Vascular Surgery
Dedication

To our families
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Preface

This revised edition of Vascular Surgery: Basic Science and Clinical Correlations was developed in order to address significant changes that have occurred in contemporary vascular surgery and to highlight new information that has developed regarding vascular imaging and interventional and endovascular procedures. The overall length of the text is slightly shorter than the first edition with relevant core chapters being retained to emphasize the basic science nature of the text, with approximately 60 percent of the material undergoing major revisions or being new chapters.

The significant change from the first text is an emphasis on vascular pathology and physiology that is relevant to current practice, including information that is currently included on the vascular board examinations. A new emphasis on endovascular therapies has been added by including five chapters on endovascular techniques and an additional section with six chapters comparing conventional vascular reconstruction with endovascular methods. These new chapters address the most important issue in contemporary vascular surgery, i.e. the role of endovascular methods in treating vascular lesions and the impact that this has on training and credentialing. A unique aspect of this book differentiating it from other texts is a comparison of conventional methods with the endovascular techniques.

Overall, the text provides a comprehensive approach to contemporary vascular surgery and future perspectives. The authors are preeminent in the field and are most capable for addressing the assigned topics, with the goals being to provide an updated and forward-looking text that accommodates the needs of practicing and training vascular surgeons.

Rodney A. White
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We would like to acknowledge the efforts of Blackwell Publishing, Futura Division, for the timely preparation of this text. In particular, we appreciate the efforts of Steve Korn, Jacques Strauss, and the invaluable expertise of Joanna Bellhouse, Development Editor, who has meticulously and efficiently organized materials and prepared the text for publication.
Vascular pathology and physiology
The vascular system develops between the third and eighth weeks of gestation. In the middle of the third week, the embryo is no longer able to meet its nutritional requirements by diffusion alone, thus prompting differentiation of extraembryonic mesodermal cells (angioblasts) located in the wall of the yolk sac. These angioblasts form angiogenic cell clusters, which canalize to form early blood vessels. Cells that are centrally located in these clusters differentiate into blood cells, while those at the periphery flatten and form endothelial cells.

Similarly, during this same period, intraembryonic mesodermal cells differentiate to form the heart tube, paired dorsal aortae, visceral arteries, and axial arteries of the developing limb buds. Woollard described the above events in the development of the vascular system in three stages: (1) the capillary network stage, an undifferentiated network of primitive blood lakes; (2) the retiform stage, when separation of the primitive arterial and venous channels occurs; and (3) the gross differentiation phase with the appearance of mature vascular channels. By the end of the eighth week of gestation, development of the vascular system is virtually complete with only minor changes occurring after this time.

**Arterial system**

**Aortic arch and great vessels**

The aortic arch and its major branches develop from the six embryologic aortic arches, which, in turn, originate from the aortic sac. Each branchial arch is supplied by one of the aortic arches. The fifth aortic arch is often not formed at all (Fig. 1.1). In the 4-mm embryo (end of fourth week), the first aortic arch has nearly disappeared with only a small portion persisting on the maxillary artery (Fig. 1.2). The second aortic arch also regresses with portions persisting as the hyoid and stapedial arteries.

In the 10-mm embryo (beginning of sixth week), the first and second aortic arches have disappeared and the third, fourth, and sixth aortic arches enlarge (Fig. 1.3). The third aortic arch is the anlage of the common carotid artery and the first portion of the internal carotid artery with the remainder of the internal carotid artery formed by the dorsal aorta (Fig. 1.4). The proximal right subclavian artery develops from the right fourth aortic arch. Its distal portion is formed by a portion of the right dorsal aorta and the seventh intersegmental artery (see Fig. 1.4). The embryologic left fourth aortic arch forms the arch of the aorta between the left common carotid and left subclavian arteries.

The fifth aortic arch is transient and never well developed. No portion persists in the extraterine life.

The sixth aortic arch (pulmonary arch) gives off branches that grow toward the developing lung bud. The right sixth aortic arch forms the proximal segment of the right pulmonary artery, while the distal left sixth aortic arch persists as the ductus arteriosus; it later becomes the ligamentum arteriosum (see Fig. 1.4).

Formation of the neck causes the heart to descend from its initial cervical position into the thoracic cavity. This results in elongation of the innominate and carotid arteries and a shift of the origin of the left subclavian artery from the level of the seventh intersegmental artery to a point closer to the origin of the left common carotid artery (Fig. 1.5). In embryologic development, the recurrent laryngeal nerves supply the sixth branchial arches. With the caudal shift of the heart and disappearance of portions of the right fifth and sixth aortic arches, the right recurrent laryngeal nerve moves up to hook around the fourth aortic arch while the left recurrent laryngeal nerve hooks around the ligamentum arteriosum (see Figs. 1.4 and 1.5).

**Visceral arteries**

Most of the differentiation of the arterial supply to the abdominal viscera has occurred by the end of the eighth week. The primordium of the celiac artery is represented by the paired cephalic roots of the vitelline arteries at the level of the 10th ventral segmental artery. The superior mesenteric artery originates by fusion of the paired vitelline arteries at the level of the 13th...
ventral segmental artery. Fusion of the vitelline arteries in a more caudal location forms the inferior mesenteric artery.

Renal arteries

The adult kidney (metanephros) begins to develop in the fifth week of gestation and is initially located in the pelvis. With diminution of the body curvature and growth of the body in the lumbar and sacral regions, the kidney ascends into the abdomen. The metanephros receives its original blood supply from a pelvic branch of the aorta but as it ascends, arteries originating from successively higher levels of the abdominal aorta supply the kidney while the lower vessels degenerate.\(^1\)
Arteries to the lower extremity

During the fifth week of development (6-mm embryo), the umbilical artery gives rise to the sciatic artery. The sciatic artery is a continuation of the internal iliac artery, which develops with the lower limb bud as its axial artery. The femoral artery, an extension of the external iliac artery, replaces the sciatic artery and its branches to the thigh during the eighth week of development.\(^1\) Adult derivatives of the sciatic system include the popliteal, anterior tibial, and peroneal arteries.

Venous system

During the fifth week of gestation, three major pairs of veins are present in the embryo: (1) vitelline or omphalomesenteric veins between the yolk sac and the sinus venosus; (2) umbilical veins, which course between the chorionic villi and the embryo; and (3) cardinal veins, which drain the body of the embryo (Fig. 1.6).

Vitelline vein derivatives

The vitelline veins pass from the yolk sac to the venous plexus surrounding the duodenum prior to passing into the septum transversum (Fig. 1.7). Liver cords budding from the duodenum grow into the septum transversum, interrupting the course of the vitelline veins to form the hepatic sinusoids. The left and right hepatocardiac channels drain the hepatic sinusoids into the sinus venosus (Fig. 1.8). With obliteration of the left hepatocardiac channel, the right hepatocardiac channel becomes the posthepatic (suprahepatic) inferior vena cava. The portal vein forms as the venous plexus surrounding the duodenum coalesces into a single vein. The superior mesenteric vein develops from the distal right vitelline vein.

Umbilical vein derivatives

The entire right umbilical vein and the proximal portion of the left umbilical vein disappear, while the distal left umbilical vein persists to carry blood to the liver from the placenta. A communication, the ductus venosus, later forms between the proximal portions of the umbilical arteries persist to form the internal iliac and superior vesical arteries.\(^1\)
left umbilical vein and the right hepatocardiac channel, by-passing the sinusoids of the liver (Fig. 1.9). After birth, the left umbilical vein and the ductus venosus are obliterated to form the ligamentum teres hepatis and ligamentum venosum, respectively.

**Cardinal vein derivatives**

In early embryologic development, the cardinal venous system is composed of three pairs of veins: (1) the anterior cardinal veins, which drain the cephalic embryo; (2) the posterior cardinal veins, which drain the remainder of the embryo; and (3) the common cardinal veins, which are formed by the junction of the anterior and posterior cardinal veins (see Fig. 1.6). During the fifth to seventh weeks of gestation, the following veins form: (1) the subcardinal veins, which drain the kidneys; (2) the sacrocardinal veins, which drain the lower extremities; and (3) the supracardinal veins, which drain the body wall via intercostal veins (Fig. 1.10).

In the formation of the vena cava, anastomoses develop between the left and right sides of the cardinal system, channeling blood from left to right. The communication between the anterior cardinal veins develops into the left brachiocephalic vein. The right common cardinal vein and the proximal portion of the right anterior cardinal vein form the superior vena cava.
The communication between the subcardinal veins forms the left renal vein. After development of this communication, the proximal left subcardinal vein disappears with its distal portion persisting as the left gonadal vein. Hence, the right subcardinal vein becomes the renal segment of the inferior vena cava.

As portions of the posterior cardinal veins disappear, the supracardinal veins become more important. The azygos vein, into which the 4th through 11th intercostal veins empty, forms from the right supracardinal vein and a portion of the right
Interrupted aortic arch is also a relatively rare anomaly, resulting from obliteration of the left fourth aortic arch (Fig. 1.13). The ductus arteriosus remains widely patent, supplying blood of low oxygen content to the systemic circulation while the aortic trunk supplies the two common carotid arteries.

Anomalies of the aortic arch branches

Common ostial origin of the innominate and left common carotid arteries, the most common anomaly of the arch branches, occurs in approximately 10% of patients. Origin of the left vertebral artery from the aortic arch proximal to the left subclavian artery occurs in 5% of patients.

Aberrant right subclavian artery (arteria lusoria) occurs in approximately 2% of patients, resulting from obliteration of the right fourth aortic arch and proximal right dorsal aorta (Fig. 1.14). In this anomaly, the right subclavian artery arises from the aortic arch just distal to the left subclavian artery, passing behind the esophagus to the right arm, frequently compressing the esophagus (dysphagia lusoria). Absence of the normal origin of the right subclavian artery results in a nonrecurrent right recurrent laryngeal nerve.

Coarctation of the aorta

Coarctation of the aorta may be congenital or acquired and
may occur in the descending thoracic aorta or the abdominal aorta. Our discussion will focus on congenital coarctation.

Several hypotheses have been proposed as causes of congenital coarctation of the aorta. According to Dean and coworkers, congenital coarctations result from either failure of maturation of the mesenchymal cell component or arrested development of the artery during the period of gross differentiation. If arrest occurs during the mesenchymal cell stage, the artery may appear as a fibrous cord. With developmental arrest during the gross differentiation phase, the aorta may appear normal in early childhood, but later may be recognized as a nonexpanding portion of aorta adjacent to a normally growing segment.

With aortic coarctation from anomalous mesenchymal cell maturation, luminal fibrous clefts and ridges causing partial obstruction may be noted on arteriography. Microscopically, dysplastic mesenchymal cell layers compose a disorganized media.

Coarctation of the thoracic aorta may be preductal or postductal. In preductal aortic coarctation, the ductus arteriosus persists supplying poorly oxygenated blood to the lower body. In the postductal type, this channel is obliterated and numerous collaterals from the subclavian and axillary arteries supply the lower body.

Coarctation of the abdominal aorta is rare, accounting for 0.5% to 2% of clinically recognized coarctations of the thoracic and
abdominal aorta. Reconstruction may be challenging because the stenosis may extend from the celiac axis to the infrarenal abdominal aorta. In about 80% of patients, renal artery stenosis with renovascular hypertension is present. Untreated abdominal coarctation may eventually result in cardiac failure or cerebral hemorrhage, the major causes of death from this anomaly. Repair often requires renal revascularization and bypass or replacement of the narrowed aorta in the second or third decade of life.

Anomalies of the visceral arteries

Congenital anomalies of the visceral arteries are not uncommon; however, visceral arterial anomalies requiring vascular surgical intervention are rare. We define a visceral artery anomaly as a difference in number or origin of the arterial supply to an organ from the accepted normal. The normal arterial supply of an organ is that pattern of arteries to a viscus that occurs most commonly. Celiac, hepatic, and renal arterial anomalies of importance to the vascular surgeon are described.

Celiac artery anomalies are found in 11% to 40% of patients. The typical celiac axis, which branches into left gastric, splenic, and common hepatic arteries, is found in 60% to 89% of patients. The most common variation is a gastroplenic trunk with the common hepatic artery arising from the aorta or the superior mesenteric artery occurring in 5% to 8% of patients. Hepatosplenic and hepatogastric trunks occur less frequently and, rarely, the celiac axis may be combined with the superior mesenteric artery (Fig. 1.15).

Hepatic artery anomalies may be of two types: replaced or accessory. A replaced hepatic substitutes for a normal hepatic artery that is absent, while an accessory hepatic is an addition to the normal one that is present. Michels, from 200 anatomic dissections, found one or more hepatic artery anomalies in 83 cases (41%). The four most common variations in the arterial supply to the liver were (1) replaced right hepatic artery, 17%; (2) replaced left hepatic artery, 16%; (3) accessory left hepatic artery, 12%; and (4) accessory right hepatic artery, 8% (Fig. 1.16). In 2.5% of his dissections, Michels noted the common hepatic artery originated from the superior mesenteric artery.

As previously described, during embryologic development, the kidney arterial supply originates from the aorta at
successively higher levels as the kidney ascends from the pelvis. Failure of lower vessels to degenerate results in multiple renal arteries, present in 25% to 33% of adults. Multiple renal arteries are slightly more common on the left than the right and may enter the renal hilum or directly into the parenchyma of one of the poles of the kidney. Supernumerary arteries most commonly enter the upper pole of the kidney and are more common in ectopic kidneys. Lower pole supernumerary arteries to the right kidney typically cross anterior to the inferior vena cava.12

As the kidneys ascend from the pelvis, they must pass between the umbilical arteries. The kidneys are closely opposed and may come into contact with each other as they ascend between the umbilical arteries. If they come into contact, their lower poles may fuse, resulting in a horseshoe kidney, which is found in 1 in 600 persons. Similarly, one or the other kidney may fail to ascend, resulting in a pelvic kidney. Usually, these ectopic kidneys are located in the pelvis close to the common iliac artery.1 Multiple renal arteries often supply horseshoe and pelvic kidneys, commonly arising from the aorta near the aortic bifurcation or from the common iliac arteries.

The Arc of Buhler is represented in intrauterine life as a longitudinal anastomosis that connects the 10th through 13th ventral segmental arteries. The 10th ventral segmental artery contributes to the formation of the celiac artery; the 11th and the 12th segmental arteries regress; and the 13th ventral segmental artery contributes to the development of the superior mesenteric artery. Normally, this longitudinal communication regresses by the eighth week of embryonic life; however, if it persists, the Arc of Buhler forms a communication between the celiac and superior mesenteric arteries. Discovered in 2% of autopsy cases and usually found in the location of the pancreaticoduodenal arteries, the Arc of Buhler may undergo aneurysmal degeneration and rupture, probably related to inherent weakness in the persistent embryonic artery13 (Fig. 1.17). If an aneurysm of this artery is identified, recommendations pertinent to other visceral artery aneurysms should be followed.

Persistent sciatic artery

Persistent sciatic artery is a congenital anomalous continuation of the internal iliac artery, which in 63% of these cases serves as the major blood supply to the lower extremity.3 If the sciatic artery is the major artery of the lower extremity, the superficial femoral artery is hypoplastic or absent. Following the course of the inferior gluteal artery, the sciatic artery passes with the sciatic nerve through the greater sciatic foramen below the piriformis muscle and enters the thigh (Fig. 1.18).14 The artery then courses along the posterior aspect of the adductor

Figure 1.16 Hepatic artery anomalies (C.A., celiac axis; L.G., left gastric; H, hepatic; M.H., middle hepatic; R.H., right hepatic; L.H., left hepatic).

Figure 1.17 Persistent arc of Buhler with associated aneurysm.
magnus muscle to the popliteal fossa, where it continues as the popliteal artery. Early atheromatous degeneration and aneurysm formation are common. Due to its proximity to the sciatic nerve, a sciatic artery aneurysm may present as a painful buttock mass or with sciatic pain. Sciatic artery aneurysms are bilateral in 12% of the cases. Palpable popliteal and pedal pulses without palpable femoral pulses are clinical findings highly suggestive of persistent sciatic artery. Magnetic resonance imaging (MRI) and arteriography provide a definitive diagnosis. Proximal and distal ligation of the aneurysm and femoropopliteal bypass graft is the preferred treatment.

Venous anomalies

Anomalies of the superior vena cava

Anomalies of the superior vena cava of importance to the vascular surgeon include left superior vena cava and double superior vena cava.

Persistence of the left anterior cardinal vein and obliteration of the right common cardinal and proximal right anterior cardinal veins after the eighth week of gestation results in a left-sided superior vena cava (Fig. 1.19). Blood from the right upper extremity and right side of the head drains into the brachiocephalic vein and then into the left superior vena cava, which courses anterolateral to the aortic arch and anterior to the hilum of the left lung. The left-sided superior vena cava then drains into the coronary sinus.

Persistence of the left anterior cardinal vein and failure of the left brachiocephalic vein to form results in double superior vena cava (Fig. 1.20). The left superior vena cava drains into the coronary sinus as previously described.
Anomalies of the inferior vena cava

Embryologic abnormalities of the inferior vena cava and renal veins pose potentially difficult problems for the vascular surgeon during abdominal aortic surgery. Important anomalies of the inferior vena cava include double inferior vena cava and left inferior vena cava.

Double inferior vena cava results when the left sacrocardinal vein fails to lose its communication with the left subcardinal vein. With this anomaly, the left iliac vein may or may not be present but the left gonadal vein is found in its normal location1 (Fig. 1.21).

Left inferior vena cava results from regression of the right sacrocardinal vein, the normal precursor of the lower infrarenal inferior vena cava, and persistence of the left sacrocardinal vein, which maintains its communication with the left subcardinal vein1 (Fig. 1.22).

If the right subcardinal vein fails to make communication with the liver, absence of the suprarenal inferior vena cava results. Blood from the caudal part of the body is shunted directly into the right supracardinal (azygous) vein (Fig. 1.23). The hepatic veins enter the right atrium at the site normally occupied by the inferior vena cava.15

Renal vein anomalies

Important renal vein anomalies include a circumaortic renal collar and a posterior (retroaortic) left renal vein. In utero, communications between the subcardinal and supracardinal veins form a venous ring around the aorta at the level of the renal veins. Failure of the dorsal portion of the ring to regress results in either a posterior renal vein if the ventral portion of

Figure 1.21 Double inferior vena cava.

Figure 1.22 Left inferior vena cava.

Figure 1.23 Absent inferior vena cava. Suprarenal inferior vena cava drains into azygos vein.
had an aneurysm of the retrohepatic vena cava. The patient Sweeny et al. reported presented with thrombosis of an infrarenal vena cava aneurysm following strenuous exercise.

Arteriovenous malformations

Congenital arteriovenous malformations (AVMs) result from anomalous development of the primitive vascular system. AVMs are usually present at birth although signs and symptoms may not be manifest until later in life. Associated with many different syndromes, AVMs have multiple clinical presentations (Table 1.2). Progression is usually the result of hemodynamic factors because tumor-like behavior with endothelial proliferations is not characteristic.

In AVMs, the pathologic vasculature is mixed arteriovenous. The amount of blood shunted through the abnormal vessels and the resultant hemodynamic factors determine the secondary morphologic changes in the feeding arteries and draining veins.

A rare, congenital venous anomaly is an aneurysm of the inferior vena cava. In Sweeny et al.’s review, only three cases had been reported before 1990: two patients had aneurysms of the supradiaphragmatic inferior vena cava and one patient had an aneurysm of the retrohepatic vena cava. The patient Sweeny et al. reported presented with thrombosis of an infrarenal vena cava aneurysm following strenuous exercise.

![Figure 1.24 Circumaortic renal collar.](image)

**Table 1.1 Incidence of major inferior vena caval and renal vein anomalies**

<table>
<thead>
<tr>
<th>Venous anomaly</th>
<th>Incidence percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Circumaortic renal collar</td>
<td>1.5–8.7</td>
</tr>
<tr>
<td>Double inferior vena cava</td>
<td>2.2–3.0</td>
</tr>
<tr>
<td>Posterior left renal vein</td>
<td>1.8–2.4</td>
</tr>
<tr>
<td>Left inferior vena cava</td>
<td>0.2–0.5</td>
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Schwartz and colleagues reviewed 185 patients at the Mayo Clinic with AVMs of the extremities and pelvis. Lesions