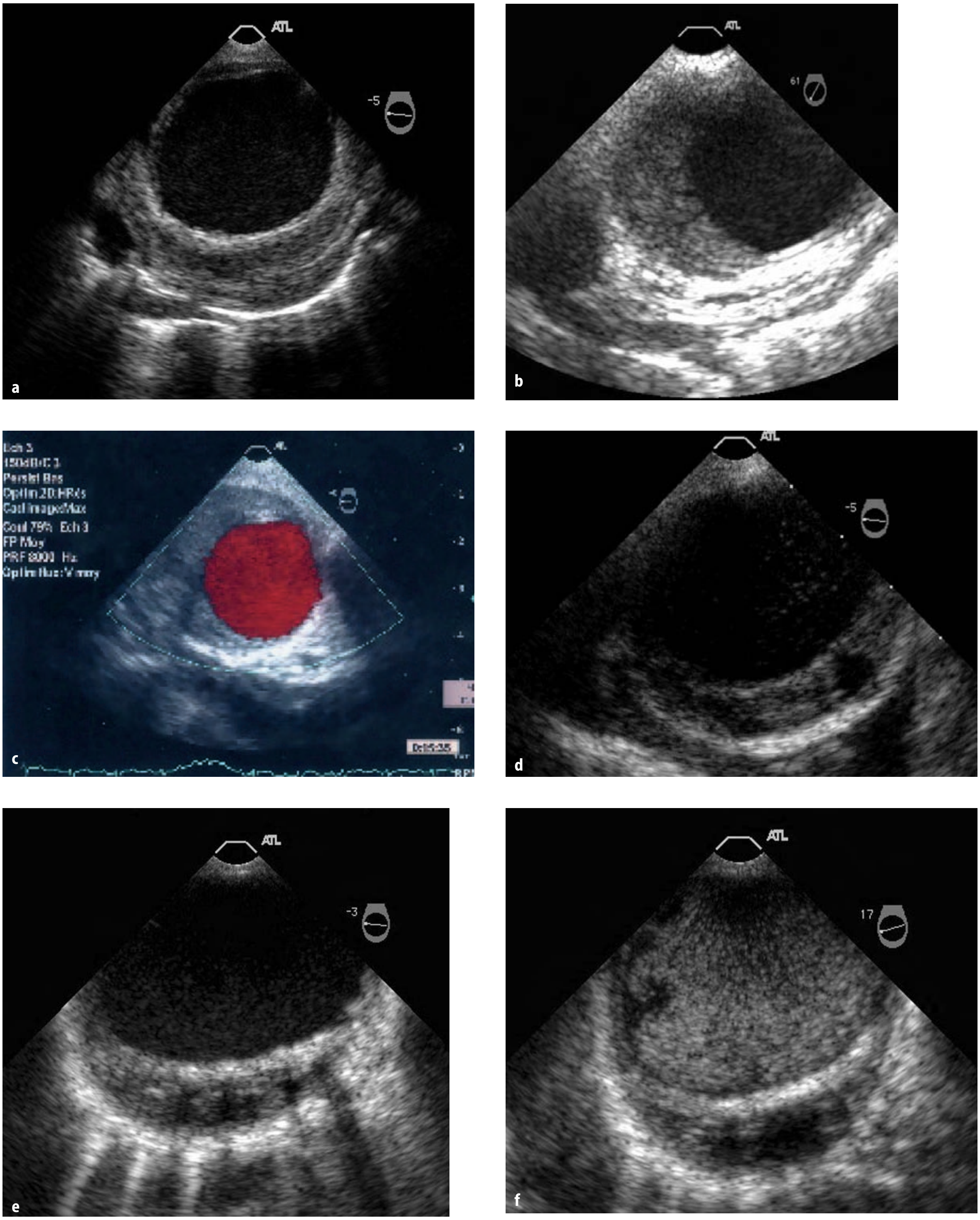

Fig. 4.12. Intramural hematoma of the descending aorta. Crescentic thickening of the aortic wall. **a**, **b** Typical aspect with thrombuslike homogeneous echodensity. **c** Crescentic aspect, color flow in the aortic lumen. **d** Inhomogeneous aspect with echo-free spaces. **e** Nonechogen aspect mimicking dissection. **f** After injection, echographic contrast agent is only present in the aortic lumen, confirming the absence of communication with the hematoma

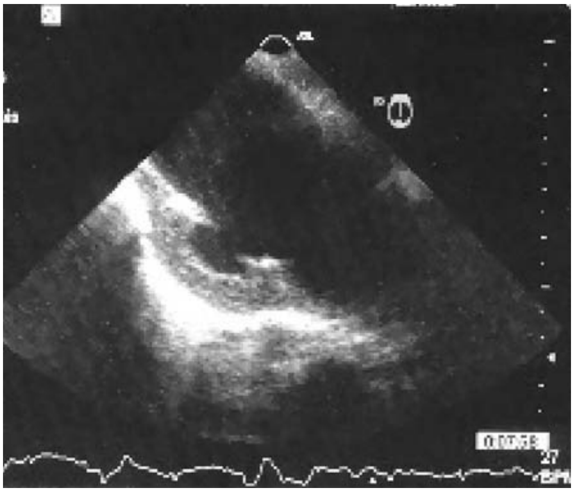

Fig. 4.13. Penetrating aortic ulcer of the descending aorta. Ulcerated plaque with crater and constitution of an adventitial false aneurysm

4.2.3 Penetrating Aortic Ulcer

A penetrating aortic ulcer is an entity described by Stanson et al. [26] as an ulceration of a plaque of atheroma that extends through intima into internal elastic lamina. It occurs generally in elderly patients (over 75 years) with hypertension, multiple-risk factors (smoking) and severe complex atheroma plaques. Symptoms are similar to those of aortic dissection. Penetrating ulcers involve a classic descending aorta. Circular and longitudinal extensions are less important than hematoma. The typical TEE aspect consists in a thick atherosclerotic plaque with a deep crater limited by irregular edges (Fig. 4.13). Color Doppler imaging enhances the detection of the ulcer and measurement of the crater dimensions. There is no intimal flap, nor false lumen. Angiography was the first method of diagnosis and showed a localized additional contrast image. CT and MRI are more efficient in the diagnosis of complications. It has been proved that a penetrating ulcer has a severe potential of evolution: aortic dissection, hematoma of the medial layer, adventitial false aneurysm or transmural rupture [14, 26].

4.2.4 Aortic Aneurysms

Aortic diameters are related to age, sex, height, weight, body surface area and site. Dimensions decrease regularly from the valve annulus to the iliac arteries. For one site, an aneurysm is defined as an increase (more than 50%) of the expected diameter and is associated with a loss of parallelism of the aortic wall. The two main etiologies are atherosclerosis (particular risk with hypertension and smoking) and dystrophy of conjunctive tissue [7].

4.2.4.1 Atherosclerotic Aneurysms

Atherosclerotic aneurysms involve generally horizontal and descending aorta. Two types are observed. The most frequent is the fusiform aneurysm characterized by an increased diameter of the lumen and nonparallelism of the wall. TEE determines the site, the diameters and the circular and longitudinal extensions, and detects associated abnormalities: wall thrombus, spontaneous echo contrast, complex atheroma plaques (Fig. 4.14).

The second type, less frequent and less extended, is the false (or pseudo) aneurysm. TEE signs are associated with a small neck and a large cavity (Fig. 4.15). These conditions promote a blood stasis, and a thrombus is frequent.

4.2.4.2 Dystrophic Aneurysms

Dystrophic aneurysms involve principally ascending aorta up to the innominate artery. The typical aspect is that of annulo-aortic ectasian disease. Etiologies include the Marfan syndrome (principally in patients younger than 40 years), characterized by mutation of genes localized on chromosome 15, and idiopathic forms. These forms present nearly similar histologic lesion of kystic medianecrosis. Dilatation involves the valve annulus, the sinus of Valsalva and sometimes the sinotubular junction. All these elements lead to raise valve commissures and to stretch the cusps. Valve movement is impaired and this results in an incomplete closure with diastolic regurgitation (Fig. 4.16). Bicuspid or valve prolapse with eccentric flow may be observed [7]. Quantification of regurgitation and consequences for the left ventricular size and function need to be evaluated by TTE. Tricuspid and mitral valve dystrophy may be associated. Aortic dissection is the main complication of dystrophic aneurysms.

4.2.4.3 Aneurysm of the Sinus of Valsalva

Aneurysm of the sinus of Valsalva is an infrequent disease, mainly observed in young men. A congenital origin is frequent and explains associations with other abnormalities: bicuspid aortic valve with regurgitation, coarctation, interventricular septal defect. Other etiologies are Marfan syndrome, endocarditis or inflammatory diseases of the aortic wall [11]. There are two TEE aspects: localized dilatation (generally right anterior part) of the sinus or fingerlike expansion of the sinus (Fig. 4.17). Color Doppler imaging is useful to detect the main complication that consists in a disruption into the right atrium, the right ventricle or more rarely the left atrium or pericardium. These images have to be distinguished from aortic annulus abscess.

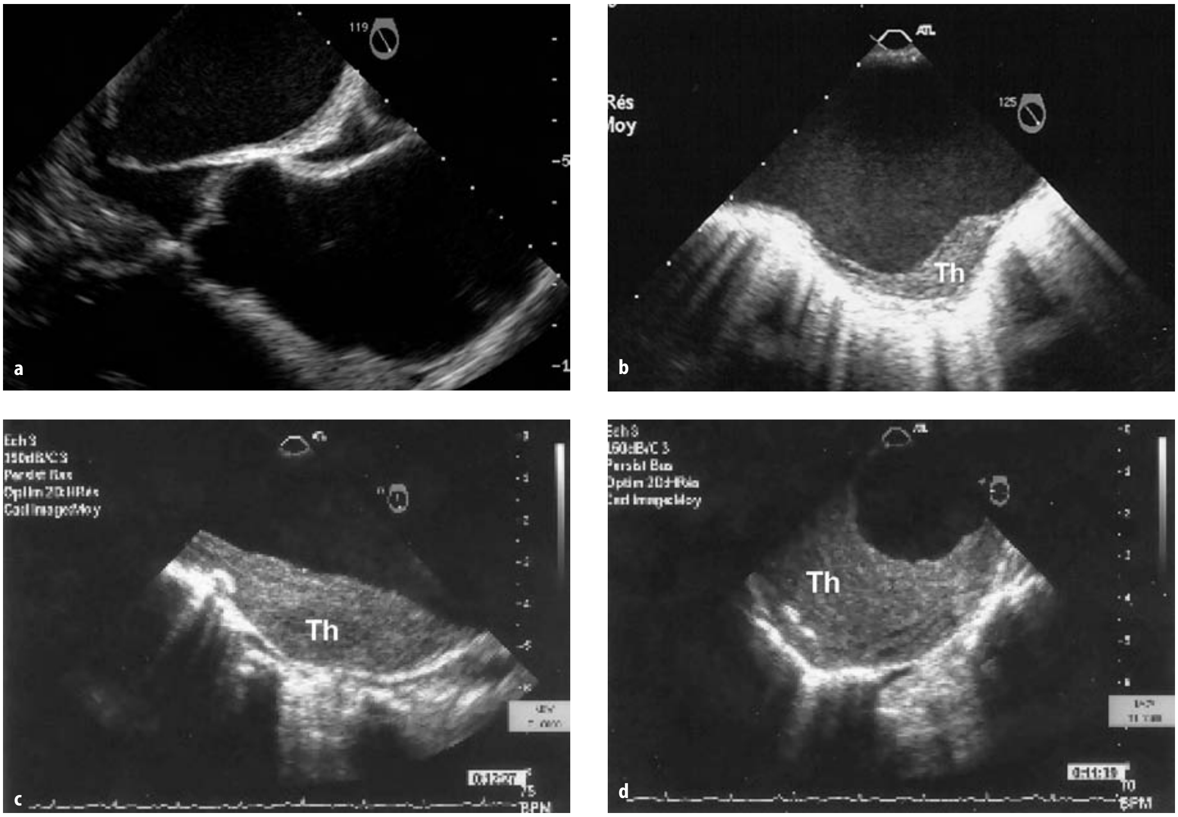

Fig. 4.14. a Aneurysm of the ascending aorta. **b** Fusiform aneurysm of the descending aorta with a mural thrombus (*Th*). **c, d** Longitudinal and transverse views of aneurysm with an important thrombus

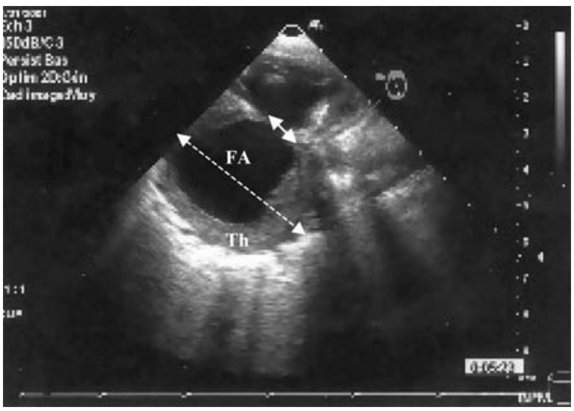

Fig. 4.15. False aneurysm (*FA*) of the descending aorta. The neck diameter (*full line*) is smaller than the aneurysm diameter (*dotted line*). *Th* mural thrombus

4.2.4.4 Aneurysms and Systemic or Inflammatory Diseases

Takayasu disease, observed in young women, involves the ascending aorta. It is associated with fusiform dilatations and stenosis. The aortic wall is thick. The main risk consists in rupture. Horton disease occurs in older patients (over 70 years). Aortic involvement is less frequent than temporal artery. In Behcet disease, pseudoaneurysms alternate with stenosis. Aneurysms can be observed in rheumatoid arthritis, Ormond and Cogan diseases and Reiter syndrome. Recently, cocaine and amphetamine have been suspected to induce dissection and aneurysm formation [7].

4.2.4.5 Aneurysms and Infectious Diseases

Syphilis involves generally the upper part of the ascending aorta. Lesions consist in pseudoaneurysm with a thrombus and calcification of the aortic wall. Actually, small mycotic aneurysms may be observed during bacterial or parasitical infections. In such situations, after aortic surgery, images of a false aneurysm may be observed at the anastomosis between the aortic tube graft and the native aorta.

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Thoracic Aortic Diseases
Rousseau, H.; Verhoye, J.-P.; Heautot, J.-F. (Eds.)
2006, XVII, 387 p., Hardcover
ISBN: 978-3-540-25734-9