Embryology and Anatomy of the Aorta

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Embryology

Aorta

Development of the aorta occurs during the third week of gestation. At this time, isolated vascular islands coalesce into plexuses to form the (initially) paired aortae. Each primitive aorta consists of a ventral and dorsal segment. The ventral and dorsal segments of the primitive aorta are continuous through the first aortic arch (Fig. 2.1). The aortic sac is formed from the fusion of the two ventral aortae and the descending aorta by fusion of the dorsal aortae. A six-paired system of aortic arches sequentially develops in cranio-caudal fashion between the ventral and dorsal aortae providing blood flow from the cardiac ventricles to the embryonic circulatory system. In addition, the dorsal aorta gives off several intersegmental arteries (Fig. 2.2).

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Vasculogenesis and Angiogenesis

Vasculogenesis is the de novo formation of endothelial cells from mesodermal precursors in the embryo. The process forms the extraembryonic yolk-sac vasculature, paired aortas, endocardium, and vascular plexus of the embryo, all before the onset of blood circulation. Angiogenesis, the rapid expansion and remodeling of the vasculature, subsequently occurs. This involves endothelial cell sprouting, vessel

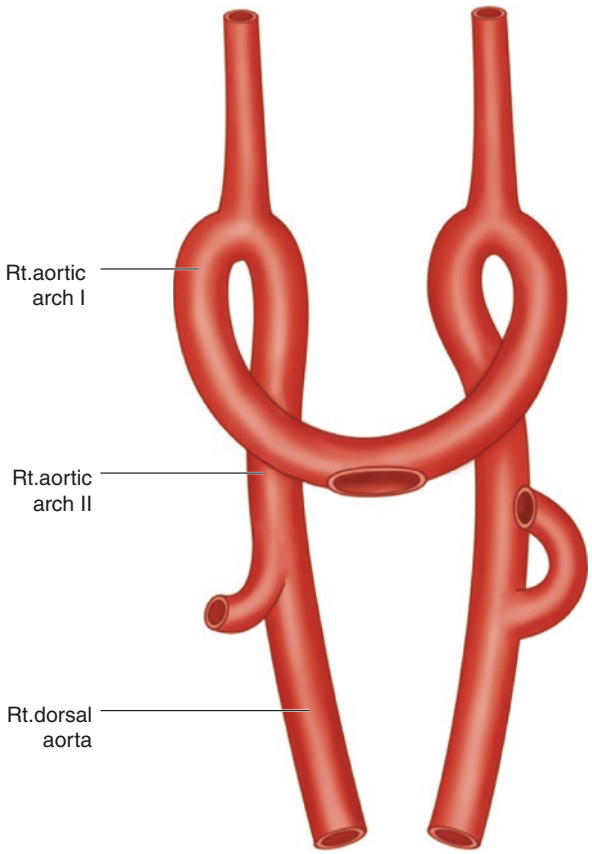

Fig. 2.1 The primitive aorta

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branching, and intussusception from existing blood vessels. This process involves complex regulatory cascades.

Aortic Arch Development

Aortic arch development includes the sequential development and then partial involution of six arch pairs which arise from paired dorsal aortae and fuse distally [1]. The ventral and dorsal segments of the primitive aorta are continuous through the first aortic arch. The second pair of aortic arches makes their appearance in the middle of gestation week 4. They give rise to the stapedial and hyoid arteries (Table 2.1). The first and second pairs of aortic arches regress rapidly and are not seen after day 31. The third pair of vascular arches arise by the end of week 4. Then, the common carotid and

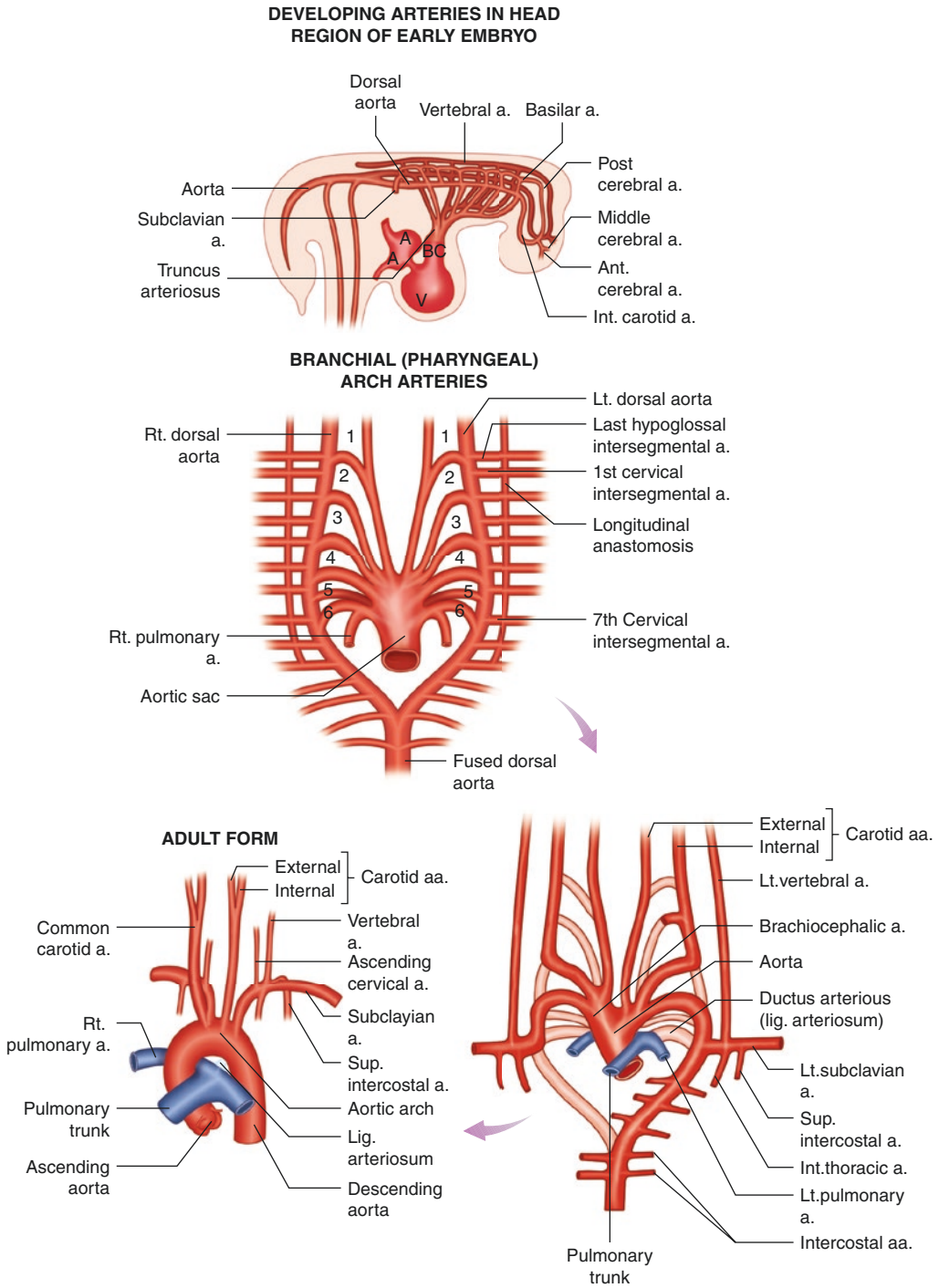

Fig. 2.2 The six-paired system of aortic arches

proximal portions of the internal carotid arteries arise from the third pair of aortic arches. The internal carotid arteries are attached to the cranial portions of the dorsal aortas, which form the remainder of the carotid artery. Next, the fourth pair of arches develops. Interestingly, their development differs depending on the side of the arch discussed. On the right side, the fourth arch forms the proximal portion of the right subclavian artery. The distal portion of the subclavian artery then forms from the right dorsal aorta. The right primitive ventral aorta forms the brachiocephalic arterial trunk and the first portion of the aortic arch. On the left side, the fourth arch becomes the arch of the aorta and is continuous with the primitive left dorsal aorta. The left subclavian artery arises directly from the aorta. Of note, in mammals, the fifth aortic arches are rudimentary and either degenerate or may never even develop. The sixth pair of arches arise by the middle of week 5 and give rise to the left and right pulmonary arteries. Once pulmonary vasculature is established, the communication between the primitive dorsal aorta and the pulmonary arteries regresses. On the right side, the regression is total and complete. On the left side however, the distal portion of the left arch remains in communication with the dorsal aorta until birth, forming the ductus arteriosus. The ductus arteriosus diverts blood from the pulmonary artery to the aorta. In the neonatal period, the functional duct becomes the anatomic ligamentum arteriosum (Table 2.1 and Fig. 2.3).

Aortic Arch Anomalies

Most aortic arch anomalies are secondary to abnormal retention or disappearance of various embryonic vascular segments.

Patent Ductus Arteriosus (Fig. 2.4)

During intrauterine life, the ductus arteriosus allows for blood flow between the pulmonary artery and aorta. In full-term infants, the duct usually closes within the first two days of life. Persistence of the ductus arteriosus postnatally often occurs in premature infants caused by delayed ductal involution [1]. Closure of the ductus involves the prostaglandin cascade as well as mitochondrial oxygen sensing and altered voltage-gated potassium channels. However, the direct pathogenesis of ductal patency has not yet been defined. Ductal patency is two to three times as common in girls as in boys, with most of the cases occurring as isolated defects. However, persistence of a large ductus arteriosus may occur in association with a variety of congenital cardiovascular malformations. Typical concomitant findings are left ventricle hypertrophy and pulmonary artery dilation. A persistent ductus arteriosus may also be associated with coarctation of the aorta, transposition of the great vessels, and ventricular septal defects.

Table 2.1 Correspondence of embryonic aortic arch arteries to their derivative adult counterparts

Embryonic	Adult
Aortic arches	
1	Maxillary artery (portion of)
2	Stapedial artery (portion of) Hyoid artery (portion of)
3	Right and left common carotid arteries (portion of) Right and left internal carotid arteries
4	Right subclavian artery (portion of) Arch of the aorta (portion of)
5	Regresses in humans
6	Right and left pulmonary arteries (portion of) Ductus arteriosus

Coarctation of the Aorta

Coarctation of the aorta is defined as a luminal narrowing of the aortic arch, usually posterior and adjacent to the insertion of the ductus arteriosus. Less frequently, coarctation can occur proximal to the left subclavian artery. The discrete narrowing results in at least a 20 mm Hg gradient across the coarctation and occurs two to five times more frequently in males than females. It is responsible for up to 8% of all cardiovascular congenital defects. Simple coarctation is the most common form. It may be detected de novo in adults and is not associated with other malformations. Complex coarctation is often associated with abnormal aortic valve (AV) morphologies (50–80% of cases of bicuspid AV), abnormal dimensions of the transverse aortic arch (isthmus), and abnormal antegrade left ventricular output in utero. It can also be seen with ventricular septal defects, patent ductus arteriosus, parachute deformity of the mitral valve, and circle of Willis cerebral artery aneurysm (berry aneurysm; 10% of cases).

Aortic pseudocoarctation is a rare congenital anomaly resulting from kinking and buckling of an excessively elongated aorta [2]. Pseudocoarctation is not usually associated with an aortic aneurysm. Aortic atresia, or complete interruption of the aorta, is usually lethal unless it is treated surgically within the first month of life.

The etiology of aortic coarctation is not known. Proposed theories for the development of congenital aortic coarctation include: the flow theory, the reduction of antegrade intrauterine blood flow causing underdevelopment of the aortic arch; the ductal theory, constriction of ductal tissue extending into the thoracic aorta; and simply a primary defect of the aortic wall. Acquired causes include inflammatory processes such as Takayasu arteritis and severe atherosclerosis (Fig. 2.5).

Right Aortic Arch (Fig. 2.6)

In the right aortic arch anomaly, the right rather than the left dorsal aorta is maintained in its entirety. The most common type is the right arch in which there is an aberrant left subcla-

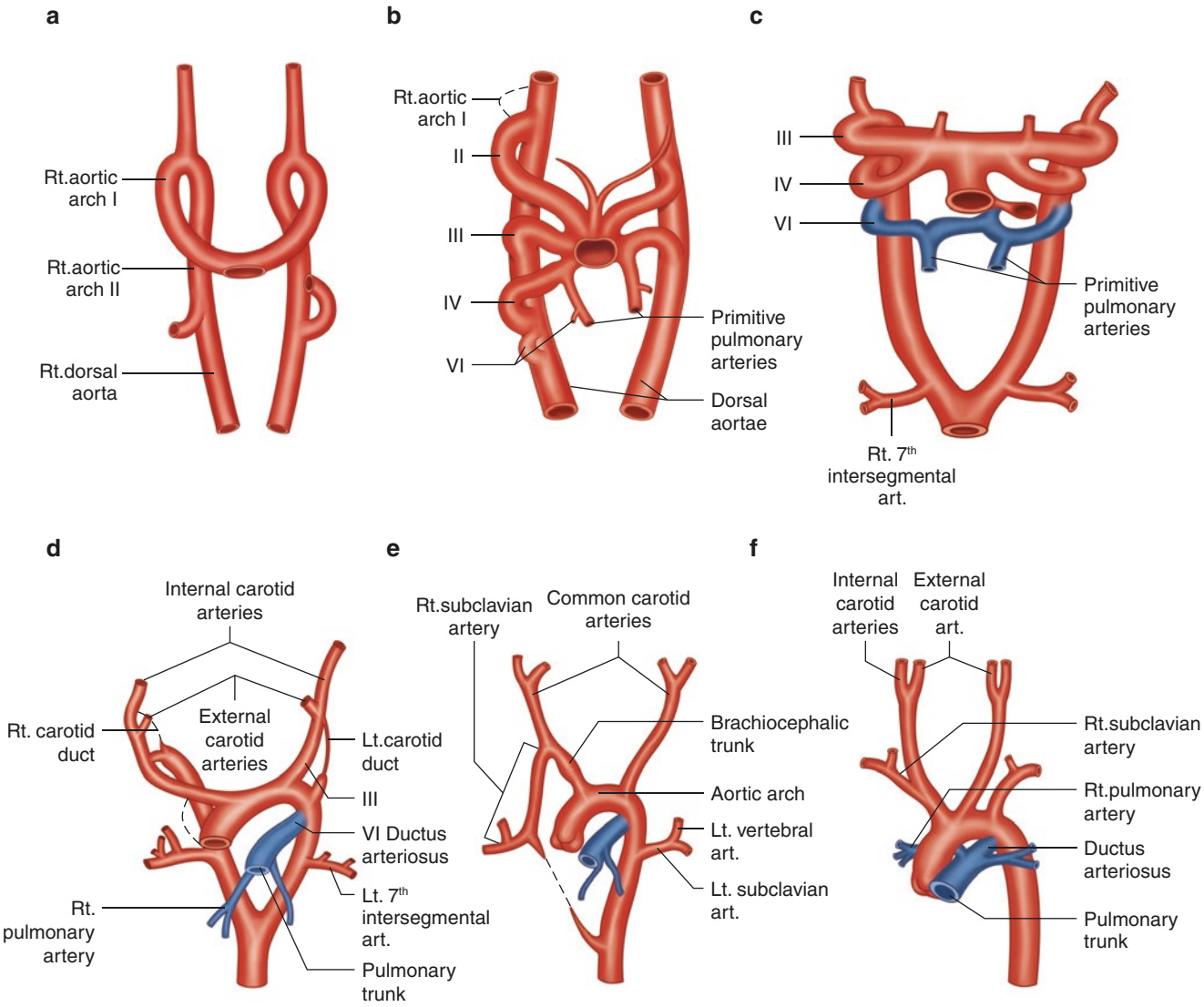

Fig. 2.3 Development of the aortic arch system. Sizes of embryos: (**a**) 3 mm, (**b**). 4 mm, (**c**). 10 mm; the first two aortic arches have regressed; the third, fourth, and sixth are present; and the truncoaortic sac has been divided by the formation of the aortopulmonary septum, so that the sixth arches are now continuous with the PT. (**d**). 14 mm; the dorsal aortas, between the third and fourth arches, have disappeared, and the third arch begins to elongate; the right sixth arch has disappeared, but the left sixth arch persists as the ductus arteriosus. (**e**). 17 mm; the right dorsal aorta has become atrophic between its junction with the left dorsal aorta; the origin of the right seventh intersegmental artery has now become attenuated and later disappears; the remaining components of the right dorsal aorta and right fourth aortic arch form the proximal subclavian artery. (**f**). neonate; the distal part of the left sixth aortic arch, the ductus arteriosus, normally involutes to form the ligamentum arteriosum. Art. artery, Lt. left, Rt. right

vian artery [3]. The vessels originate in the following order: left common carotid, right common carotid, right subclavian, and left subclavian artery. This type is rarely associated with congenital heart disease. Symptoms can result from vascular ring formation. The mirror-image type (branching pattern of the aortic arch is the mirror image of normal – left brachiocephalic trunk, right common carotid, and subclavian arteries) is almost always associated with congenital heart disease, especially cyanotic heart disease. In this case, the arch lies anterior and to the right of the trachea and esophagus.

Double Aortic Arch (Fig. 2.7)

Double aortic arch is the result of persistence and continued patency of the segment of the right dorsal aorta between the origin of the right seventh intersegmental artery and its junction with the left dorsal aorta, allowing for an ascending aorta that divides anterior to the trachea and esophagus, with one arch coursing to the left and one to the right. The arches completely encase the trachea and esophagus and rejoin posteriorly to form the descending thoracic aorta. This encasement can exert a compressive effect and lead to symptoms.

The double aortic arch patient often becomes symptomatic in the first few weeks of life secondary to this constriction, causing airway compression. The most classic sign is nonpositional stridor that is not relieved by bronchodilators. Double aortic arch can also lead to feeding problems, due to compression of the esophagus. Rarely, this can present with other congenital malformations, including ventricular septal defect, atrial septal defect, patent ductus arteriosus, and tetralogy of Fallot. Double aortic arch can be categorized as dominant right, dominant left, or balanced arches.

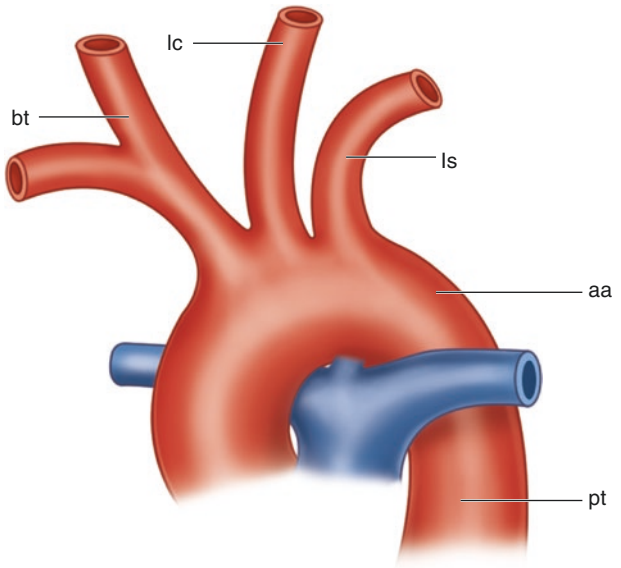

Fig. 2.4 Schematic drawing of a patent ductus arteriosus. Bt brachiocephalic trunk, lc left carotid artery, ls left subclavian artery, aa aortic arch, pt pulmonary trunk, * ductus arteriosus

Interrupted Aortic Arch

Interrupted aortic arch results from a complete interruption or atresia of a segment of the aortic arch [4]. It is classified into three subtypes based on the anatomic location of the atretic segment, known as the Celoria and Patton classification (Fig. 2.8). Type A, the arch interruption occurs distal to the left subclavian artery (1/3 of cases). Type B, the interruption occurs between the left subclavian artery and the left common carotid artery (most common; nearly 2/3 of cases). Type C, interruption occurs proximal to the origin of the left common carotid artery (least common; around 1% of cases). Clinically, the presentation is similar to that of coarctation. Patients remain stable as long as the ductus arteriosus remains patent. This anomaly has been associated with single ventricle, ventricular septal defect, left ventricular outflow tract obstruction, anomalous right subclavian artery, aortopulmonary window, truncus arteriosus, and transposition of the great vessels.

Anomalous Right Subclavian Artery (Fig. 2.9)

The origin of the right subclavian artery is from the right fourth aortic arch, the dorsal portion of the aorta, and the sev-

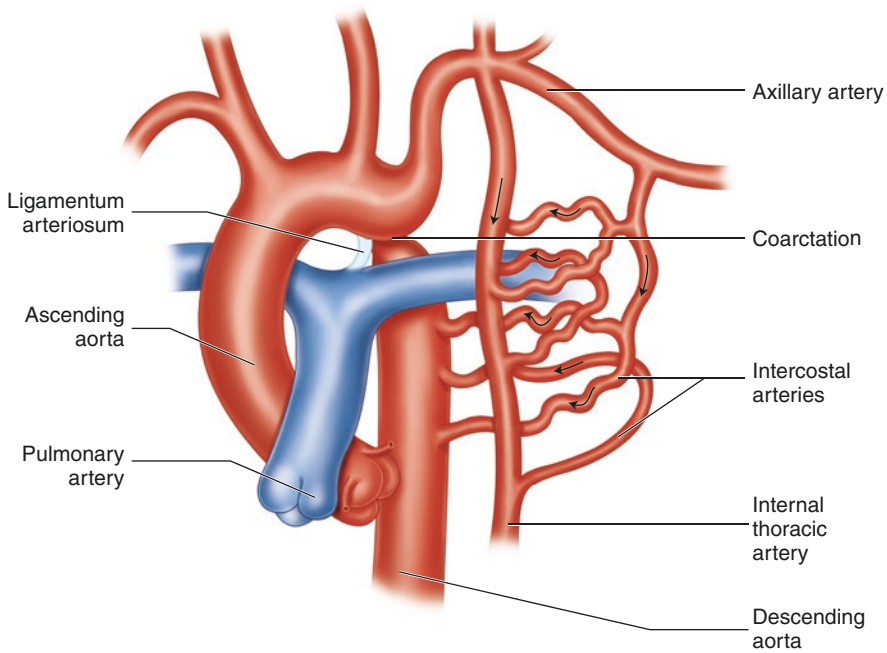

Fig. 2.5 Coarctation causes severe obstruction of blood flow in the descending thoracic aorta. The descending aorta and its branches are perfused by collateral channels from the axillary and internal thoracic arteries through the intercostal arteries (arrows)

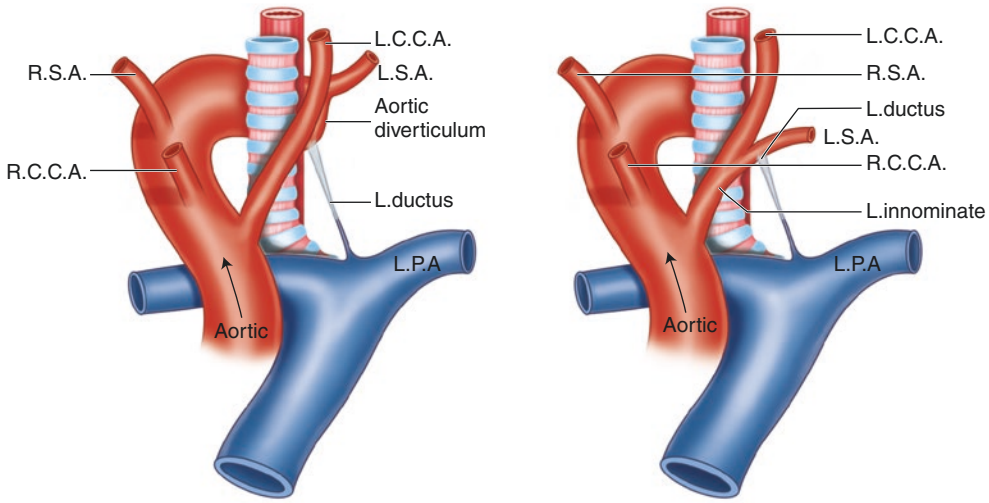

Fig. 2.6 (**a**) Right aortic arch with aberrant left subclavian artery. (**b**) Right aortic arch with mirror-image branching pattern

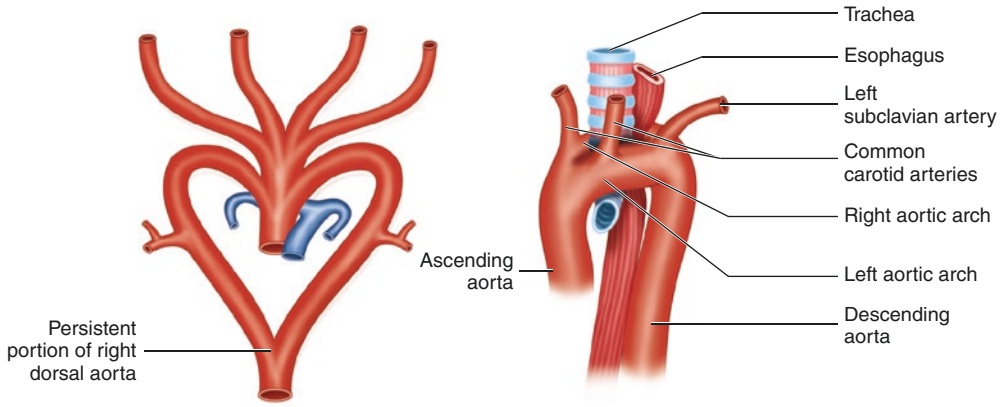

Fig. 2.7 Double aortic arch

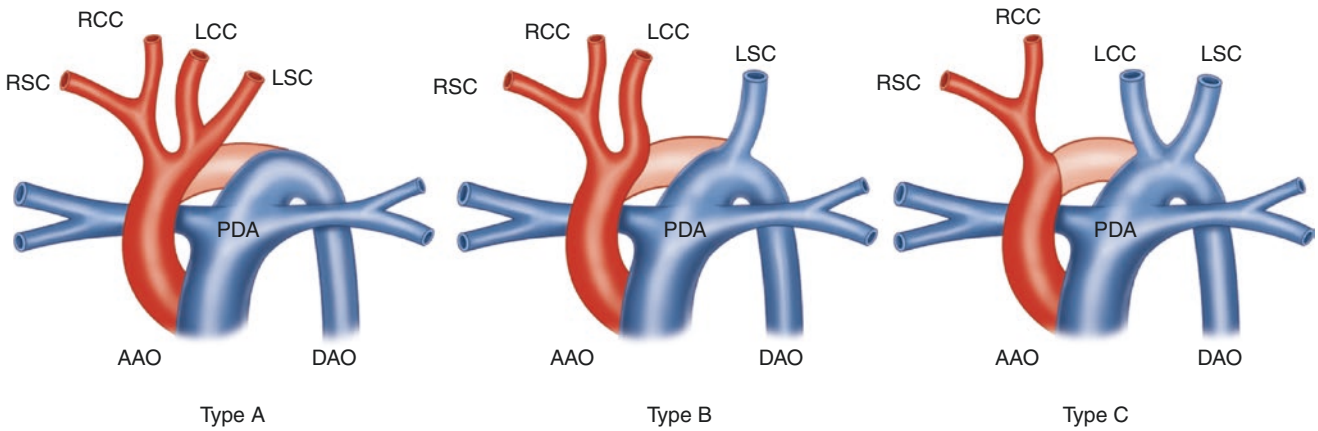

Fig. 2.8 Celoria and Patton classification of interrupted aortic arches

enth intersegmental artery. When this patterned segment is absent, the right subclavian artery can arise from the aortic arch distal to the left subclavian artery [5]. This can only occur if the right dorsal aorta, between the origin of the right seventh intersegmental artery and the junction with the left dorsal aorta, is maintained, so as to form the proximal portion of the right subclavian artery. An anomalous right subclavian artery arising from the proximal portion of the descending thoracic aorta is the most common aortic arch anomaly [6]. However, few patients have clinical symptoms directly attributable to this anomaly. Symptoms that do occur, like airway obstruction and dysphagia, are rare without aneurysmal degeneration of the vessel. Most patients with dysphagia, but without aneurysmal changes, are infants [7].

Absent Left Pulmonary Artery

The left pulmonary artery can be absent when it arises from a left-sided ductus arteriosus (or ligamentum arteriosum), as a result of abnormal disappearance of the proximal left sixth arch.

Absence of one of the pulmonary arteries in itself produces few or no symptoms. If there is no associated cardiac defect, patients may present with slight dyspnea, exercise intolerance, cough, recurrent respiratory infections, pulmonary hypertension in the contralateral lung arteries, or occasional hemoptysis due to the bronchial arterial supply to the affected lung. However, most patients are asymptomatic, and the diagnosis is first suggested by the appearance of the involved lung on a routine chest radiograph. The ipsilateral lung will be smaller than normal, and the contralateral lung will be overinflated and may herniate across the midline [8–10]. When associated with congenital heart disease, it can be seen with right-sided aortic arch, septal defects, truncus arteriosus, and tetralogy of Fallot. Patients who have congenital heart disease and a unilateral absent pulmonary artery usually present with symptoms that are due to their congenital heart lesions.

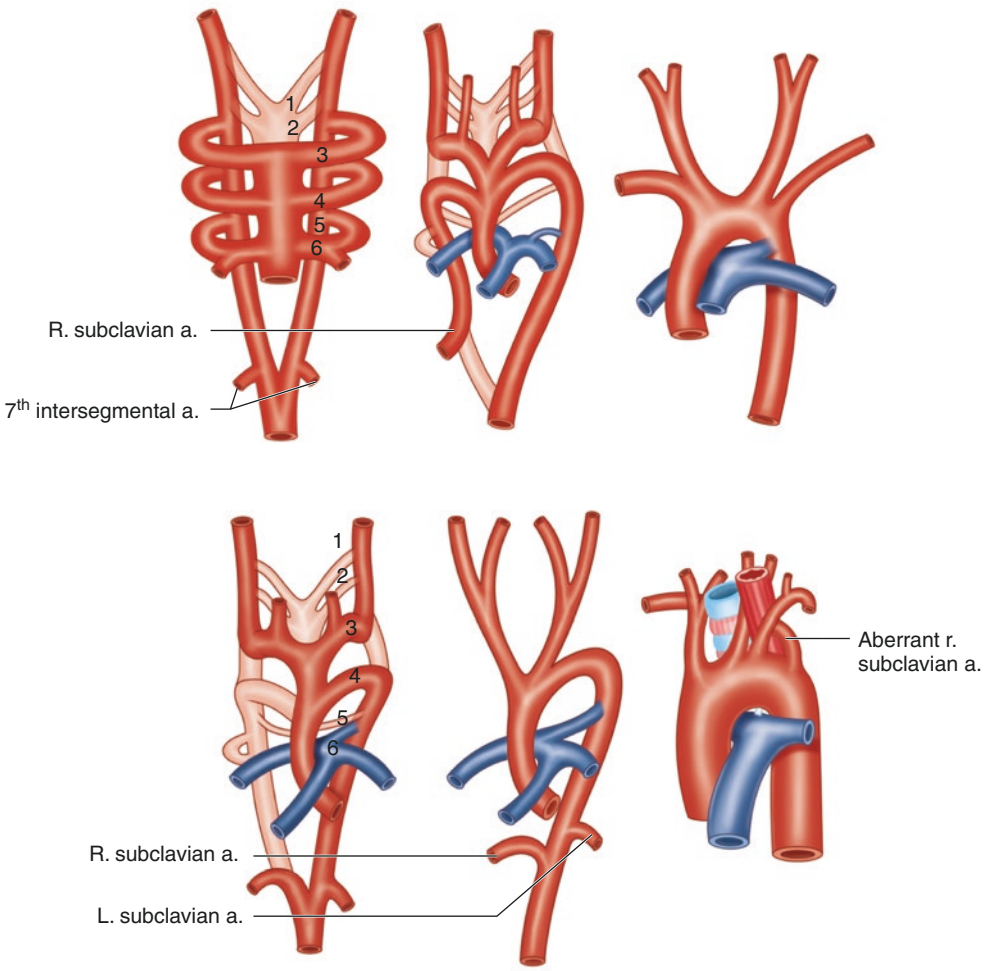

Fig. 2.9 Schematic of aberrant (anomalous) subclavian artery. The shaded arterial segment represents normal right subclavian artery formation in top row and anomalous formation in the bottom row

Normal Aortic Anatomy

The aorta is the largest artery of the human body. Though a continuous structure, for the sake of clinical reference, the aorta is divided logically into the ascending, arch, descending, suprarenal, and infrarenal divisions, and terminates at the iliac bifurcation.

Less than two millimeters (mm) thick in young adults, the aortic wall is, nevertheless, highly resilient [11]. Various modalities (direct observation, ultrasound, CT scan, and MRI) have been used to determine norms for external and internal diameters [12]. In one CT study, mean internal and external aortic diameter for the ascending aorta at end systole in "normal" individuals of both sexes were assessed with findings as follows: external diameter of 35.6, 38.3, and 40 mm for females and 37.8, 40.5, and 42.6 mm for males in age groups 20–40, 41 to 60, and above 60 years, respectively [13], and internal diameter of 38.0, 40.7, and 42.4 mm for females and 40.2, 42.9, and 45.0 mm for males in the same age brackets.

Of course, as volume is carried away by branching muscular arteries arising in its distal course, the volume in the aorta, and hence its diameter, decreases. Age, sex, and size are important covariants as demonstrated in Table 2.2 [14].

A Danish study to establish ultrasonic norms for the abdominal aorta demonstrated a mean suprarenal aortic diameter of 18.4 mm for male vs.16.6 mm for females and for the distal aorta, 16.0 mm vs. 13.7 mm, respectively [15].

The aorta demonstrates the tri-layer pattern seen in all vessels apart from capillaries – tunica intima, tunica media, and tunica externa. The aorta is the prototype "elastic artery" featuring an incredibly thick tunica media rich in smooth muscle and elastic fibers. In fact, the aorta is so thick that it requires its own nutritive capillary network, the vasa vasorum. In dogs, it has been shown that the percent smooth muscle volume varies little across the arterial tree (45–55%), but the aorta has a dramatically greater elastic tissue volume (22.6%) [16].

Ventricular ejection is a very energetic event, and a small but significant portion of the energy is briefly sequestered in the stretching of aortic elastic fibers. This energy is returned an instant later as elastic recoil, sustaining flow to coronary arteries and peripheral vessels beyond systole into diastole (Windkessel effect). This also means that the same flow is delivered at a lower systolic pressure than would be the case for an inelastic tube, so the aorta is not simply a pipe. With aging, the aorta does in fact become more "pipelike," accounting, at least in part, for the higher peak systolic pressures and widening of pulse pressure as years advance [17]. Ultrasound observations of the abdominal aorta confirm a loss of pulsatile expansion with age [15].

Histologically, the aging aorta is characterized by thickening and atherosclerosis of the intima, along with cystic necrosis, elastin fragmentation, fibrosis, and medial necrosis of the media as well as fibrosis in adventitia. These changes of aortic aging decrease aortic elasticity (distensibility) [14].

Thoracic Aorta

The thoracic aorta's greatest relational complexity occurs within the mediastinum. It anchors in the transverse fibrous skeleton of the heart and its ascending segment lies within the pericardium. It ascends from the annulus in intimate rela-

Table 2.2 Gender-specific effects of obesity on regional aortic diameter – data presented as mean with normal range (+/− 2SD)

Aortic diameter (mm)	Normal weight	Overweight	Obese	ANOVA p
		Male		
Aortic valve annulus	24.0 (18.8–29.2)	24.7 (19.5–29.9)	25.7 (20.7–30.7)	<0.05
Sinus of Valsalva	32.2 (24.6–39.8)	32.9 (25.3–40.5)	33.3 (25.3–31.3)	<0.05
Sino-tubular junction	24.9 (18.1–31.7)	25.8 (17.0–34.6)	25.9 (19.1–32.7)	<0.05
Ascending aorta	26.6 (18.2–35.0)	27.8 (18.8–36.8	28.6 (23.2–34.0)	<0.01
Proximal descending aorta	20.4 (14.6–26.2)	21.2 (15.6–26.8)	22.1 (16.5–27.7)	<0.01
Distal descending aorta	17.4 (12.0–22.8)	18.3 (12.7–23.9)	19.0 (14.8–23.2)	<0.01
BMI (kg/m2)	22 +/−1.7	27 +/−1.6	34 +/−4.8	<0.01
BSA (m2)	1.9 (+/−0.1)	2.0 (+/−0.1)	2.3 (+/−0.2)	<0.01
		Female		
Aortic valve annulus	20.2 (17.0–23.4)	21.7 (18.5–23.9)	21.6 (17.6–25.6)	<0.01
Sinus of Valsalva	27.6 (22.0–33.2)	28.6 (21.6–35.6)	27.8 (22.2–33.4)	<0.05
Sino-tubular junction	21.7 (16.7–26.7)	22.5 (16.5–28.5)	22.3 (16.5–28.1)	<0.05
Ascending aorta	24.8 (17.6–32.0)	26.7 (19.3–34.1)	26.9 (19.3–34.5)	<0.01
Proximal descending aorta	18.6 (14.6–22.6)	19.5 (14.9–24.1)	20.1 (15.5–24.7)	<0.01
Distal descending aorta	16.1 (14.1–18.1)	16.9 (14.7–19.1)	17.6 (15.7–19.5)	<0.01
BMI (kg/m2)	22.0 (+/−1.6)	27.0 (+/−1.5)	37.0 (+/−4.8)	<0.01
BSA (m2)	1.7 (+/−0.1)	1.8 (+/−0.1)	2.0 (+/−0.2)	<0.01

tion to the pulmonary artery to the left with the remaining circumference hugged by the right atrium. Its first branches, the coronary arteries, run to the right and left within the atrioventricular sulcus.

As it ascends from the annulus it points just a bit rightward and anterior, in the axis of the heart, and so runs anterior to the posteriorly arching pulmonary trunk to the left and vena cava on the right.

Crossing the left right pulmonary artery, it ascends for approximately five centimeters (cm) where the pericardium fuses with the adventitia as it transitions to the arch. Overlying this pericardial sheet anteriorly is the thymic remnant.

The general orientation of the arch is from right to left and from anterior to posterior. It is a place of significant turbulence as red cells are shouldering one another around the geometric challenge of the arch and the orifices of the brachiocephalic, left carotid, and left subclavian arteries. The resultant microtrauma to the endothelium (tunica intima) contributes to endothelial dysfunction and makes this a site for early (and, often, eventually severe) atherosclerotic transformation [18].

On the lesser curve of the arch, typically just beyond the origin of the left carotid, is a thick, fibrotic tether to the pulmonary trunk bifurcation, the ligamentum arteriosum (LA). Just distal to the LA, the left vagus nerve sends the recurrent laryngeal nerve looping from posterior to anterior around the aorta.

Beyond the left subclavian artery, the arch moves posterior, transitioning to the descending aorta (T4–T5 intervertebral level) which parallels the spine on its downward journey through the diaphragmatic aortic hiatus where it emerges as the abdominal aorta. Throughout the length of the descending thoracic and abdominal aorta, small posterolateral branches emerge to supply various thoracic and abdominal structures (intercostal, subcostal, mediastinal, bronchial, diaphragmatic, esophageal, pericardial, and lumber) (Figs. 2.10 and 2.11).

Before moving on to the abdominal aorta, it is worth noting that congenital variations in the thoracic aorta are not rare. In fact, the most common congenital arterial anomaly is a bicuspid aortic valve, existing in roughly 1% of the population and leading to premature valvular dysfunction. Deviations from the typical brachiocephalic-left common carotid-left subclavian orientation of the great vessels are not rare and, of course, patent ductus arteriosus occurs in 2/1000 term births and 8/1000 premature births [19].

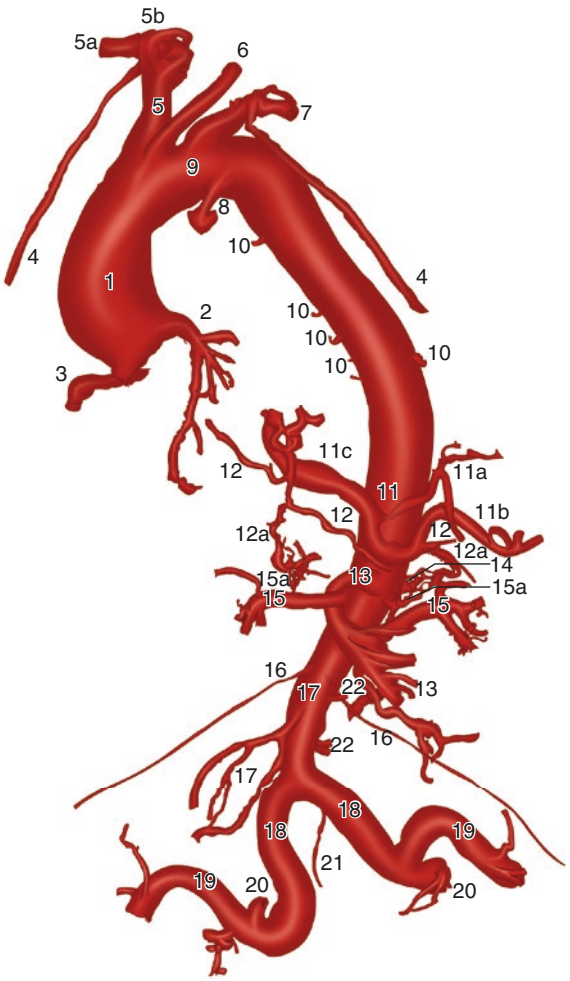

Fig. 2.10 Anterior anatomic dissection of the aorta from aortic valve to aortic bifurcation with labeling of all branches (numbering). 1. Aorta; 2. Left coronary artery; 3. Right coronary artery; 4. Internal thoracic arteries; 5. Brachiocephalic trunk (innominate), (a) Right subclavian artery and (b) Right common carotid artery; 6. Left common carotid artery; 7. Left subclavian artery; 8. Ligamentum arteriosum; 9. Aortic arch; 10. Thoracic intercostal arteries; 11. Celiac trunk, (a) Left gastric artery, (b) Splenic artery, and (c) Hepatic artery; 12. Inferior phrenic arteries, (a) Superior suprarenal arteries; 13. Superior mesenteric artery; 14. Middle suprarenal artery (left only); 15. Renal artery, (a) Inferior suprarenal arteries; 16. Gonadal (testicular or ovarian) arteries; 17. Inferior mesenteric artery; 18. Common iliac arteries; 19. External iliac arteries; 20. Internal iliac arteries; 21. Median sacral artery; and 22. Third and fourth lumbar arteries

Abdominal Aorta

Just beyond the diaphragmatic aortic hiatus, from the anterior aortic wall, the celiac trunk and superior mesenteric artery (SMA) arise in quick succession. Typically, just below the level of the SMA, the left and right renal arteries come off the aorta laterally, though it must be noted that renal arteries not infrequently show significant variation in location and number. Multiple (typically two) renal arteries on one or both sides may exist. Gonadal arteries arise anterolaterally and typically a few centimeters inferior to the renal arteries.

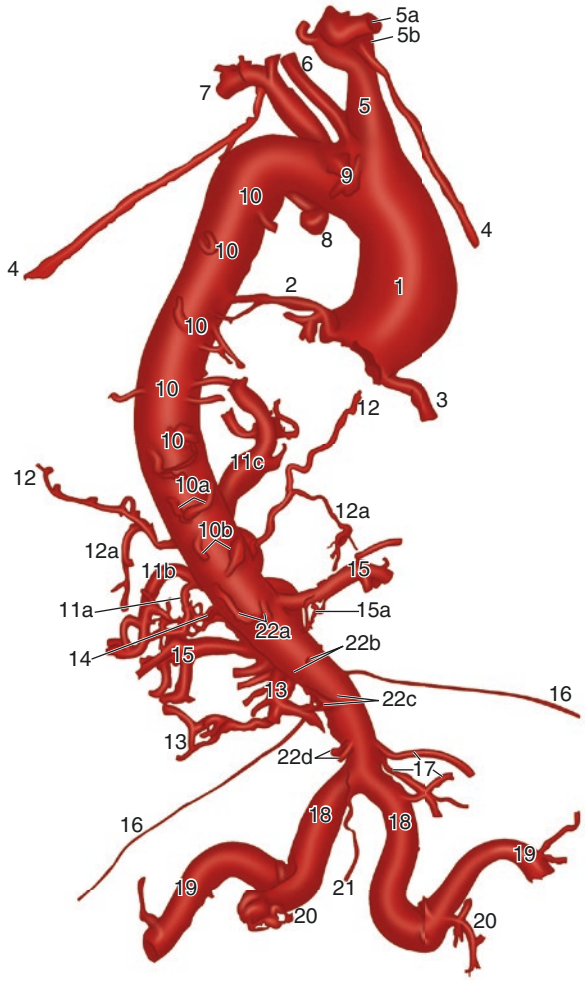

Fig. 2.11 Posterior anatomic dissection of the entire adult aorta from the aortic valve to aortic bifurcation with labeling of all branches. 1. Aorta; 2. Left coronary artery; 3. Right coronary artery; 4. Internal thoracic arteries; 5. Brachiocephalic trunk (innominate), (a) Right subclavian artery and (b) Right common carotid artery; 6. Left common carotid artery; 7. Left subclavian artery; 8. Ligamentum arteriosum; 9. Aortic arch; 10. Thoracic intercostal arteries, (a) T11 and (b) T12. 11; Celiac trunk, (a) Left gastric artery, (b) Splenic artery, and (c) Hepatic artery; 12. Inferior phrenic arteries, (a) Superior suprarenal arteries; 13. Superior mesenteric artery; 14. Middle suprarenal artery (left only); 15. Renal artery, (a) Inferior suprarenal arteries; 16. Gonadal (testicular or ovarian) arteries; 17. Inferior mesenteric artery; 18. Common iliac arteries; 19. External iliac arteries; 20. Internal iliac arteries; 21. Median sacral artery; and 22. Lumbar arteries, (a) L1, (b) L2, (c) L3, and (d) L4

The inferior mesenteric artery (IMA) is the last major branch to arise from the anterior aortic wall and supplies the hind gut just as the celiac trunk supplied the foregut and the SMA the midgut. It arises substantially distal to the renal and gonadal vessels at the level of L3, approximately 5 cm above the iliac bifurcation. It is also very susceptible to atherosclerotic occlusion in the elderly, and in such cases, may be an asymptomatic finding or may be a source for the development of ischemic colitis. Numerous lumbar arteries arise from the aorta. The middle (median) sacral artery is typically the last branch at the level of the aorto-iliac bifurcation.

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