Evaluation of the Great Vessels

7

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Evaluation of the great vessels should include the following:

(a) Outflow tracts. These may be switched, narrowed, absent, or aneurysmal.

(b) Aorta
   (i) It may be discontinuous (interruption of the aortic arch), stenotic in patients with coarctation of the aorta or supravalvular aortic stenosis, or hypoplastic.
   (ii) Identify whether the arch is on the left or right, and determine the number and location of vessels arising from the arch. Vascular rings, such as a double arch or right arch with an aberrant left subclavian artery, may cause airway obstruction.
   (iii) Look for aortopulmonary collaterals. These typically are seen along the descending aorta but may arise from the arch or great vessels.
   (iv) Measure the size and caliber of the aorta at the aortic annulus, sinus of Valsalva, sinotubular junction, transverse arch, and descending aorta. Comparison with normative data (z-score) may be helpful.

(c) Pulmonary arteries
   (i) They may be atretic, hypoplastic, or anomalous. Determine the origin and course of the pulmonary arteries. Some pulmonary arteries may arise from the aorta, a patent ductus arteriosus (PDA), or the other pulmonary artery. (In pulmonary sling, the left pulmonary artery arises from the right and courses around the trachea, often causing tracheal or bronchial stenosis.)
   (ii) Evaluate the size of the main pulmonary artery and the right and left proximal and distal pulmonary arteries.
   (iii) Look for other vessels connected to the pulmonary arteries, such as a PDA. Aortopulmonary collaterals may be connected to the pulmonary arteries and may supply the lung directly.

(d) PDA
   (i) Typically, a PDA originates from the undersurface of the descending aorta or left brachiocephalic/subclavian artery but rarely may have other anomalous origins and may be bilateral, with one arising from the aorta and the other from the brachiocephalic artery.
   (ii) It may be large and tortuous, especially in patients with complex congenital heart disease. A diverticulum may be seen at the origin of the ductus (Kommerell's diverticulum). Look for mass effect from the enlarged PDA on other adjacent structures (especially the trachea and bronchi).

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(iii) Later in life, a calcification often is seen in the region of the ligamentum arteriosum that forms when the ductus closes.

Normal Anatomy

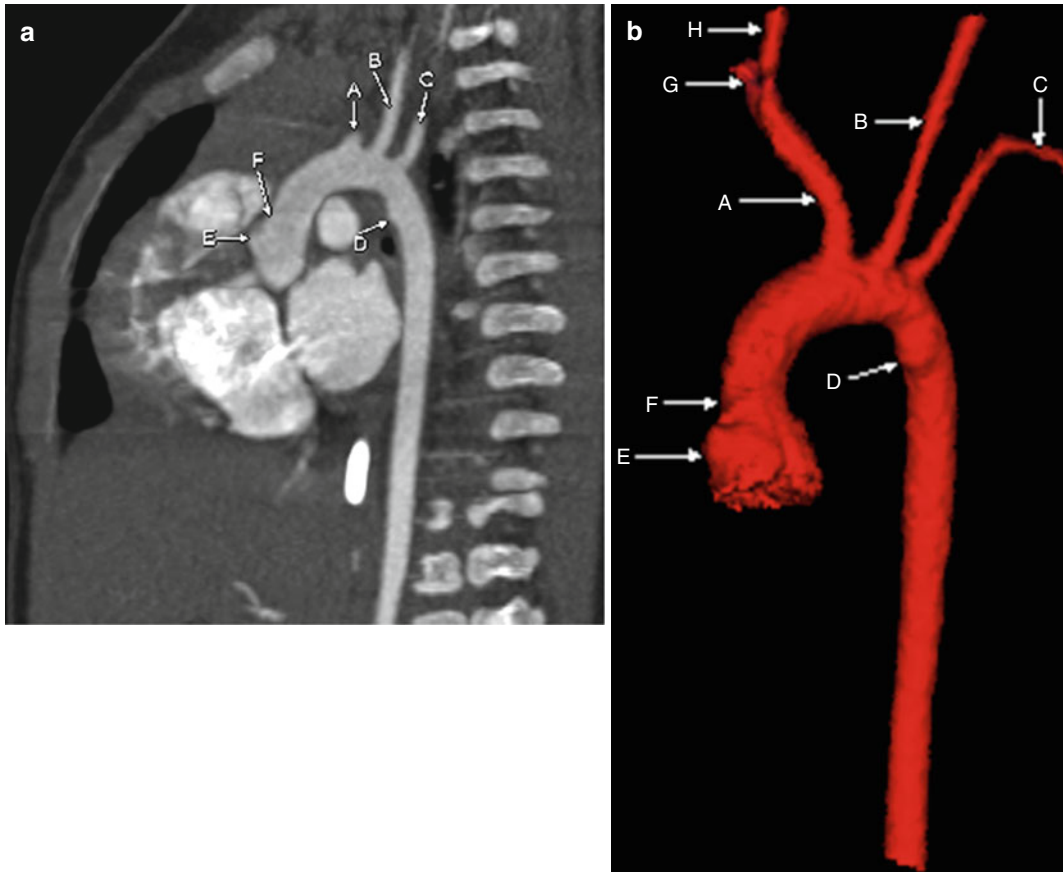


Fig. 7.1 Sagittal maximum-intensity projection (MIP; **a**) and lateral projection color-coded three-dimensional (3D; **b**) images from a cardiac CTA demonstrating a normal three-vessel aortic arch. The sinus of Valsalva (*E*) typically bulges, with immediate narrowing at the sinotubular junction (*F*). The first vessel of a left aortic arch is the right brachiocephalic artery (*A*), which divides further into right subclavian (*G*) and right common carotid (*H*) arteries. The second vessel off the arch is the left common carotid artery (*B*) and the last is the left subclavian artery (*C*). Notice the typical ductus bump (*D*), which is seen along the undersurface of the descending aortic arch

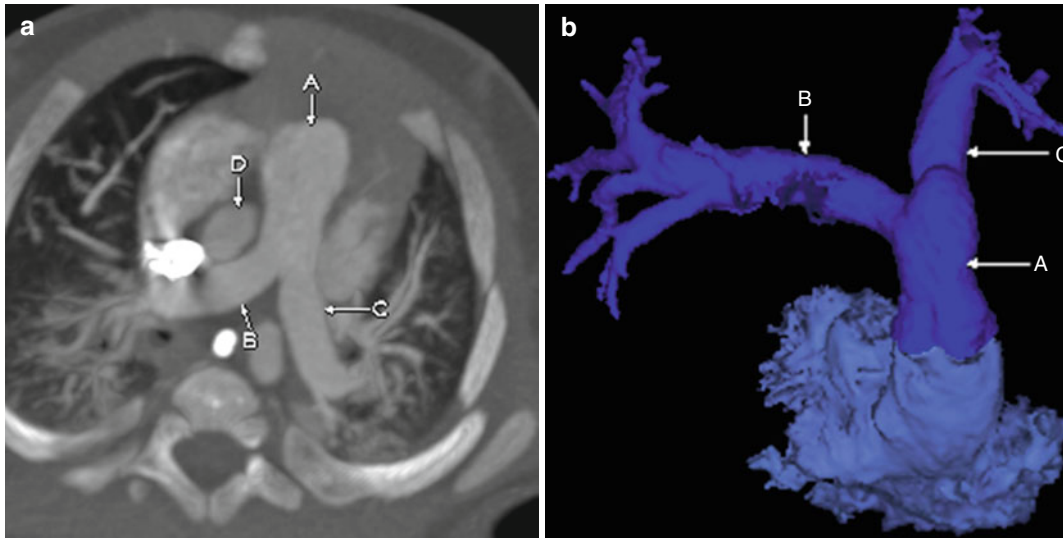


Fig. 7.2 Axial MIP (**a**) and 3D color-coded (**b**) images from a cardiac CTA. The main (*A*), right (*B*), and left (*C*) pulmonary arteries are shown. Notice the normal position of the ascending aorta (*D*) posterior to and to the right of the pulmonary outflow tract

PDA

PDA is the persistence of the normal prenatal vascular connection between the pulmonary artery and proximal descending aorta or brachiocephalic artery. In utero, the ductus arteriosus functions as a right-to-left shunt to bypass the pulmonary vasculature. After birth, it becomes a left-to-right shunt because of the higher pressures in the aorta relative to those in the pulmonary system. A PDA is a type of acyanotic heart disease with increased pulmonary vasculature, cardiomegaly, and a prominent "ductus bump." A longstanding PDA may cause pulmonary hypertension, flow reversal, and cyanosis (Eisenmenger's physiology). When it closes, a PDA forms a ligamentum arteriosum, which often calcifies.

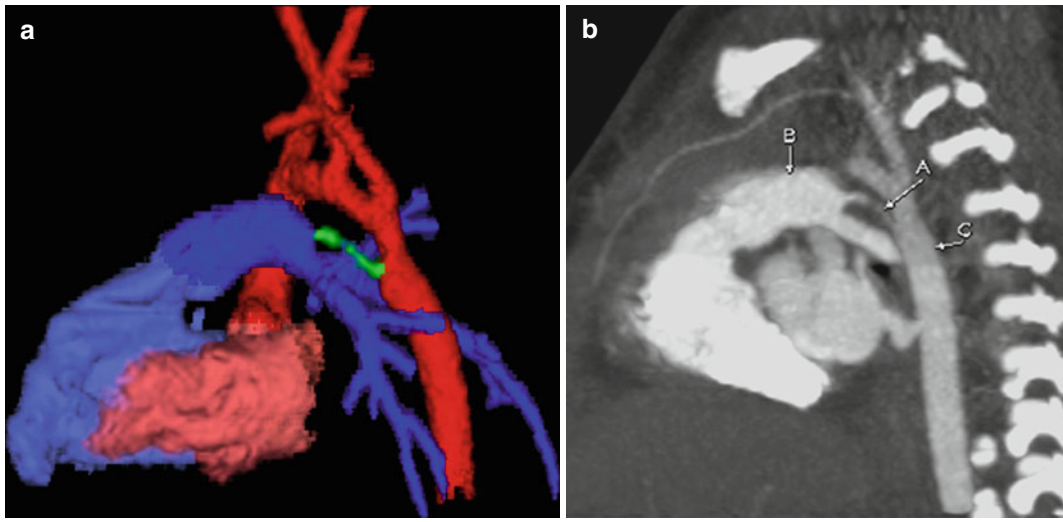


Fig. 7.3 Color-coded 3D (**a**) and sagittal MIP (**b**) images from a cardiac CT showing a small PDA (*green*, *A*) connecting the pulmonary artery (*blue*, *B*) to the proximal descending aorta (*red*, *C*)

PDA from the Left Brachiocephalic Artery

A PDA may arise from the aorta or left brachiocephalic/left subclavian artery. With a right arch and an aberrant subclavian artery, the PDA may complete a vascular ring when it arises from an aberrant left subclavian artery.

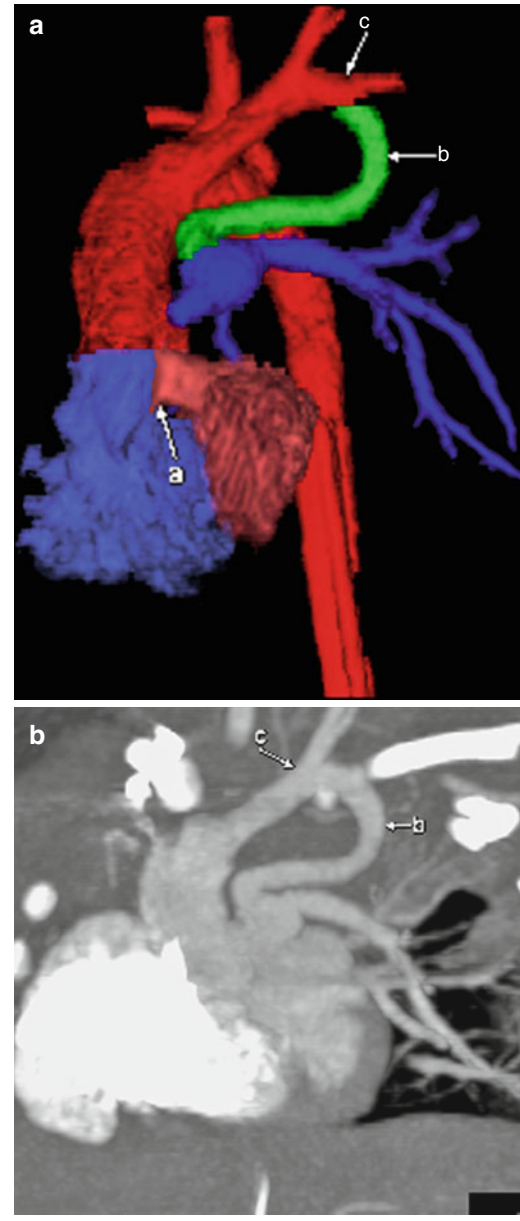


Fig. 7.4 Color-coded 3D (**a**) and sagittal MIP (**b**) images from a cardiac CT showing a PDA (*b*) attached to the left subclavian artery (*c*) in a patient with a right aortic arch. A ventricular septal defect (VSD; *a*) also is shown on the 3D image

PDA from the Descending Aortic Arch

A PDA often is necessary for survival, especially if there is obstruction to aortic flow, as seen in severe aortic stenosis, coarctation of the aorta, interruption of the aortic arch, and hypoplastic left ventricle. The PDA may be stented (hybrid procedure) in some patients with hypoplastic left ventricle.

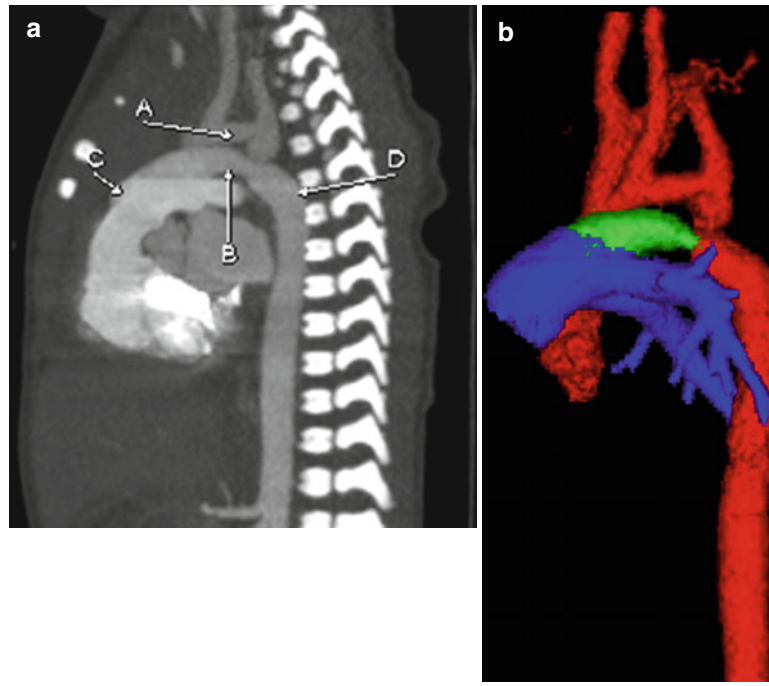


Fig. 7.5 Sagittal MIP (**a**) and color-coded 3D (**b**) images from a cardiac CT scan showing a PDA (*B*, *green*) connecting the descending aortic arch (*D*, *red*) and the pulmonary artery (*C*, *blue*). A hypoplastic aortic arch (*A*, *red*) also is shown

Bilateral PDA

Rarely, a PDA may be separate and bilateral, arising from the left brachiocephalic artery and aorta. In these cases, the patient typically has complex congenital heart disease with several other anomalies.

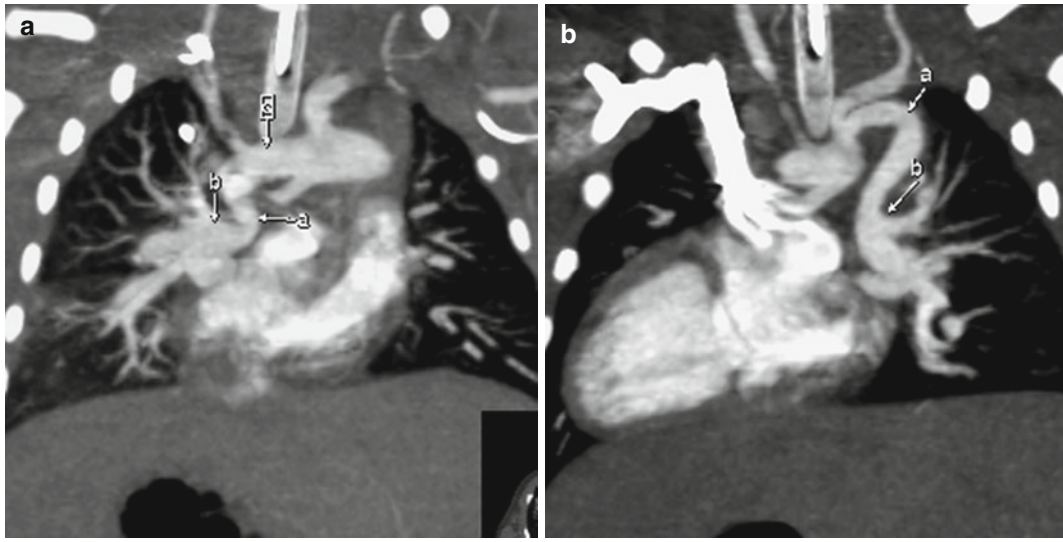


Fig. 7.6 (**a**) MIP image from a cardiac CT demonstrating a PDA (*a*) extending from the aortic arch (*c*) to give rise to the right pulmonary artery (*b*). (**b**) Coronal MIP image showing a second, tortuous PDA (*a*) from the descending aortic arch, giving rise to the left pulmonary artery (*b*)

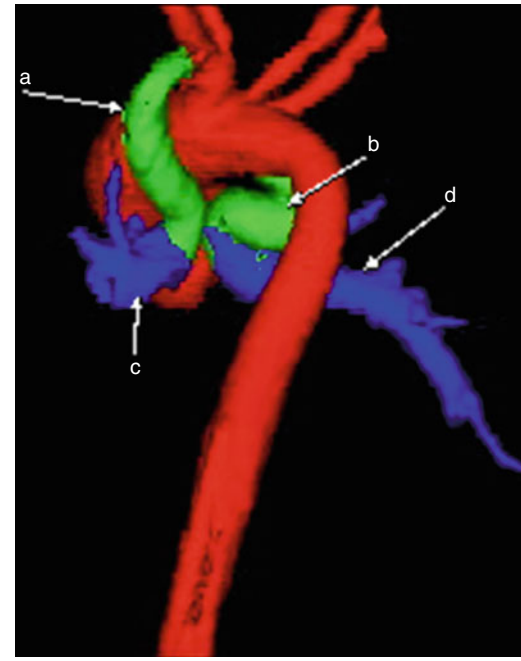


Fig. 7.7 Color-coded 3D image from a cardiac CT scan showing bilateral PDAs (*a* and *b*) giving rise to the right (*d*) and left (*c*) pulmonary arteries

Aortic Atresia

Aortic atresia is part of the hypoplastic left heart syndrome and involves hypoplasia/atresia of the ascending aorta, aortic valve, left ventricle, and mitral valve. Patients depend on a PDA for survival; death occurs within days (as the PDA closes) if the condition is untreated. Patients have cyanosis, with chest radiography demonstrating cardiomegaly and pulmonary venous congestion. Echocardiography, CT, MRI, and angiography may be used for treatment planning. A variety of surgical options exist.

In aortic atresia, the coronary arteries receive retrograde blood flow from the PDA up to the aortic arch and then down the coronary arteries via a hypoplastic ascending aorta. The brain and upper extremities also depend on the retrograde flow from the PDA.

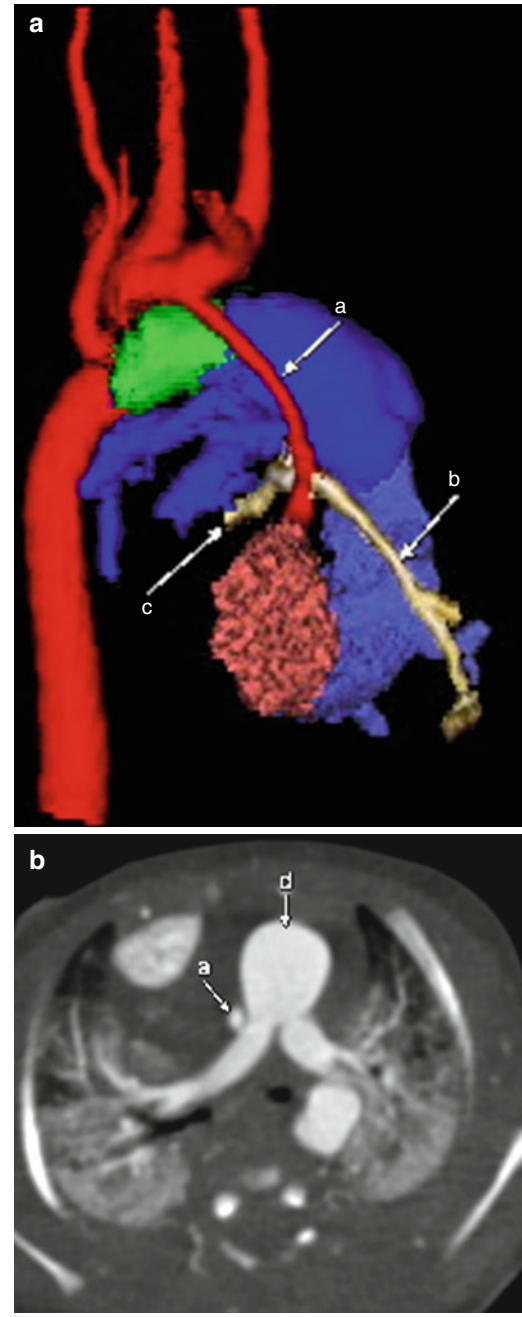


Fig. 7.8 3D color-coded (**a**) and axial (**b**) images demonstrating hypoplasia of the ascending aorta (*a*). The main pulmonary artery (*d*, *blue*) provides blood flow through a PDA (*green*), allowing retrograde flow to the great vessels coming off the arch (*red*) and the coronary arteries (*tan*, *b* and *c*)

Aortic Stenosis

Aortic stenosis may be valvular, subvalvular, or supravalvular (Williams syndrome). In neonates/infants, chest radiography is normal or shows mild cardiomegaly and pulmonary edema. In children/adolescents, chest radiography is often normal, even if the aortic stenosis is severe. Patients with aortic stenosis have thickening with fusion of the aortic valve leaflets, poststenotic dilatation of the ascending aorta, a systolic flow jet into the ascending aorta, and left ventricular hypertrophy. MRI and echocardiography may be used to assess the transvalvular pressure gradient, the regurgitant fraction, and ventricular function.

Valvular Aortic Stenosis

Valvular aortic stenosis, reported in 1–2 % of the population, most commonly is the result of a bicuspid aortic valve, although bicuspid aortic valves do not always cause significant stenosis. It is more common in males and has a familial predilection.

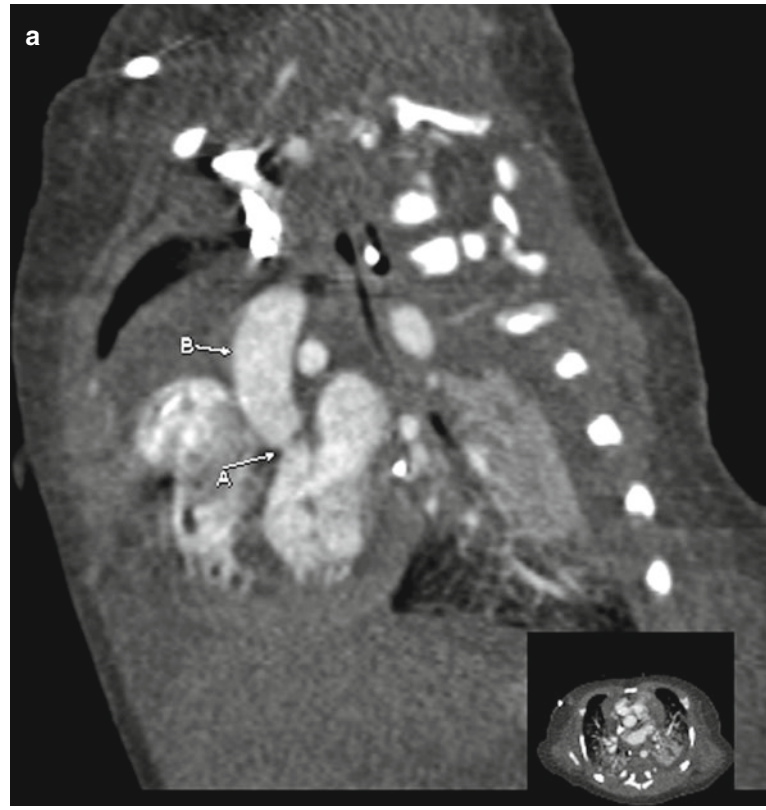


Fig. 7.9 Sagittal (**a**) and axial (**b**) contrast-enhanced cardiac CT images demonstrating narrowing of the aorta at the level of the aortic valve (*A*) with thickening of the bicuspid valve leaflets (*A*). Poststenotic dilatation of the ascending aorta (*B*) also is seen

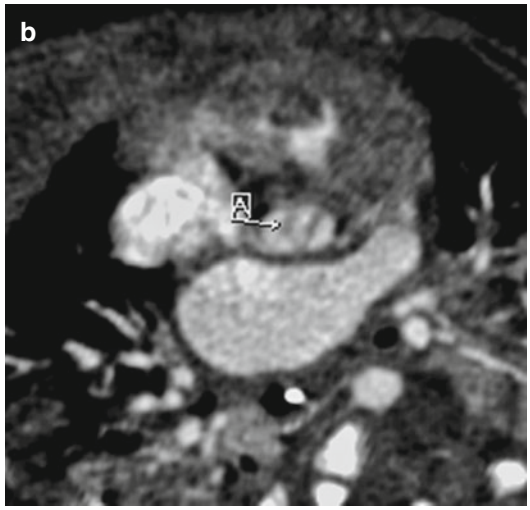


Fig. 7.9 (continued)

Supravalvular Aortic Stenosis (Williams Syndrome)

Supravalvular aortic stenosis occurs above the aortic valve, giving the proximal aorta an "hourglass" appearance. It is associated with stenosis of other vessels, such as the pulmonary and coronary arteries and descending abdominal aorta. Children with this disease may have elf-like facial features and mental retardation.

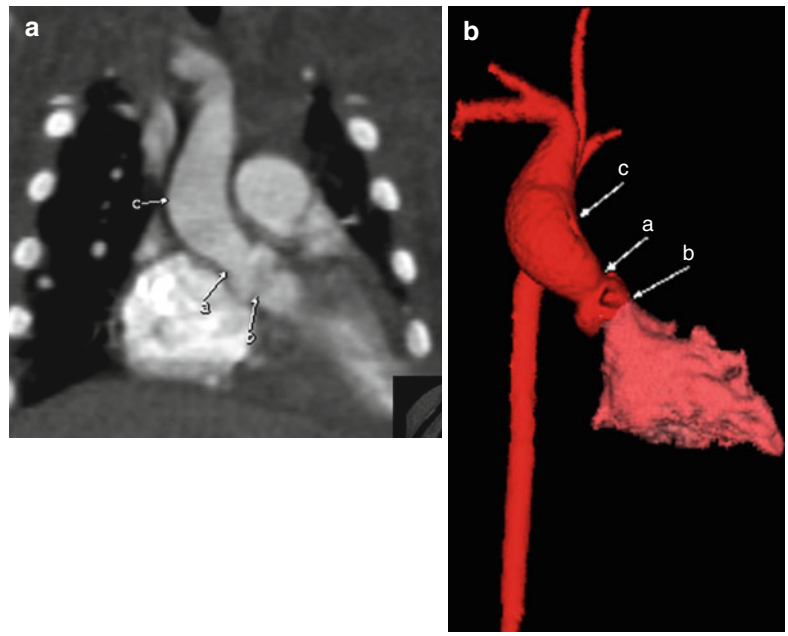


Fig. 7.10 Coronal MIP (**a**) and color-coded 3D (**b**) images from a cardiac CT scan in a patient with Williams syndrome. The images show supravalvar aortic stenosis (*a*) with poststenotic dilatation (*c*) of the aorta. Notice that the subvalvular region is of normal caliber (*b*). The contours of the ascending aorta often are described as hourglass shaped

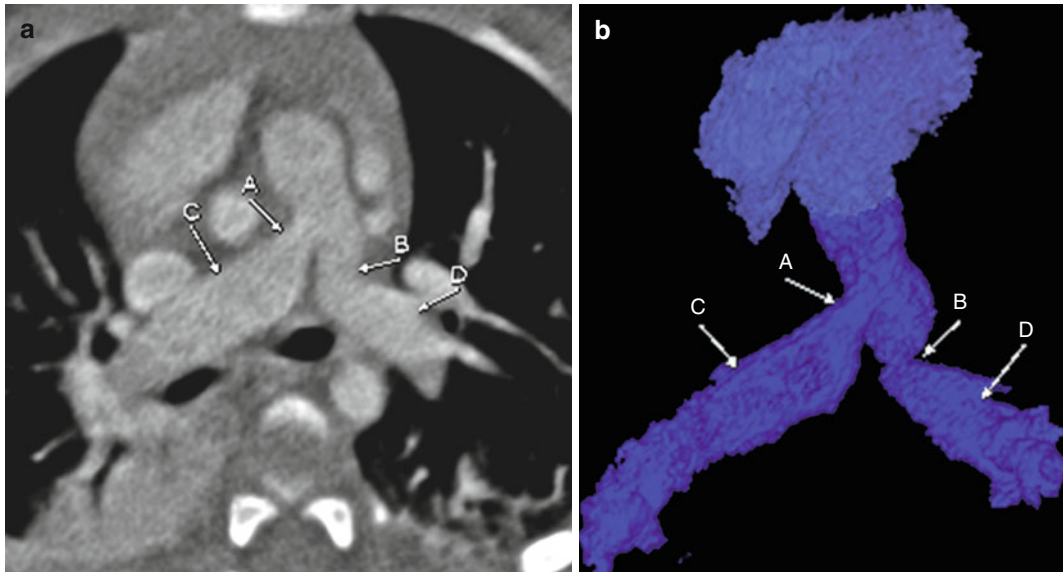
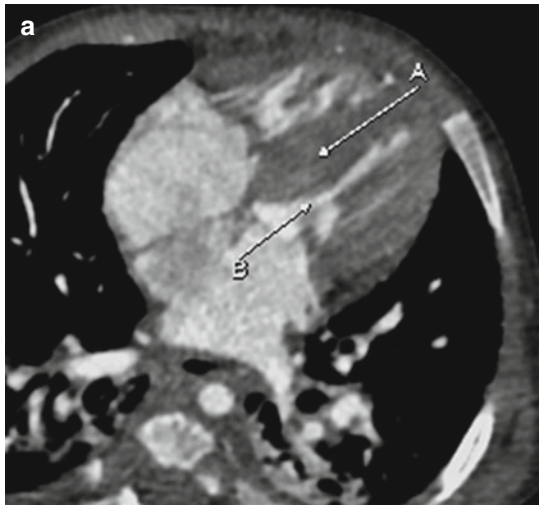


Fig. 7.11 Axial (**a**) and color-coded 3D (**b**) images in a patient with known Williams syndrome. The images demonstrate stenosis of the proximal left (*B*) and right (*A*) pulmonary arteries with associated poststenotic dilatation (*C* and *D*). Pulmonic stenosis is a common finding in patients with Williams syndrome

Hypertrophic Cardiomyopathy with Subaortic Stenosis

Hypertrophic cardiomyopathy with subaortic stenosis, the most common form of cardiomyopathy in children, is marked by thickening of the interventricular septum, causing left ventricular outflow tract obstruction (LVOT). The etiology may be idiopathic. Infants of diabetic mothers also may develop significant hypertrophy of the interventricular septum and may have subvalvular stenosis.



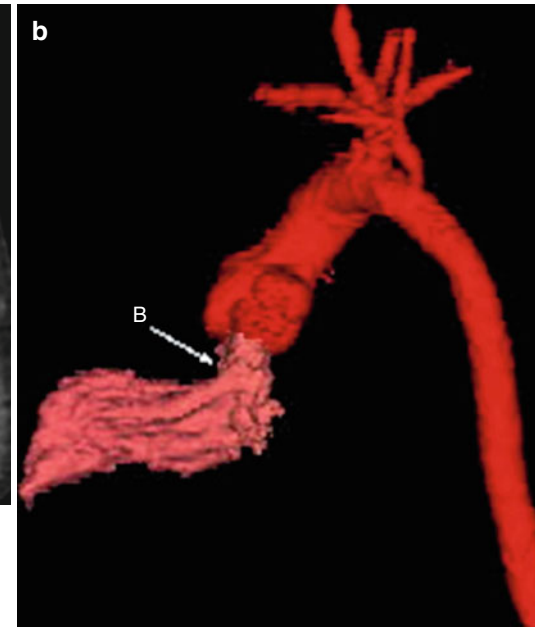


Fig. 7.12 Axial contrast-enhanced CT image through the heart (**a**) and corresponding color-coded 3D image (**b**) demonstrating severe thickening of the myocardial septum (*A*) and the free wall of the left ventricle, which results in narrowing of the aortic outflow tract (*B*) in the subvalvular region

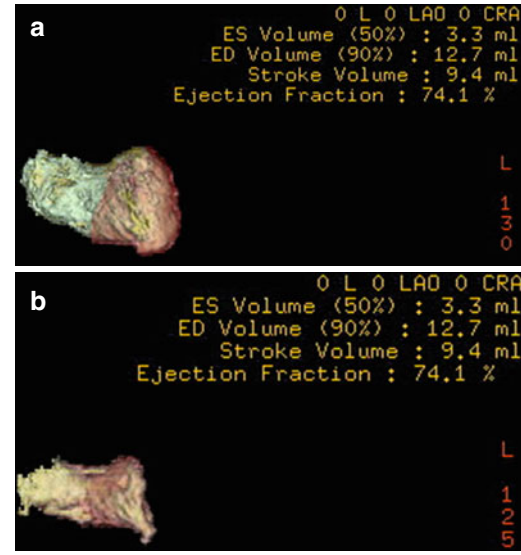


Fig. 7.13 Functional end-diastolic (**a**) and end-systolic (**b**) images from a cardiac CTA scan showing hyperdynamic function of the left ventricle with an ejection fraction of 74.1 % in a patient with hypertrophic cardiomyopathy

Pulmonic Stenosis

Pulmonic stenosis may be valvular (>90 % of cases) or supravalvular, or occur at the branches of the distal pulmonary arteries. Patients with this disease have a thickened, stenotic valve with poststenotic dilatation of the pulmonary artery. There is associated right ventricular hypertrophy. Heart size is normal, with a dilated main pulmonary artery segment. The clinical presentation is determined by the severity of stenosis and ranges from asymptomatic to severely cyanotic in infancy. Pulmonic stenosis is associated with Noonan syndrome, Williams syndrome, tetralogy of Fallot (TOF), Ellaville syndrome, and congenital rubella.

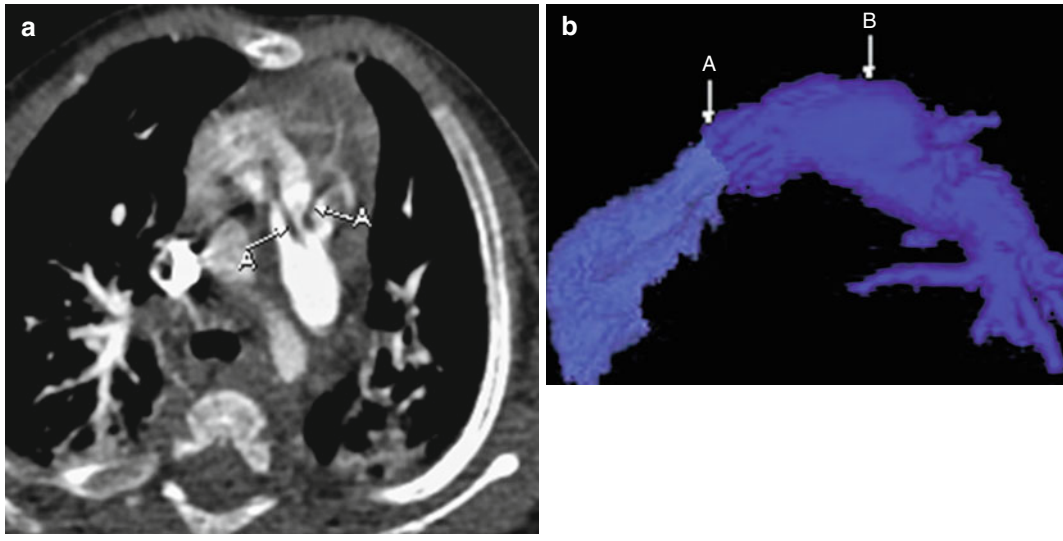


Fig. 7.14 Axial MIP (**a**) and color-coded (**b**) images from a cardiac CT scan demonstrating thickening and stenosis of the pulmonic valve (*A*) with associated poststenotic dilatation of the main pulmonary artery (*B*)

Dextro-Type Transposition of the Great Vessels

In dextro-transposition of the great vessels (DTGA), the aorta arises from the right ventricle and the pulmonary artery from the left ventricle. There is ventriculoarterial discordance with atrioventricular concordance. A shunt (PDA, VSD, atrial septal defect [ASD]) to mix oxygenated and deoxygenated blood is necessary for survival. The aortic valve is anterior to and to the left of the pulmonic valve. Chest radiography demonstrates a narrow mediastinum, cardiomegaly ("egg on a string"), and increased pulmonary vascularity. The right coronary artery typically arises from the noncoronary sinus in patients with DTGA. The coronary arteries typically arise from "facing" sinuses, meaning they arise from the coronary sinuses (noncoronary and left) facing the pulmonary artery.

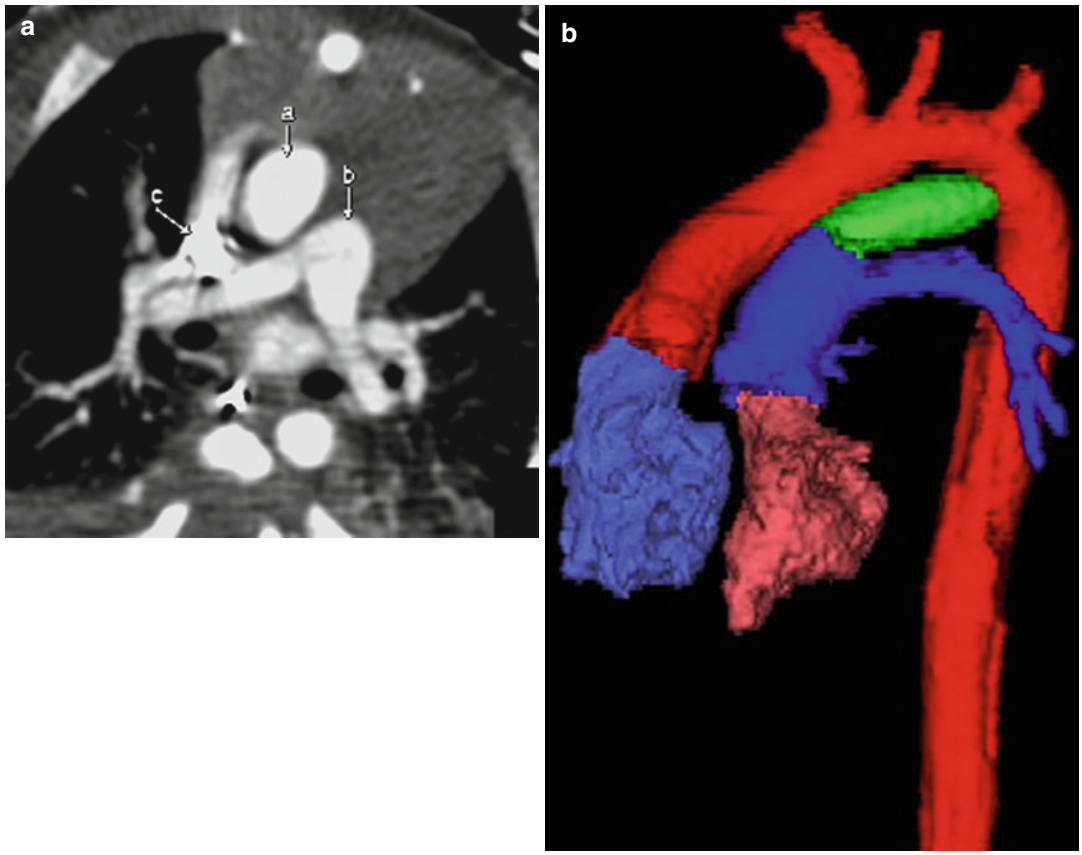


Fig. 7.15 (**a**) Axial MIP image from cardiac CT demonstrating the aortic root (*a*) anterior to and to the right of the pulmonary outflow tract (*b*). The SVC (*c*) is in the normal position. (**b**) Lateral view of a color-coded 3D image from a cardiac CT scan demonstrating the aorta (*red*) arising from the right ventricle (*purple*) while the pulmonary artery (*blue*) arises from the left ventricle (*salmon*) in this patient with DTGA. A PDA (*green*) is seen connecting the pulmonary artery (*blue*) to the aorta (*red*)

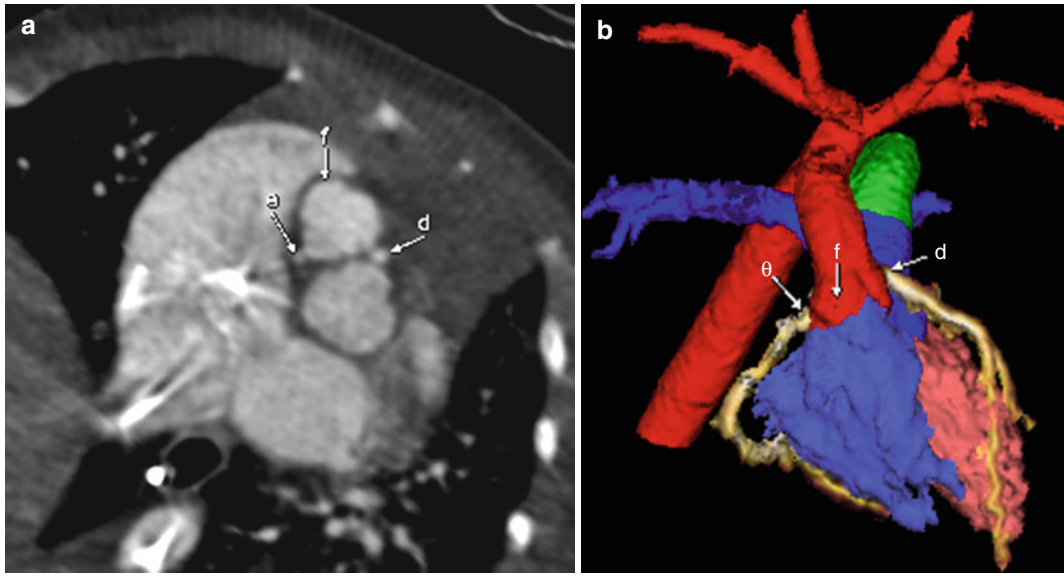
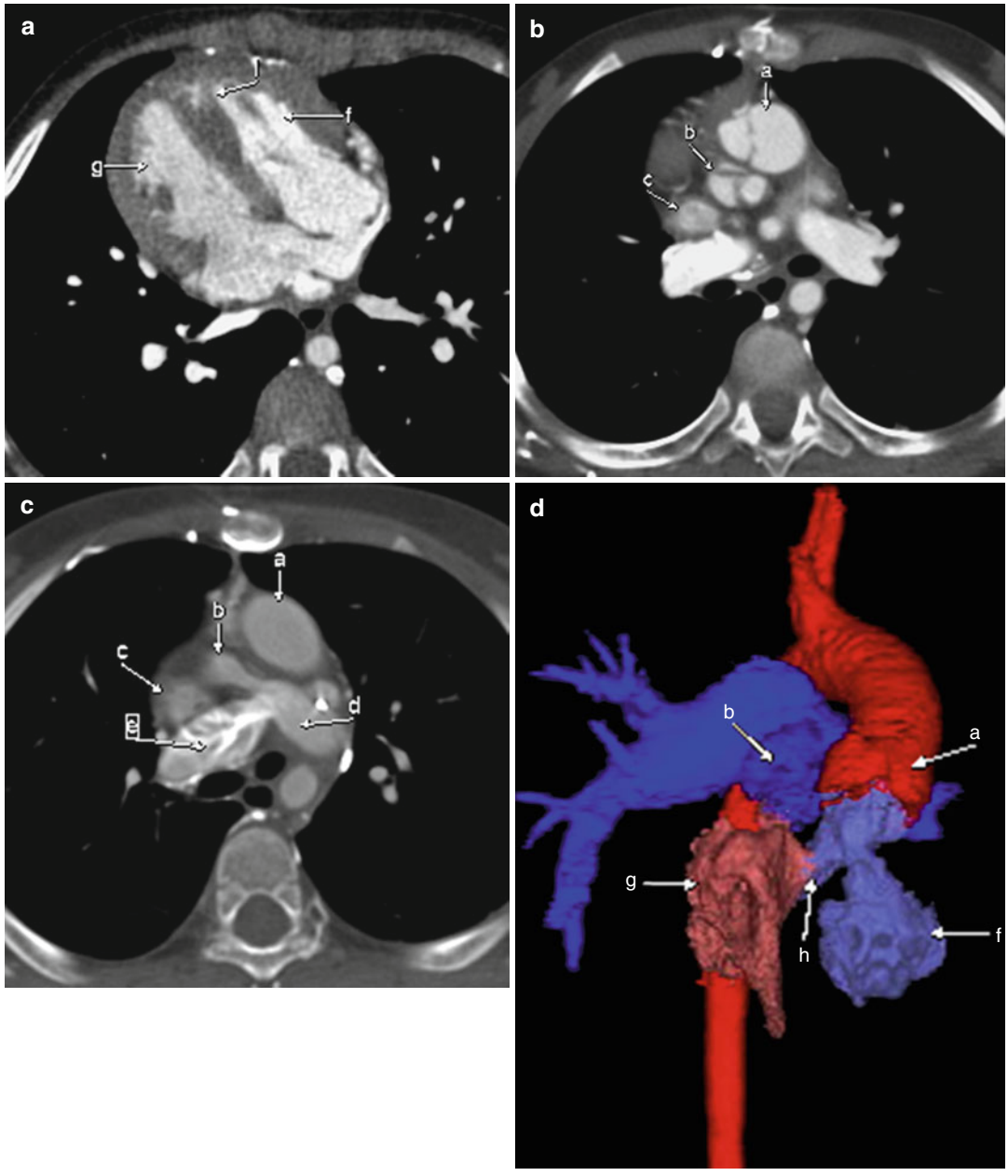


Fig. 7.16 Axial MIP (**a**) and color-coded 3D (**b**) images from a cardiac CT scan in a patient with DTGA showing the right coronary artery (*e*) arising from the noncoronary sinus and the left coronary artery (*d*) arising from the left coronary sinus. Notice that the right coronary sinus (*f*) is free of any coronary artery origin

Levo-Transposition of the Great Vessels

In levo-transposition, the great vessels and ventricles are inverted and there is atrioventricular and ventriculoarterial discordance. Right-sided blood flow is as follows: inferior/superior vena cava → right atrium → mitral valve → morphologic left ventricle (on right) → pulmonary circulation. Left-sided flow is as follows: pulmonary veins → left atrium → tricuspid valve → morphologic right ventricle (on left) → systemic circulation. The term *congenitally corrected transposition* is applied if there are no associated abnormalities (1 % of cases). The most common associated abnormalities include VSD (60–70 %) and LVOT obstruction (30–50 %). Chest radiography often demonstrates a straight left heart border.



Double-Outlet Right Ventricle

In double-outlet right ventricle, the aorta and pulmonary artery arise completely or predominantly from the morphologic right ventricle. It often is part of complex heart disease. There are 16 variations based on the positioning of the great arteries and VSD; the most common is subaortic VSD with normal positioning of the great vessels. The radiographic appearance depends on the physiology of the lesion. Patients with double-outlet right ventricle have mild cardiomegaly and decreased pulmonary vasculature, often similar in appearance to TOF.

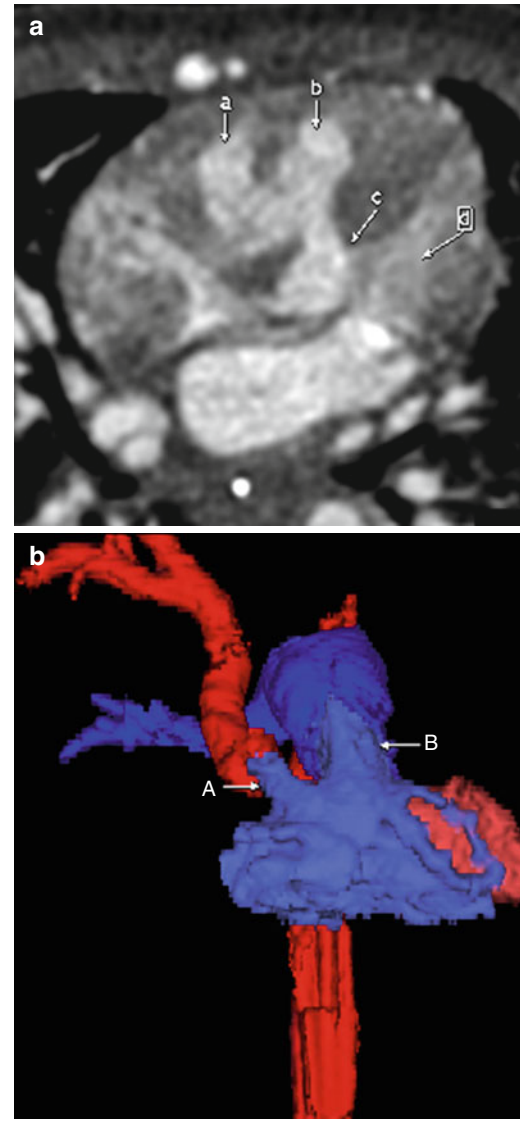
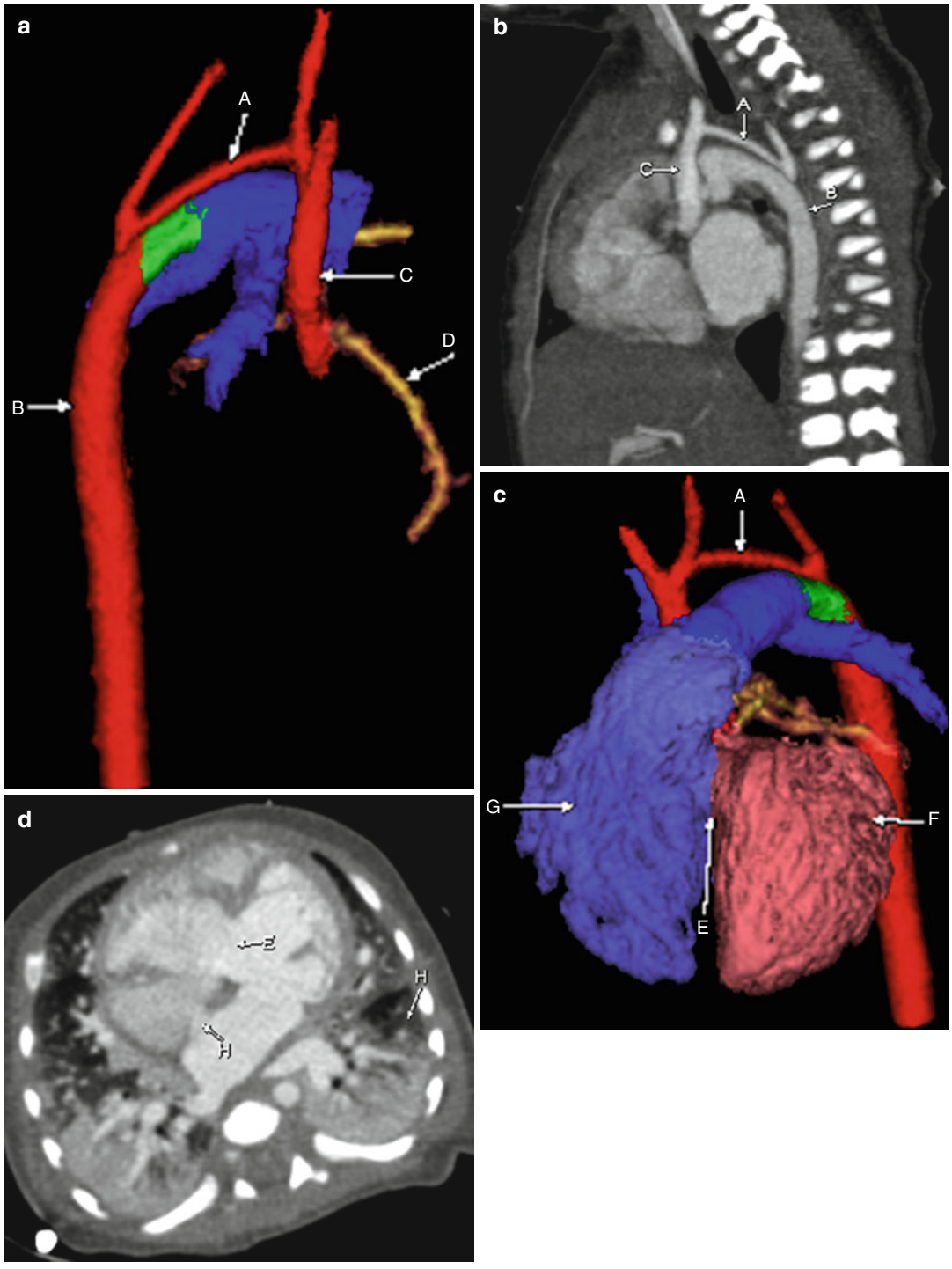


Fig. 7.18 Axial MIP image from cardiac CT (**a**) and anterior view of a color-coded 3D reconstruction (**b**) demonstrating the aorta outflow (*a*, *A*) to the right of the main pulmonary outflow (*b*, *B*), with both arising from the right ventricle (*purple*). A VSD (*c*) also is seen connecting the right and left (*d*) ventricles

Fig. 7.17 Three contrast-enhanced axial cardiac CT images (**a**–**c**) and corresponding color-coded 3D image (**d**) showing the morphologic left ventricle (*g*) positioned on the right with a relatively smooth wall. The morphologic right ventricle (*f*) is on the left and can be identified by its trabeculated wall and moderator band (*l*). The aorta (*a*) arises from the morphologic right ventricle and lies anterior to and to the left of the pulmonary artery (*b*), which arises from the morphologic left ventricle. The right (*e*) and left (*d*) pulmonary arteries, along with the superior vena cava (*c*), are seen on the axial images. The 3D image demonstrates a VSD (*h*), which is present in 60–70 % of levo-transposition cases

Hypoplastic-Type Coarctation of the Aorta

Hypoplastic coarctation of the aorta also has been referred to as *preductal* or *infantile-type coarctation*. In infants with this condition, no significant collaterals have had time to develop. Patients with severe coarctation often present in infancy with heart failure once the PDA closes. Heart size is normal in these patients as long as the ventricular hypertrophy can overcome the stenosis.



Focal-Type Coarctation of the Aorta

In focal coarctation, there is focal obstruction of the descending aortic arch, which may be preductal, juxtaductal, or postductal. This condition typically presents in older children and adolescents. Collateral vessels are common. Chest radiography demonstrates a normal heart size and pulmonary vasculature with dilatation of the descending aorta from poststenotic dilatation. On plain x-ray film, this forms a figure "3" sign. Rib notching is seen from the large collateral vessels.

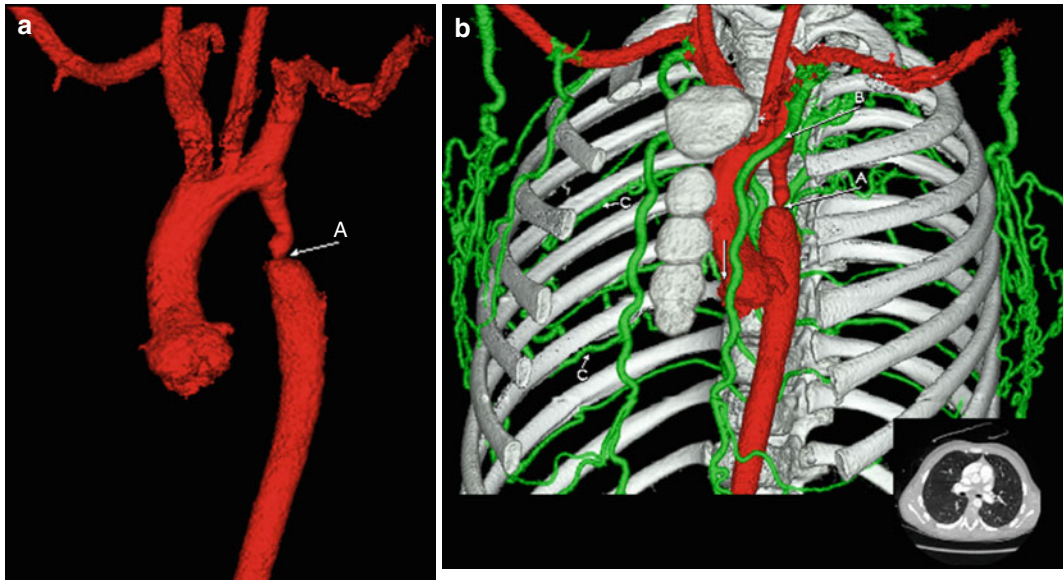


Fig. 7.20 Color-coded 3D (**a** and **b**) and sagittal (**c**) MIP images from cardiac CT showing a focal stenosis (*A*) of the descending aortic arch with multiple enlarged collateral vessels, including internal mammary (*B*) and intercostal arteries (*C*)

Fig. 7.19 Lateral (**a**) and oblique (**c**) projection color-coded 3D models and sagittal (**b**) and axial (**d**) MIP images from a cardiac CTA scan demonstrating a severely hypoplastic-type coarctation (*A*) of the transverse aortic arch proximal to the origin of the left subclavian artery. Notice that the descending aorta (*B*) is fed by a PDA (*green*). There is some hypoplasia of the ascending aorta (*C*), with the right coronary artery (*D*) arising from the right coronary sinus. A large VSD (*E*) is noted between the right (*G*) and left (*F*) ventricles, with a secundum-type ASD (*H*, heart) also seen. There is massive cardiomegaly and bilateral pulmonary edema (*H*, lungs), as this patient is in heart failure

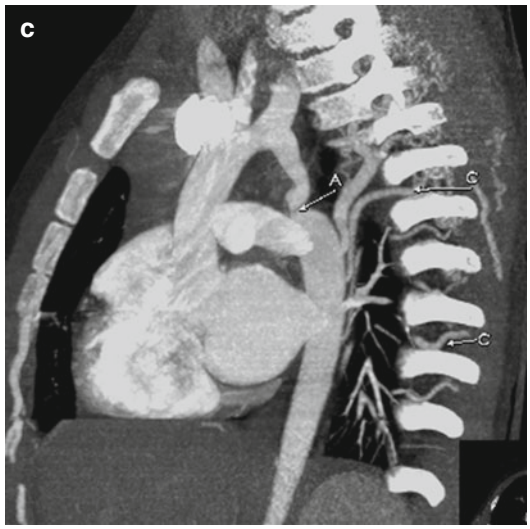


Fig. 7.20 (continued)

Pulmonic Atresia

Pulmonic atresia is marked by an imperforate pulmonary valve due to abnormal formation. A PDA or collaterals are necessary to provide blood flow to the pulmonary vessels. Patients with this condition have right ventricular hypoplasia with associated coronary artery abnormalities.

Pulmonic atresia may occur with or without a VSD; with a VSD, it is considered the most severe form of TOF. Imaging shows cardiomegaly and normal pulmonary vasculature.

Pulmonic Atresia with an Intact Ventricular Septum and Right Ventricular Hypoplasia

In patients with pulmonic atresia with an intact ventricular septum and right ventricular hypoplasia, the right ventricle may be severely hypoplastic. Coronary artery fistulas (sinusoids) frequently are present. A PDA must be present with an ASD or a patent foramen ovale.

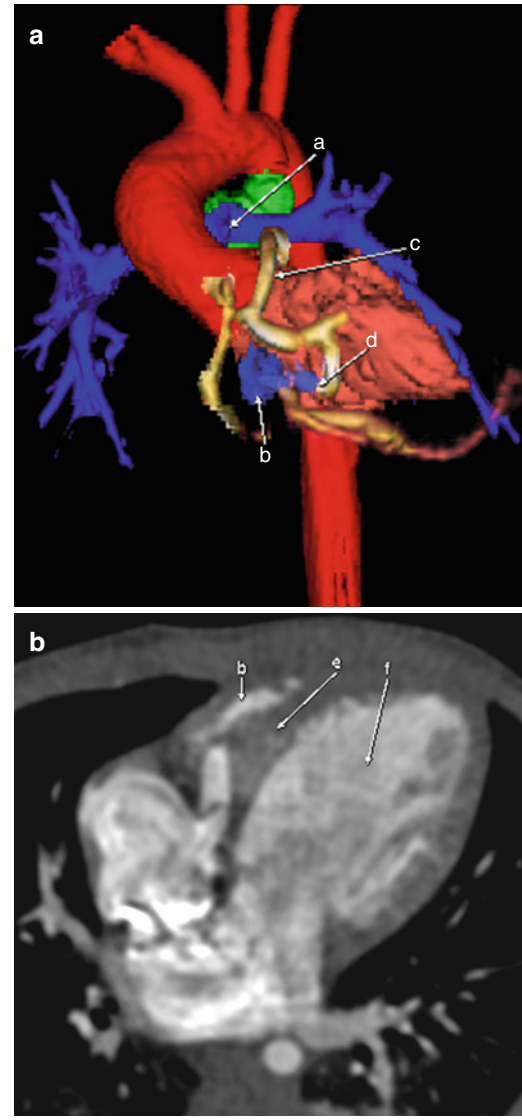


Fig. 7.21 Frontal color-coded 3D (**a**) and axial (**b**) images from a cardiac CT scan demonstrating right and left pulmonary arteries (*blue*) without direct attachment to the hypoplastic right ventricle (*b*) because of the long atretic segment (*a*) of the pulmonary artery. The left coronary artery is enlarged (*c*) and forms a fistula with the right ventricle (*d*). Notice that the interventricular septum is intact (*e*) and the left ventricle is enlarged (*f*)

Pulmonic Atresia with VSD, Type 1

In type 1 pulmonic atresia with VSD, most of the flow goes through the PDA to the pulmonary arteries. Some small collaterals may be present. Closure of the PDA is life threatening.

Fig. 7.22 3D color-coded image from a cardiac CT scan showing a VSD (*B*) connecting the left (*arrow*) and right (*purple*) ventricles with severe pulmonary artery stenosis (*A*). The pulmonary arteries receive blood flow from the PDA (*green*), and no other significant collateral flow is present

Pulmonic Atresia with VSD, Type 2

In type 2 pulmonic atresia with VSD, the flow from the pulmonary arteries and aortopulmonary collaterals is approximately equal. A PDA may be present. The pulmonary arteries are smaller than normal.

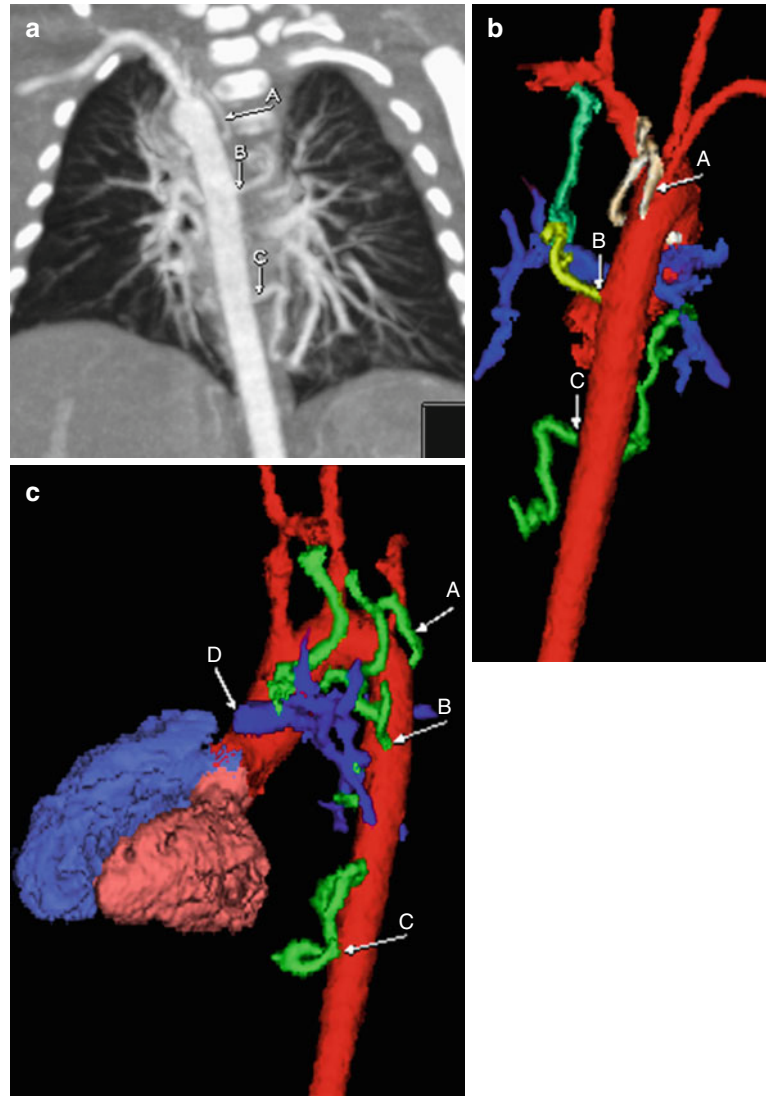


Fig. 7.23 Coronal MIP image (**a**) along with anterior (**b**) and lateral (**c**) color-coded 3D images from cardiac CT demonstrating multiple systemic-to-pulmonary collaterals (*A*, *B*, and *C*), which can provide blood flow to the pulmonary vasculature. The atretic segment is missing (*D*) between the main pulmonary artery and the right ventricle. Both native pulmonary arteries are only mildly hypoplastic

Pulmonic Atresia with VSD, Type 3

In type 3 pulmonic atresia with VSD, most of the pulmonary blood flow comes from the aortopulmonary collaterals. The pulmonary arteries are markedly hypoplastic and often difficult to identify.

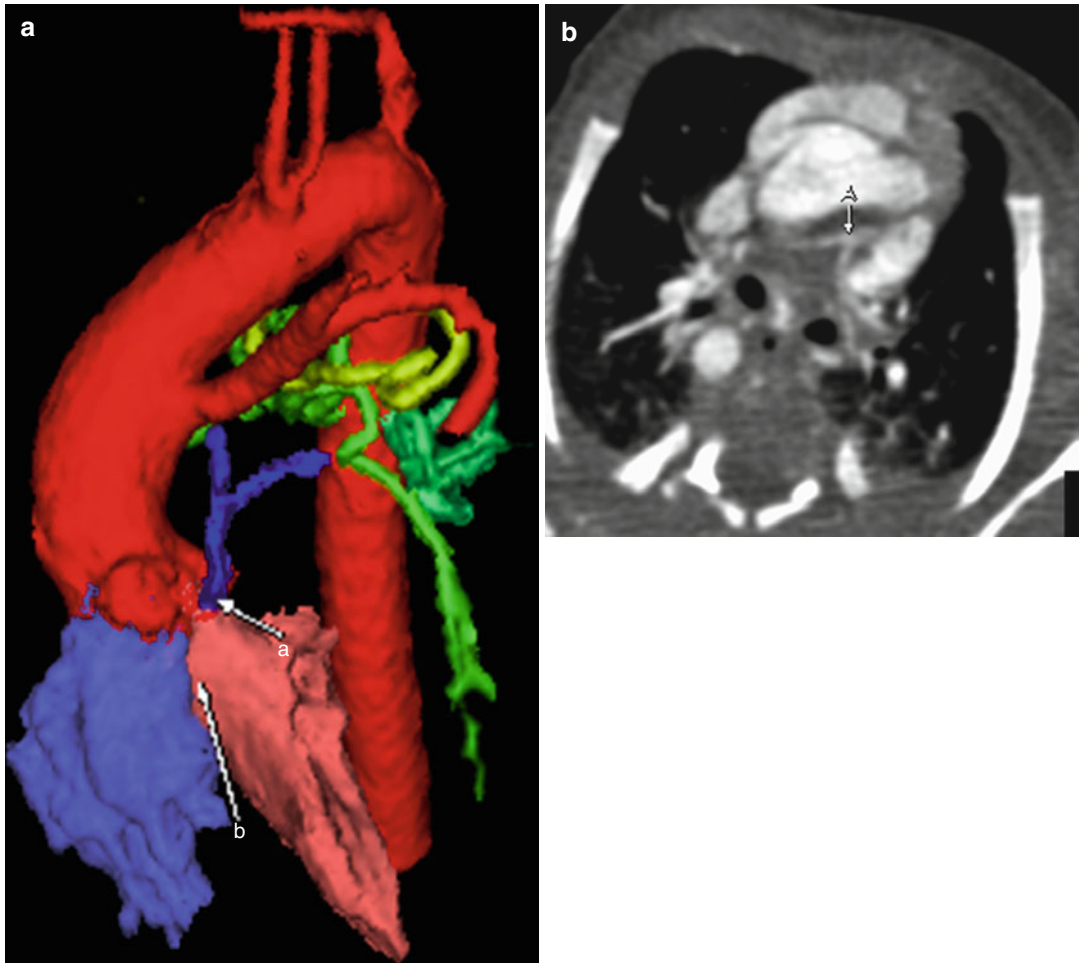


Fig. 7.24 Color-coded 3D (**a**) and axial contrast-enhanced (**b**) images from cardiac CT demonstrating markedly hypoplastic pulmonary arteries (*A*; *a*, *blue*) with an atretic proximal segment. Multiple collaterals (*green*, *yellow*) are necessary to provide blood flow to the lungs. A VSD (*b*) is shown

Interruption of the Aortic Arch

Interruption of the aortic arch is the absence or discontinuation of a portion of the aortic arch. Different types exist based on the location of the interruption: distal to the left subclavian artery origin (type A), between the left common carotid and left subclavian arteries (type B), or between the innominate and left common carotid arteries (type C). A PDA is necessary to oxygenate the lower extremities and abdominal organs. This condition often is associated with a VSD.

Interruption of the Aortic Arch, Type A

Type A interruption of the aortic arch occurs between the left subclavian artery and the descending aorta. Severe coarctation may be misinterpreted as this type of interruption.

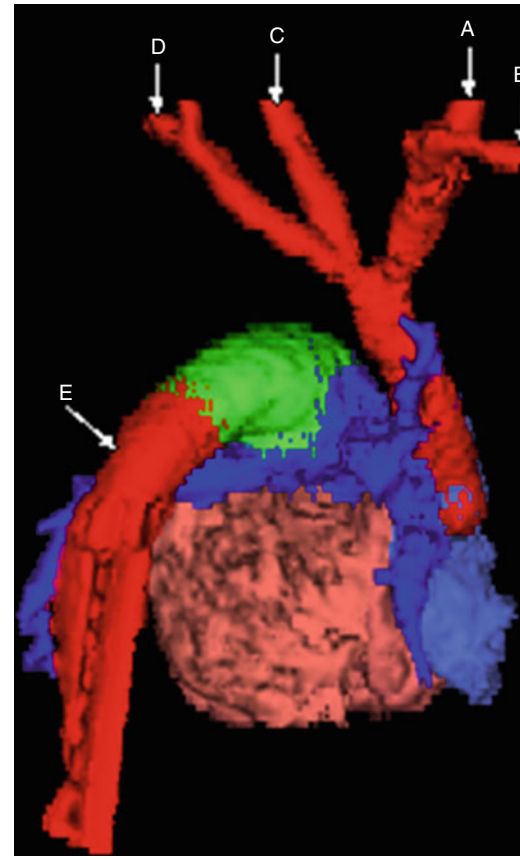


Fig. 7.25 Color-coded 3D image from cardiac CT demonstrating an interruption of the aortic arch distal to the left subclavian artery (*D*). The descending thoracic aorta is fed from a large PDA (*green*). The right common carotid (*A*), right subclavian (*B*), and left common carotid (*C*) arteries also are seen. Notice that the descending aorta (*E*) is supplied by the PDA (*green*)

Interruption of the Aortic Arch, Type B

Type B interruption is defined as an interruption of the aortic arch distal to the left common carotid artery and proximal to the left subclavian artery. It is the most common type of aortic arch interruption.

Interruption of the Aortic Arch, Type C

Type C interruption is defined as an interruption of the aortic arch between the brachiocephalic and left common carotid arteries. It is the least common type of aortic arch interruption.

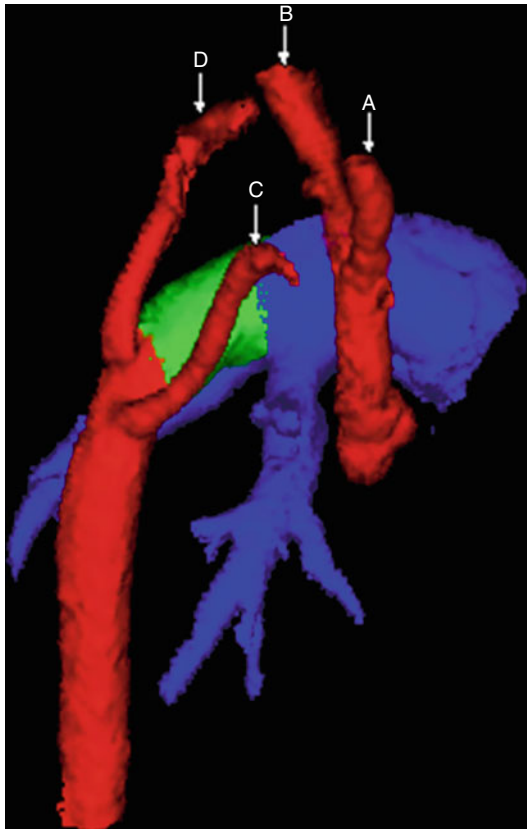


Fig. 7.26 Color-coded 3D image from a cardiac CT scan showing interruption of the aortic arch (*type B*) between the left common carotid (*B*) and left subclavian (*D*) arteries. Other vessels include the right common carotid (*A*) and aberrant right subclavian (*C*) arteries. Notice that the descending aorta and bilateral subclavian arteries (*D* and *C*) are supplied by the PDA (*green*)

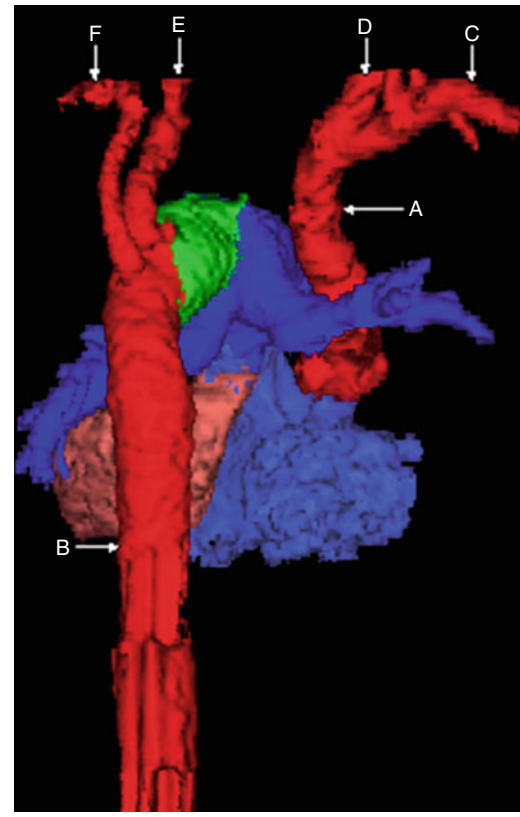


Fig. 7.27 3D color-coded image from cardiac CT demonstrating an interruption of the aortic arch (*type C*) between the innominate (*A*) and left common carotid (*E*) arteries. The left subclavian (*F*), right subclavian (*C*), and right common carotid (*D*) arteries also are displayed. Note that the PDA (*green*) gives rise to the descending thoracic aorta (*B*)

Truncus Arteriosus

In truncus arteriosus, a common arterial vessel arises from the heart and gives rise to the aorta, pulmonary artery, and coronary arteries. It is associated with right aortic arch in 30–40 % of cases. A high VSD exists immediately below the truncal valve. Imaging reveals cardiomegaly, increased pulmonary vascularity, a narrow mediastinum, and a right aortic arch. If untreated, truncus arteriosus leads to intractable heart failure. Classification schemes include the Van Praagh and the Collette and Edwards classifications.

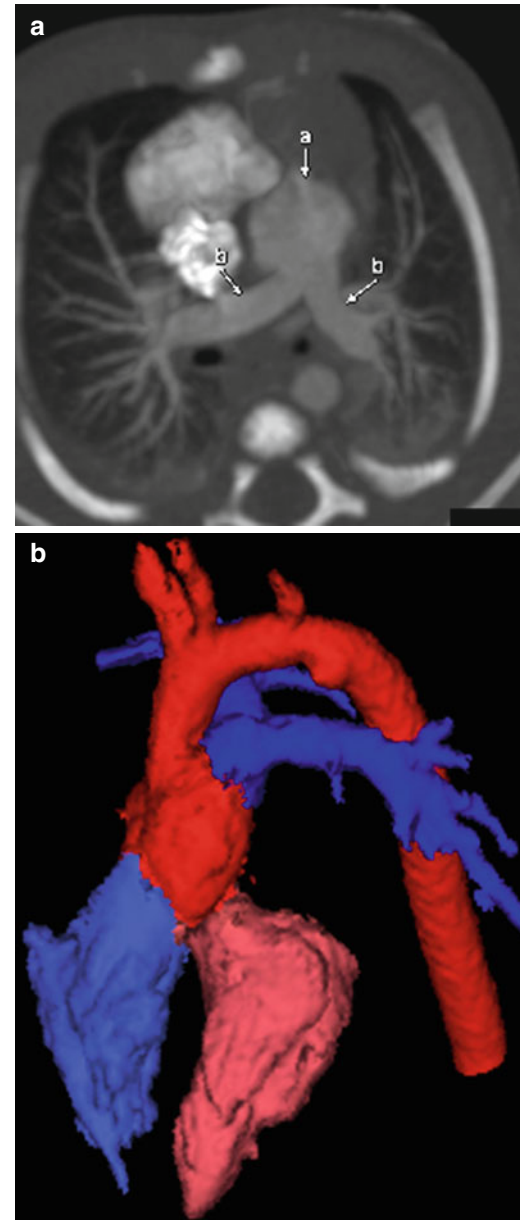


Fig. 7.28 Axial MIP (**a**) and lateral color-coded 3D (**b**) images from cardiac CT demonstrating a common truncus (*a*) giving rise to the right and left pulmonary arteries (*b*, *blue*) and the aorta (*red*)

Hemitruncus

In hemitruncus, one pulmonary artery arises from the aorta and the other is a continuation of the main pulmonary artery. Patients develop heart failure from the large left-to-right shunt. Chest radiography reveals increased pulmonary vascularity and cardiomegaly.

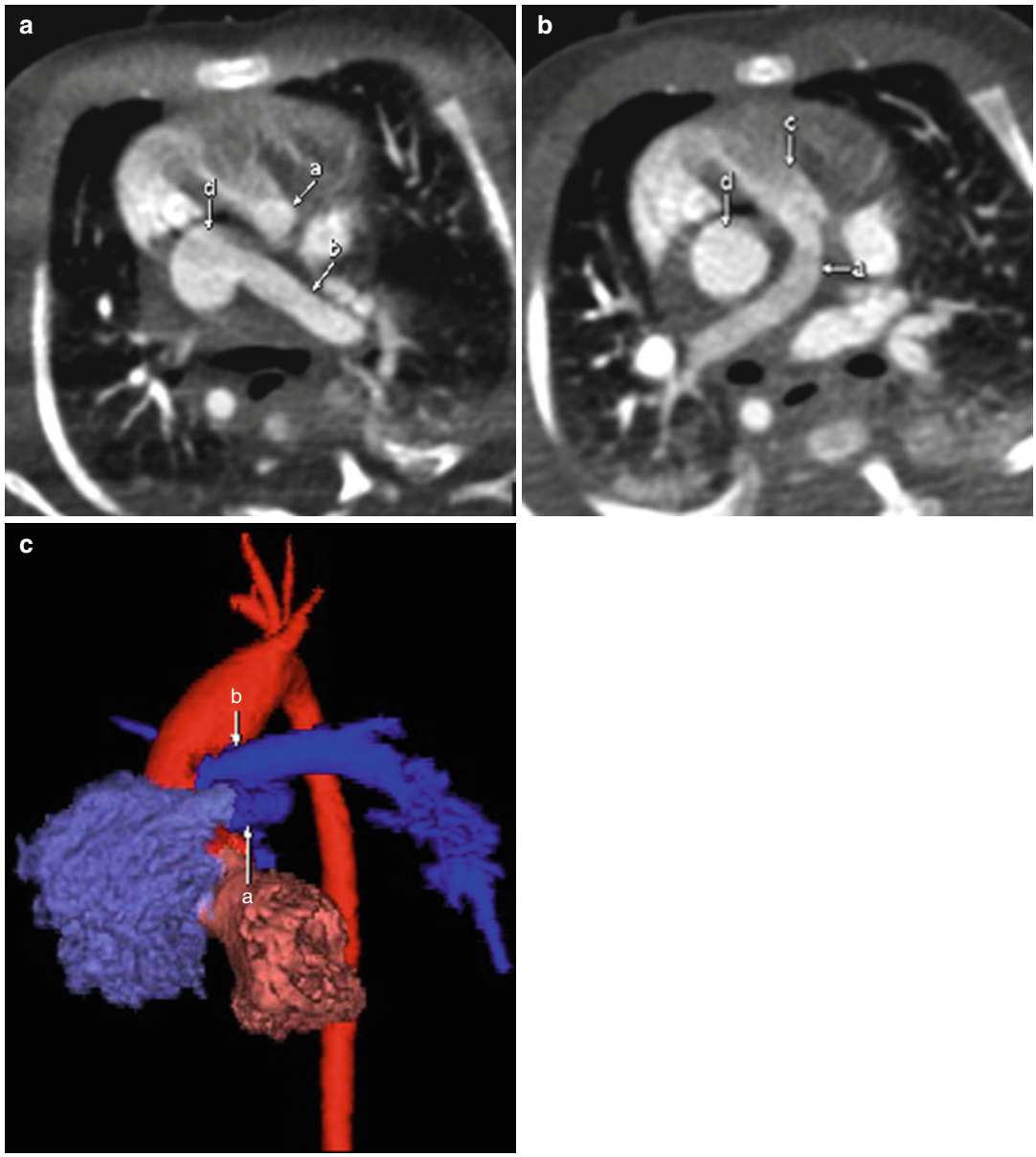


Fig. 7.29 Axial contrast-enhanced (**a** **b**) and anterior-view color-coded 3D (**c**) images from a cardiac CT scan demonstrating a right ventricular outflow tract (*c*) giving rise to the right pulmonary artery (*a*). The left pulmonary artery (*b*) arises from the ascending aorta (*d*, *red*). This combination is known as a hemitruncus with only half the pulmonary supply coming from the aorta

Pseudotruncus Arteriosus

Pseudotruncus arteriosus is a usually severe form of TOF or pulmonic atresia in which most of the blood flow comes from the aortopulmonary collaterals. In addition to the collaterals, one should look for typical TOF findings: a VSD, right ventricular hypertrophy, an overriding aorta, and pulmonic stenosis.

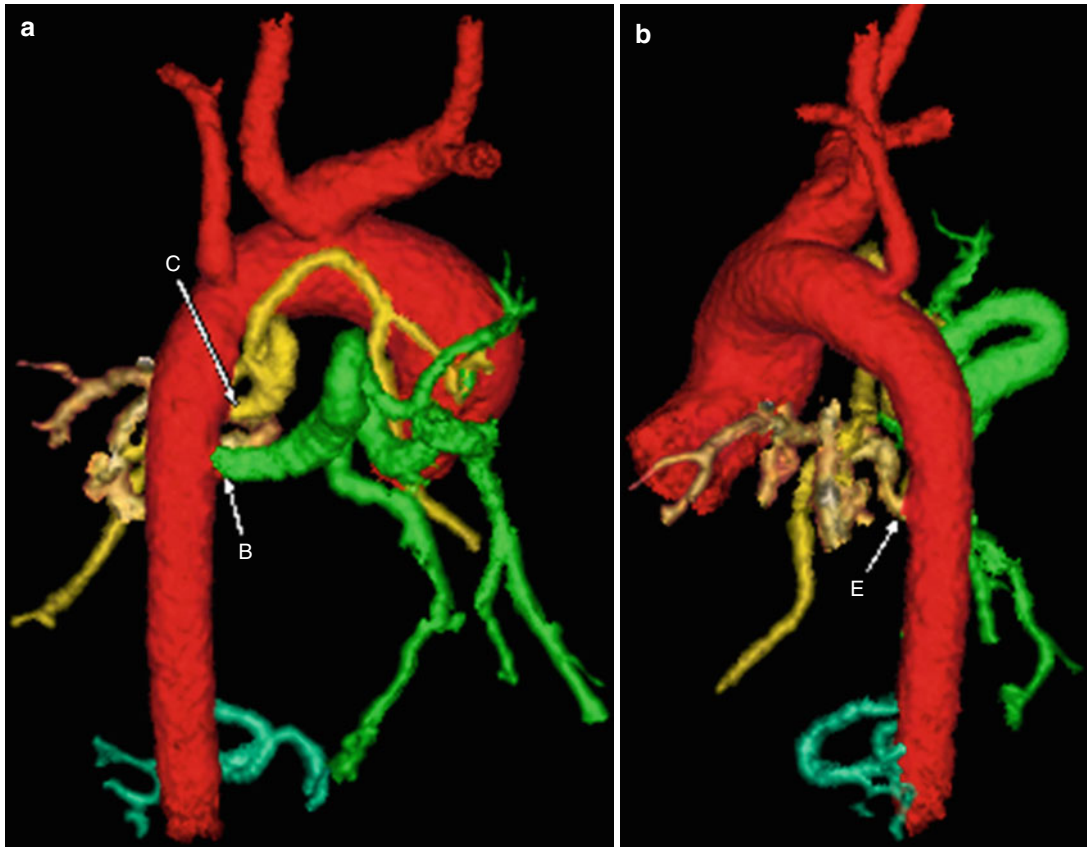
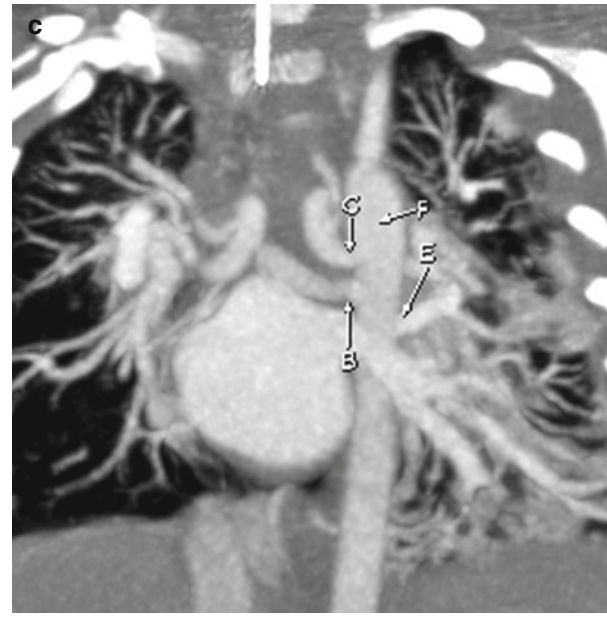


Fig. 7.30 Color-coded 3D (**a** and **b**) and coronal MIP (**c**) images from cardiac CT showing no pulmonary arteries. Instead, all the pulmonary vascular flow is provided by multiple collaterals (*B*, *C*, *E*, and *F*) originating from the descending thoracic aorta

Fig. 7.30 (continued)



Aortopulmonary Window

Aortopulmonary window is the failure of the conotruncal ridges to fuse. Unlike in truncus arteriosus, the pulmonary and aortic valves are normal, with normal proximal aortic and pulmonic outflow tracts. Chest radiography may demonstrate cardiomegaly and increased pulmonary vascularity. A variety of clinical presentations exist depending on the size of the lesion.

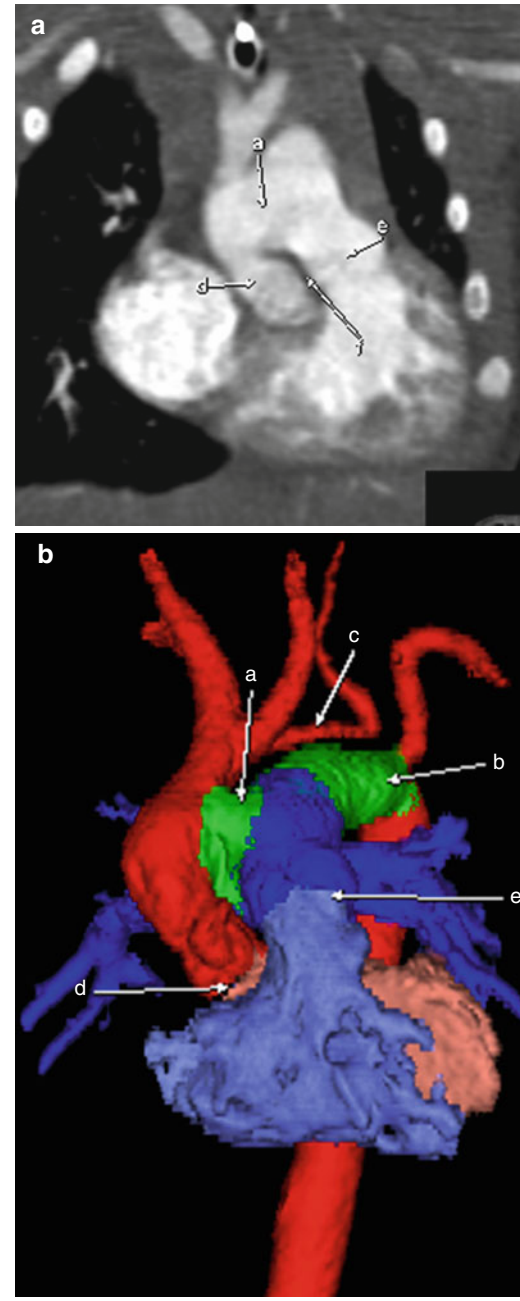


Fig. 7.31 Coronal MIP (**a**) and color-coded 3D (**b**) images from a cardiac CT scan showing an aortopulmonary window lesion (*a*) between the aorta and main pulmonary artery. The pulmonary (*e*) and aortic (*d*) valves are normal in caliber. A PDA (*b*) and an interrupted aortic arch distal to the left common carotid (*c*) also are seen in this case. Notice the normal separation between the pulmonary artery and aorta proximally (*f*)