

ULTRA P.A.S.S. Vascular Technology Registry Review Workbook 7th Edition

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for the Ultrasound Registry*

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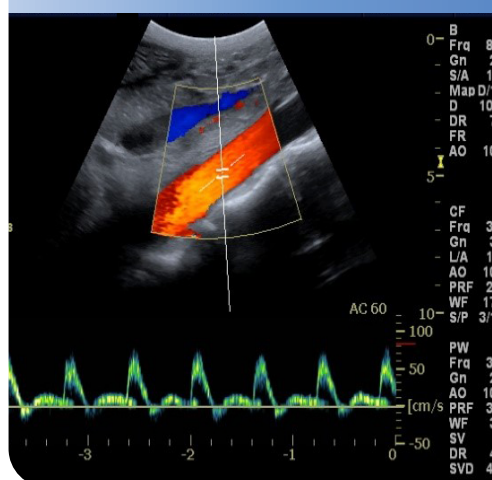
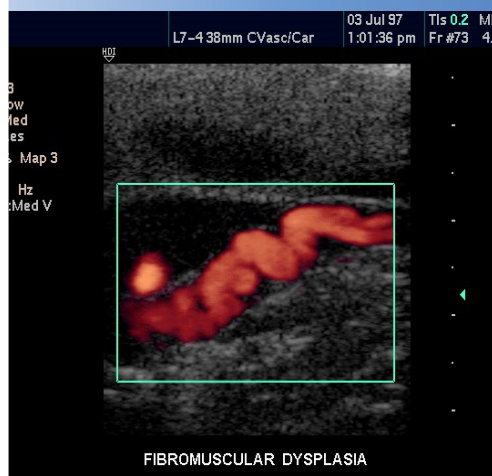
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and
Mark Swanson, RDMS, RVT**



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ULTRA P.A.S.S.
Vascular Technology
Registry Review Workbook
7th Edition

Featuring the Online Media Center
Containing Video Clips and Audio Narration

Written by
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EDUCATIONAL NEEDS STATEMENT:

The planning committee has determined a need for this educational activity based on request from the medical community, expanded utilization of ultrasound, and lab accreditation requirements.

ENDURING MATERIALS COURSE OBJECTIVES:

Please see opening statements at each section.

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Vascular Technology Registry Review Workbook

Seventh Edition

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Section 1: CEREBROVASCULAR

Objectives

Upon completion of this module you should be able to:

- ◆ Recognize cerebrovascular anatomy and physiology
- ◆ Identify normal imaging and Doppler characteristics of the cerebrovascular system
- ◆ Outline routine carotid duplex/color scanning protocols
- ◆ Discuss patient presentation and common indications for ultrasound examination of the cerebrovascular system
- ◆ Recognize characteristics of commonly seen carotid artery disease
- ◆ List the various types of treatment for carotid artery disease
- ◆ Compare the testing methods, interpretation, and limitations of arteriography, MR angiography, and computed tomography



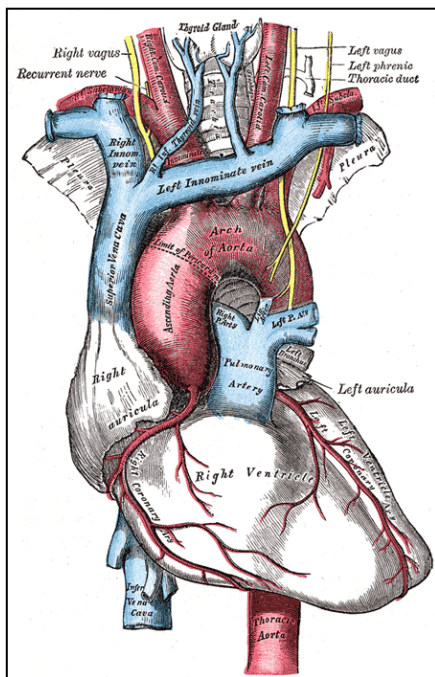
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CEREBROVASCULAR

ANATOMY/PHYSIOLOGY

The aortic arch commences at the upper portion of the left ventricle and consists of the ascending aorta, the arch, and descending aorta. There are three main branches of the aortic arch. The first branch is the innominate or brachiocephalic artery. It divides into the right common carotid and the right subclavian artery. The second branch of the aortic arch is the left common carotid artery followed by the left subclavian artery.



Aortic Arch and Branches

Diagram used with permission from Gray's Anatomy, Bartleby.com

axillary artery runs to the lower border of the axilla and is bordered in front by the pectoral muscle, anterior thoracic nerve, acromio-thoracic and cephalic veins.

The **brachial artery** arises from the lower axilla to the level of the elbow. It then bifurcates into the radial and ulnar arteries.

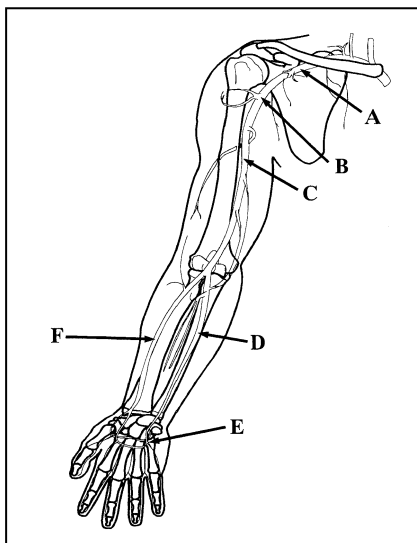
The **radial artery** courses along the lateral aspect of the forearm (with the patient in the anatomical position) or the radial side of the forearm down to the wrist.

The **ulnar artery** courses along the medial side of the forearm along the ulnar border. It is the larger of the two terminal branches of the brachial artery.

The **palmar arch** is formed by the ulnar artery in the hand and is completed by anastomosing with the branch of the radialis indicis. The palmar arch courses over the palm in a curve to the space between the index finger and thumb. There are four digital branches which lie superficial to the flexor tendons and course between the fingers. The digital branches provide blood supply to the fingers.

UPPER EXTREMITY ARTERIES

The **subclavian arteries** are located posterior to the clavicle and scalene muscles. The right subclavian artery branches off of the innominate artery and the left subclavian artery arises directly off of the aortic arch. The subclavian artery forms the axillary artery at the first rib. The



- A. Subclavian Artery
- B. Axillary Artery
- C. Brachial Artery
- D. Ulnar Artery
- E. Palmar Arch
- F. Radial Artery

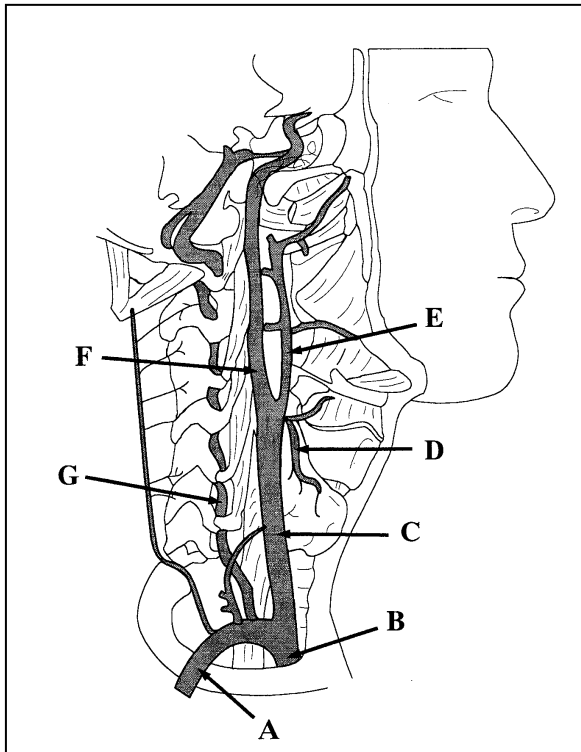
CERVICAL CAROTID/VERTEBRAL

The **common carotid artery (CCA)** is located lateral to the trachea and thyroid gland. The right common carotid artery arises from the innominate artery and the left common carotid artery originates directly from the aortic arch. The origin of the left common carotid artery varies more often than the right. It may arise off the innominate artery or both common carotid arteries can arise from a single trunk when there is an absent innominate artery. The common carotid artery courses up the lateral aspect of the neck to the superior aspect of the thyroid where it bifurcates into the internal and external carotid arteries.

The **internal carotid artery (ICA)** provides blood supply to the anterior portion of the brain, eye, and its appendages with branches to the forehead and nose. The internal carotid artery is usually located posterolateral to the external carotid artery.

The ICA is divided into four segments: The **cervical portion** begins at the bifurcation, opposite the upper border of the thyroid. It courses in front of the cervical transverse processes to the carotid canal. No branches are typically seen in the cervical ICA. The **petrous portion** is where the vessel enters the carotid canal. The **cavernous portion** is where the ICA is located between the layers of the dura mater forming the cavernous sinus. This cerebral segment is in the portion of the ICA passing between the second and third cranial nerves at the inner extremity of the sylvian fissure where it gives rise to its terminal branches. The ICA typically has no cervical branches. The first major branch is the **ophthalmic artery**. The ophthalmic artery enters the orbit through the optic foramen and divides into its terminal branches, the frontal, nasal, and supraorbital arteries.

The anterior cerebral and middle cerebral arteries arise from the internal carotid artery. The anterior cerebral artery connects with the opposite artery by the anterior communicating artery. The anterior and middle cerebral arteries serve as the terminal branches of the internal carotid artery.



- A. Right subclavian artery
- B. Innominate artery
- C. Common carotid artery
- D. Superior thyroid artery
- E. External carotid artery
- F. Internal carotid artery
- G. Vertebral artery

The **external carotid artery (ECA)** provides blood supply to the face and the scalp. It does not normally provide blood supply to the brain, but the ECA branches can become collateral pathways if an occlusion occurs in the ICA or vertebral arteries.

The ECA commences at the upper border of the thyroid cartilage. There are usually eight branches of the ECA within the neck. The most vital are those which interconnect with the ophthalmic, occipital, and vertebral arteries. The first branch of the ECA is the **superior thyroid artery**. The **lingual artery** is an anterior branch arising between the superior thyroid and facial arteries, and

provides blood supply to the tongue. The **facial artery** arises just above the lingual artery. It courses along the lower mandible, across the cheek to the angle of the mouth. It continues superior along the side of the nose to the inner canthus of the eye. It is then referred to as the angular artery. The **facial/angular artery** provides blood supply to the tongue, lips, nose, and lacrimal sac.

The **occipital artery** arises from the posterior portion of the ECA opposite the facial artery. It has important communication with the muscular branches of the vertebral artery.

The **posterior auricular artery** originates above the digastric and stylohyoid muscles opposite the apex of the styloid process. It gives rise to three branches, which supply the membranous tympani, back of the ear, and muscle.

The **ascending pharyngeal artery** is the smallest branch of the ECA and usually arises at the level of the carotid artery bifurcation. Its four branches provide blood supply to the longus coli muscle, lymph glands, palate, typani, and dura mater.

The **superficial temporal artery** is the smaller of the two terminal branches of the ECA. It arises between the neck and the lower jaw and external auditory meatus. It divides into the anterior temporal and posterior temporal arteries providing blood supply to the muscles and supraorbital rim.

The **internal maxillary artery** is the larger of the two terminal ECA branches. It arises at the level of the parotid gland opposite the neck of the condyle of the lower jaw. It is divided into three segments: the maxillary, pterygoid, and spheno maxillary.

The **vertebral arteries** arise from the first segment of the subclavian artery. They enter the transverse cervical foramina coursing up the posterior aspect of the neck. The arteries then pass through the atlas, winding around and passing beneath

the posterior atlantooccipital ligament. The vertebral arteries pierce the dura mater and arachnoid, entering the skull through the foramen magnum. Both vertebral arteries join in the midline at the lower border of the Pons Varolii to form the basilar artery. The vertebral arteries provide blood supply to the posterior aspect of the brain. Branches of the vertebral arteries supply the medulla and the interior surface of the cerebellum.

INTRACRANIAL

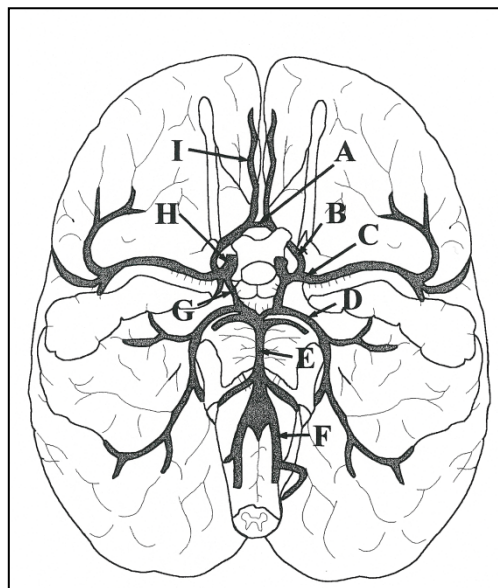
The **Circle of Willis** is formed in front by the anterior cerebral arteries (ACA), and in back by the two posterior cerebral arteries. The ACA branches of the ICA are connected by the anterior communicating artery and the branches of the basilar artery are connected by the posterior communicating arteries to the middle cerebral arteries.

Three trunks arise from the Circle of Willis and together supply each cerebral hemisphere. These include the two anterior cerebral arteries, middle cerebral arteries, and the posterior cerebral arteries.

The Circle of Willis is an important source of collateral circulation. There are about nine congenital abnormalities that may occur with the most common malformation being absence or hypoplasia of one or more of the communicating arteries (Pellerito / Polak 130).

The vessels of the **central ganglionic system** arise from the Circle of Willis or from vessels immediately after their origin from the Circle of Willis.

The **cortical arterial system** is formed by vessels, which are terminal branches of the anterior, middle, and posterior cerebral arteries. They give rise to nutrient arteries that penetrate the cortex.



- A. Anterior communicating artery
- B. Anterior cerebral artery (A-1 Pre-commun)
- C. Middle cerebral artery
- D. Posterior cerebral artery
- E. Basilar artery
- F. Vertebral artery
- G. Posterior communicating artery
- H. Internal carotid artery
- I. Anterior cerebral artery (A-2 Post-commun)

COLLATERAL POSSIBILITIES

The brain is supplied by two types of arterial branches. The **penetrating arteries** provide nutrient supply to the central nervous system to support neuronal function. The **superficial or circumferential arteries** are major arterial trunks that spread over the entire surface of the cerebral hemispheres, brain stem, and spinal cord.

There are three categories of intracranial collateral circulation. **Large interarterial connections** are formed by the Circle of Willis. This is a major collateral pathway that provides communication between two common carotid arteries or between the

basilar artery and the right or left common carotid artery.

The second most important source of collateral circulation is via the **intra-cranial extracranial pathway**. This is formed by an anastomosis between the ECA and ICA via orbital and ophthalmic and other ECA/ICA connections. The most important ECA branches are those which communicate with branches of the ophthalmic artery and include: the facial with the nasal, superficial temporal to the supraorbital, the internal maxillary to the ophthalmic, the occipital with the vertebral, and the ascending pharyngeal to the vertebral artery. Other possible connections include the occipital artery with the atlantic branch of the vertebral or the deep cervical and ascending cervical branches of the subclavian with the vertebral artery. The intracranial- extracranial connections are also known as the **pre-Willisian anastomosis**.

The third source of collateral circulation is via the small interarterial communications. The **Rete Mirabile** or “wonderful net” is a transdural anastomosis crossing the subdural space from the dural arteries on the surface of the brain.

There is no effective anastomosis between neighboring cerebral arterial branches, deep penetrating arteries, or superficial and deep cerebral arteries.

MECHANISMS OF DISEASE

Risk Factors

Non-invasive evaluation of the cerebrovascular system is performed on patients with asymptomatic bruits, history of TIA, or stroke, non-localizing symptoms, and patients undergoing a major cardiac or vascular procedure.

Stroke is a devastating event and is the 3rd leading cause of death in the United States (Young 288). There are several risk factors

that contribute to the development of atherosclerosis and subsequent stroke.

High blood pressure is the number one risk factor associated with stroke patients.

The theory is the accelerated flow rates seen with high blood pressure will increase the rate of damage to the vessel wall at the carotid bifurcation where there is already some degree of continual wear and tear due to turbulence. Additionally, if the patient has existing stenotic disease, high blood pressure can cause plaque components to become dislodged resulting in an embolic event.

Diabetes is a highly recognized risk factor in the development of atherosclerosis.

Smoking is well documented in the literature as a source of damage to the vascular system, lungs, heart, and other organs. Other risk factors include obesity, family history of vascular disease, hyperlipidemia, and stress.

Atherosclerosis/Thromboembolic Event

The mechanisms for extracranial disease include stenosis, thrombosis, embolism, and cardiac disease. **Stenosis** is a narrowing of the vessel lumen usually due to an atherosclerotic plaque. An embolic event can occur due to plaque ulceration and fibrinoplatelet material breaking off to embolize distally. **Thrombus formation** can occur when there is stagnation of blood flow. Components of the thrombus can embolize distally or totally occlude the vessel.

The most common source of stroke is due to an ischemic event, usually carotid bifurcation disease, but can also be due to cardiac disease. Some cardiac sources of an embolic event include:

- atrial fibrillation
- left ventricular dysfunction
- bacterial endocarditis
- and prosthetic heart valves

Section 1

Up to 10% of strokes are due to intracerebral hemorrhage or subarachnoid hemorrhage (Young 288).



Play Video Clip 1A: Carotid Artery Stenosis

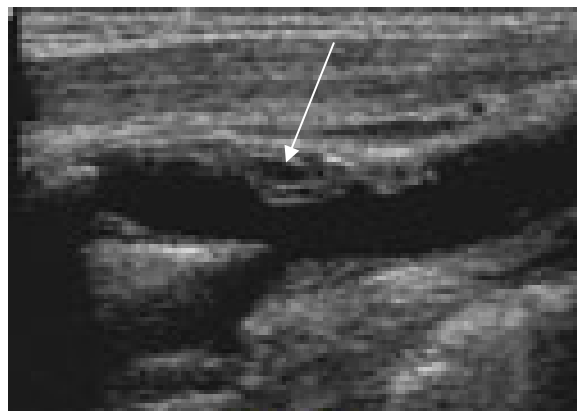
Atherosclerosis is a progressive disease where fat deposits (lipids), calcifications, and fibers develop in the intima and media of the arterial wall.

The permanent form of atherosclerosis begins as a fibrous plaque, primarily lipid in content surrounded by an elastic and collagenous tissue. The plaque is generally uniform with smooth walls. Progression of the disease leads to a complicated plaque with areas of necrosis and surface irregularities. **Plaque ulceration** can be the source of an embolic event or thrombus formation.



Play Video Clip 1B: Plaque progression

An **intraplaque hemorrhage** occurs when there is bleeding into the plaque. These types of plaques are very unstable and may rupture sending a shower of emboli that travel distally or cause rapid occlusion of the vessel.



Intraplaque Hemorrhage

Dissection

Arterial dissection occurs when blood flows through a tear in the media causing a separation of the intima and sometimes the media creating a new or false lumen.

The most common cause of dissection is weakening of the arterial media associated with the aging process. Other etiologies include Marfan's syndrome, Fibromuscular dysplasia, chiropractic manipulation, and blunt trauma.

Patient presenting symptoms include:

- headache
- neck/face pain
- Hemispheric symptoms
- Cranial nerve palsy

Dissections are classified according to the DeBakey Models:

Type I: Dissection begins at the root extending the entire length of the arch and into the abdomen. Ascending and descending aorta involved. Type II: involves ascending aorta only. Type I and II are considered to be the most dangerous especially if it extends to the common carotid artery and subclavian artery.

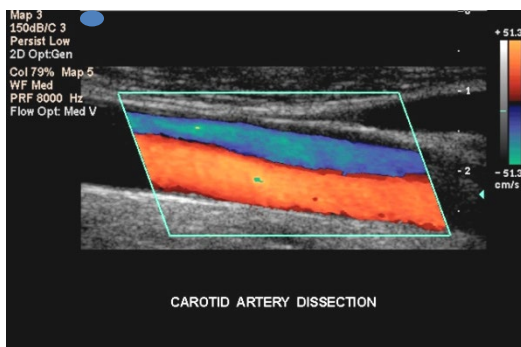
Most are hereditary, secondary to Marfan's disease which causes a weakening of the arterial wall. Marfan's disease is a

connective tissue disorder commonly seen in tall, thin, double-jointed individuals.

Subclavian Level: These extend to the descending aorta, but may or may not continue into the abdomen.

Type III: Begins at the lower descending aorta and extends into the abdomen.
Descending aorta only.

Sonographically, aortic dissection will reveal two lumens with an echogenic intimal flap. Doppler/color evaluation may be helpful in documenting higher resistant flow in the false lumen.



Carotid artery Dissection



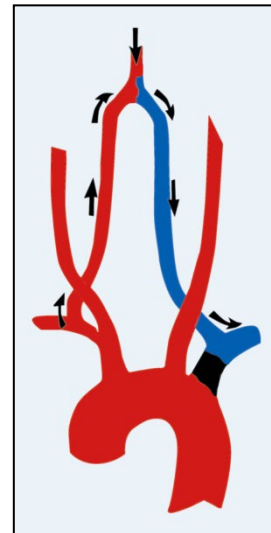
**Play Video 1C:
Carotid Dissection**
(Courtesy of Patrick Washko, RVT)

Subclavian Steal

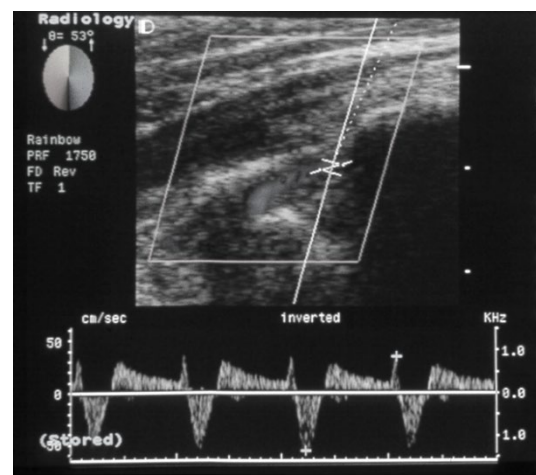
Subclavian steal occurs when there is a flow-reducing lesion in the subclavian or innominate artery *proximal* to the origin of the vertebral artery. The reduced flow to the arm results in a brachial pressure drop on the affected side. Flow is reversed in the vertebral artery on the side with a pressure drop to serve as a collateral vessel to

supply the arm. Therefore, reverse flow in the vertebral artery is indicative of subclavian steal.

Some patients will demonstrate a waveform alternating between forward and reverse flow. This is an intermediate stage where the subclavian artery stenosis is not causing a sufficient pressure gradient to produce continuous reversal of flow.



Subclavian Steal



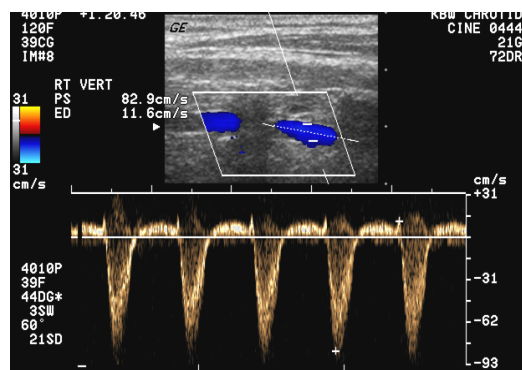
Subclavian Steal: Alternating Flow Pattern

Subclavian steal is more commonly seen in men and most often occurs on the left side. The patients are frequently asymptomatic

Section 1

and the diagnosis is an incidental finding during a cerebrovascular exam.

Symptomatic patients may experience arm claudication (pain with exertion) due to a lack of blood supply to the muscle during exercise and/or they may experience vertigo since blood supply is stolen from the posterior circulation.



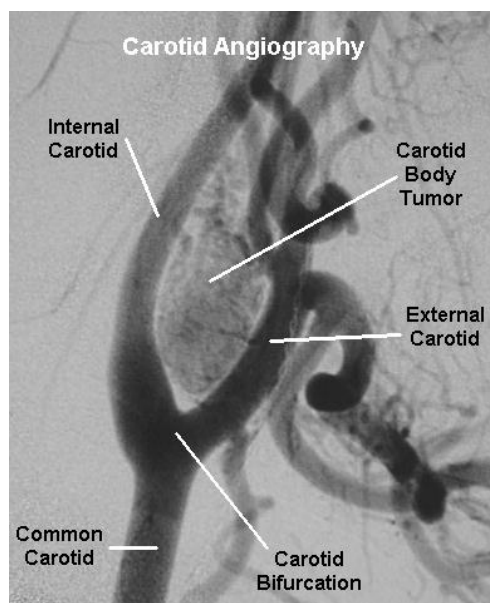
Complete Subclavian Steal

Carotid Body Tumor

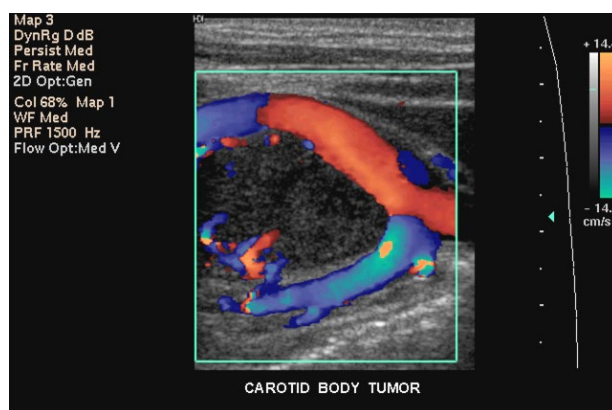
The normal carotid body is located in the adventitia of the carotid bifurcation, measuring approximately 1 – 1.5 mm in size.

Carotid body tumors are relatively rare highly vascular tumors with presenting symptoms of a palpable neck mass, headache, and neck pain. These tumors are usually benign and exhibit relentless growth.

Sonographically, the mass will be seen separating or “splaying” the ICA and ECA. Color or spectral Doppler will reveal a high degree of vascularity. In some situations, the mass may totally surround the ECA or ICA. Surgical resection is the usual treatment to prevent other associated complications.



Carotid Body Tumor



Carotid Body Tumor

Fibromuscular Dysplasia (FMD)

Fibromuscular dysplasia is a non-inflammatory disease of the arteries of unknown etiology. FMD is typically identified in the mid to distal segments of a vessel and occurs more frequently in women. It is most commonly seen in the renal arteries (85%). The ICA is the second most common location for FMD. Symptoms include TIA and thromboembolic stroke. The most common referral for evaluation is asymptomatic cervical bruit (Daigle 64).

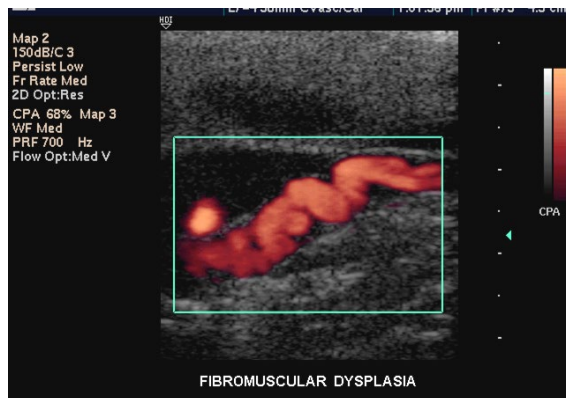
There are 4 types of FMD defined according to the layers of the vessel involved. The subtypes include:

- Intimal Fibroplasia
- Medial Fibroplasia
- Medial Hyperplasia
- Perimedial Dysplasia

Intimal and medial fibroplasia are predominantly seen in the ICA, usually adjacent to C1-C2 vertebral bodies.

The sonographic and angiographic appearance is usually described as a “string of beads”, seen as alternating dilation and narrowing of the vessel.

Fibromuscular dysplasia may be symptomatic or asymptomatic and treatment ranges from observation to surgical intervention.



Fibromuscular Dysplasia

Neointimal Hyperplasia

Neointimal hyperplasia is due to an overgrowth of endothelial cells on the intimal surface after surgery. Smooth muscle and fibrous overgrowth is the most common cause of restenosis.

With atypical or nonatherosclerotic disease such as Fibromuscular Dysplasia or Takayasu disease, standard velocity criteria does not apply.

SIGNS AND SYMPTOMS

Presenting symptoms are helpful in determining the location of disease. ICA territory symptoms are usually unilateral hemiparesis or hemiparesthesia. The disease in this case will be on the contralateral side since the right brain controls the left side of the body and vice-versa.

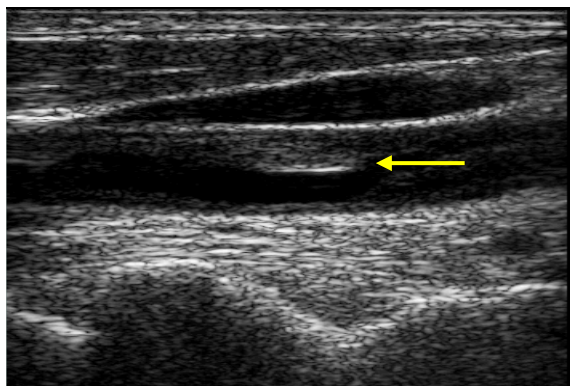
A classic sign of ICA territory ischemia is **amaurosis fugax**. This is a transient loss of vision in one eye. It is commonly described as a shade being pulled down over the eye, either from the top down or the bottom up. The visual disturbance is caused by embolic central retinal artery occlusion or a retinal cholesterol emboli called a **Hollenhorst plaque**. The disease in this instance will be on the same side as the symptoms since the ophthalmic artery is the first major branch of the ICA.

Other possible symptoms include language or communication defects or headaches in the supraorbital or temporal region on the same side of disease.

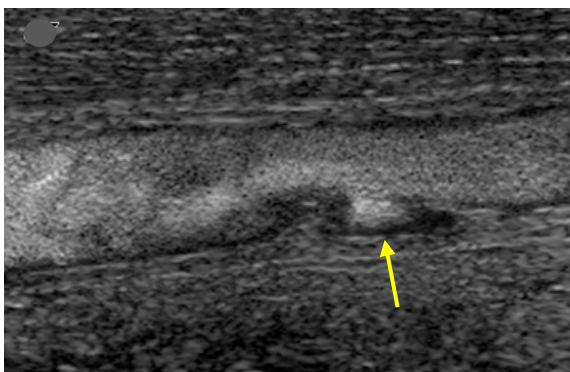
Vertebrobasilar symptoms are generally bilateral. These include:

- Ataxia
- Limb weakness
- Drop attacks
- Visual disturbances
- Vertigo
- Dysarthria
- Headaches
- Vomiting

Vertigo and syncope are two common presenting symptoms and are considered to be non-localizing since they can be due to ICA territory ischemia, vertebrobasilar disease, cardiac disease, or other medical problems.



Arterial Plaque



Arterial Ulceration
(Photo courtesy of GE Medical)

Embolus

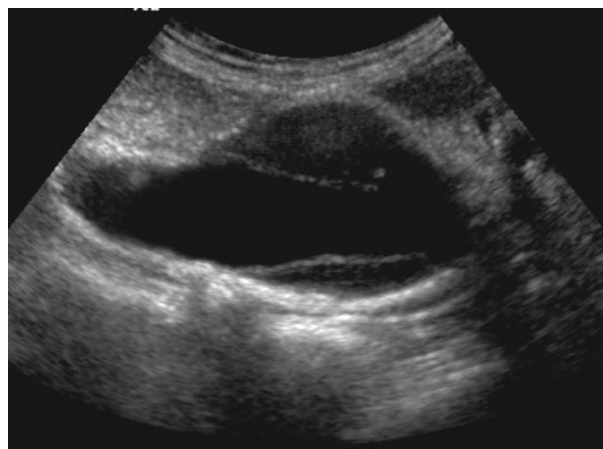
An embolus is due to a piece of plaque, thrombus, or platelet aggregate which dislodges, travels within the vessel, and may obstruct a distal vessel of smaller size. For example, blue toe syndrome occurs when an embolus obstructs the small vessels of the foot and causes ischemia of the toe.



Blue toe syndrome

Aneurysm

An aneurysm is an abnormal dilation of the 3 layers of the vessel wall. This dilation can be focal or diffuse. The most common location is the infrarenal aorta. The exact cause is not clearly understood but contributing factors include genetic predisposition, cigarette smoking, and elevated blood pressure. They are commonly associated with atherosclerotic changes even though there is no evidence that the actual atherosclerotic changes are a direct cause. A thrombus can form within the vessel lumen and result in an embolic event or occlusion. The larger the aneurysm, the greater the risk of rupture.



Abdominal Aneurysm

Non-Atherosclerotic Lesions

There is a group of non-atherosclerotic diseases, which can be the source of ischemic disease, particularly in those patients with upper extremity symptoms or those who are at low risk for atherosclerosis.

Fibromuscular Dysplasia

Fibromuscular dysplasia is a non-atherosclerotic, non-inflammatory disease that involves small and medium sized arteries. Women are affected three times more commonly than men, and the disorder usually presents in adults 25 to 50 years old

Section 4: ABDOMEN / VISCERAL

Objectives

Upon completion of this module you should be able to:

- Identify normal abdominal vasculature and recognize commonly seen anatomical variations
- Recognize normal abdominal vasculature Doppler waveform characteristics and routine measurements
- Recall limited scanning protocols for the ultrasound evaluation of the abdominal aorta, IVC, hepatic, portal-splenic mesenteric circulation.
- Recognize imaging and Doppler characteristics of commonly seen pathology of the abdominal vasculature



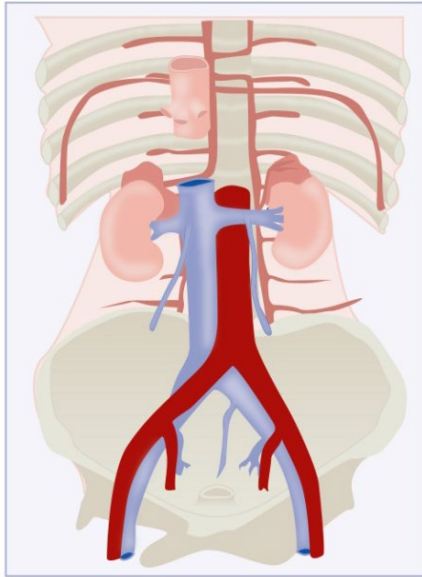
When you see this Video Screen Icon in a chapter paragraph, play the indicated video clip from the **On-line Media Center**. You can view them on your computer/smartphone or web-enabled device

1. Point your browser to <https://www.gcus.com>
2. Log into your account
3. Select "My Activities" from the top menu
4. Click the "Media Center" button for: **PAU-OL7-VT7**

ARTERIAL ANATOMY

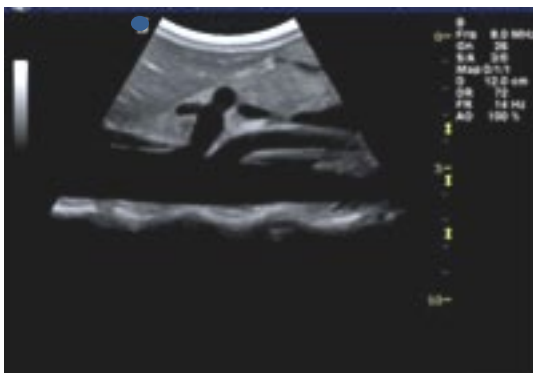
ABDOMINAL AORTA & BRANCHES

The **abdominal aorta** begins after it passes through the diaphragm. It lies to the left of midline, coursing down to the level of L-4 where it bifurcates into the left and right common iliac arteries.



Abdominal Aorta/IVC

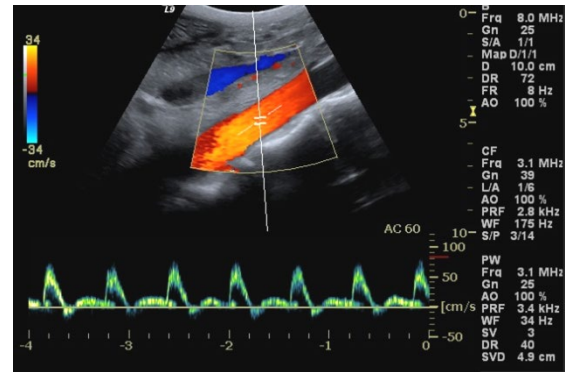
The normal diameter of the abdominal aorta is < 3 cm. Normal values vary with age, progressively increasing from childhood to adulthood. (Pollerito / Polak 450).



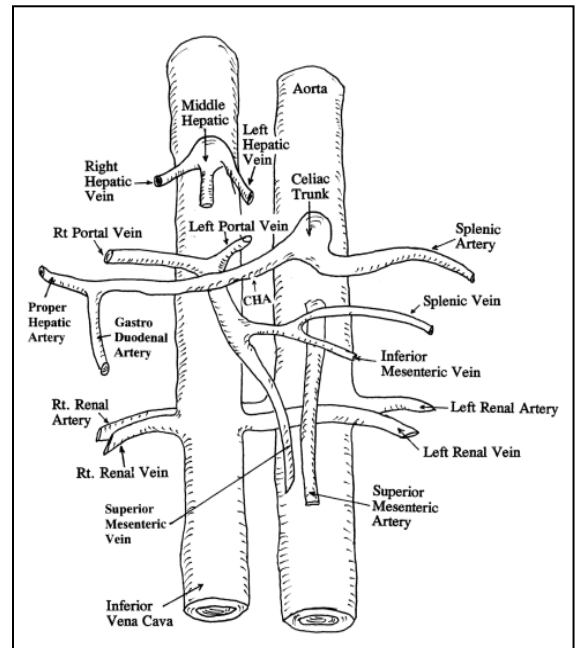
Normal Proximal Aorta

The average velocity in the adult patient ranges from 70 to 110 cm/sec with proximal

assessment displaying a triphasic waveform pattern with noticeable forward flow during diastole, which is a reflection of the low resistance vascular beds of the liver, spleen, and kidneys. Waveforms below the renal arteries will show a typical "peripheral" waveform (high resistance) pattern with a more noticeable flow reversal in early diastole.



Normal Abdominal Aorta Spectral Doppler



Abdominal Vasculature

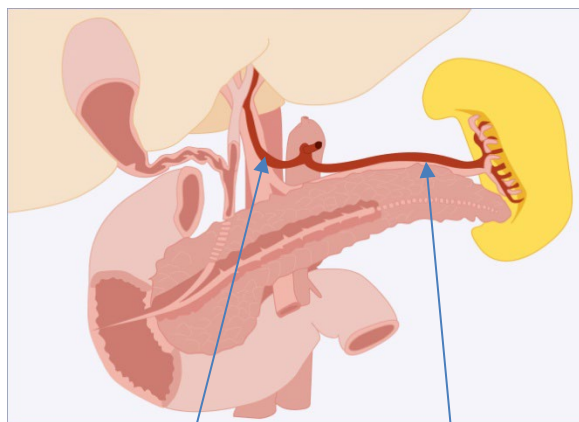
The first branch of the aorta is the **celiac artery**. It arises anteriorly off the aorta just below the level of the xyphoid process. The celiac artery divides into the common hepatic artery, splenic artery and the left

Section 4

gastric artery. The **common hepatic artery (CHA)** courses towards the right. It lies anterior to the portal vein and adjacent to the CBD. As it courses towards the porta hepatis, the CHA gives rise to the gastroduodenal artery and then changes names to the proper hepatic artery.

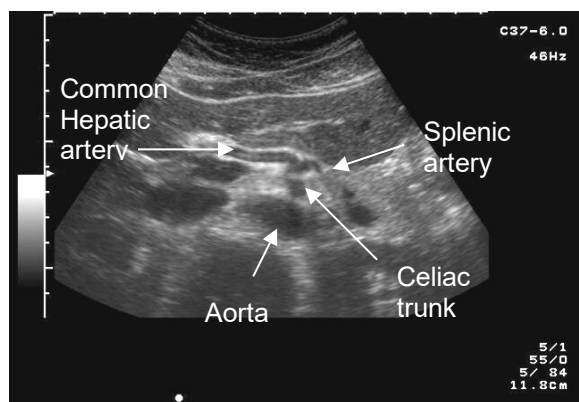
The **proper hepatic artery** enters the liver at the porta hepatis, where it divides into right, middle and left hepatic arteries.

The **right hepatic artery (RHA)** is generally seen lying anterior to the PV and posterior to the bile duct.



Celiac, Common Hepatic and Splenic Arteries

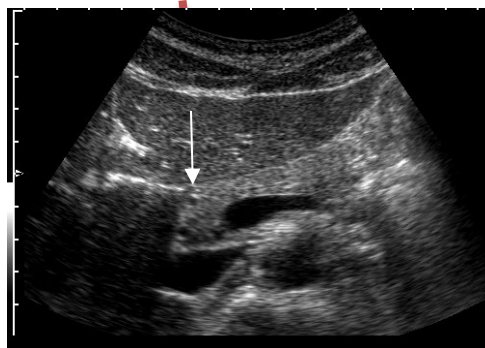
Anatomical variations include the common (4%) or right (11%) hepatic artery arising from the SMA instead of the celiac artery (Pollerito / Polak 441).



Celiac trunk and two of its branches

The **gastroduodenal artery (GDA)** courses caudally towards the posteromedial

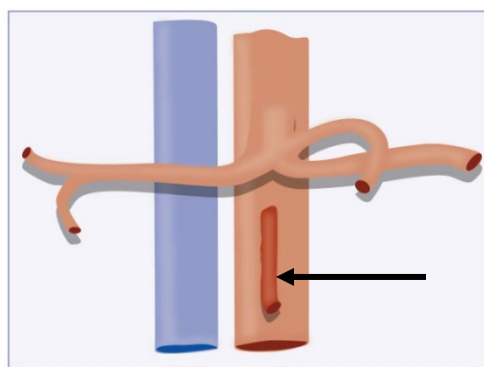
segment of the duodenum and the anterolateral aspect of the pancreatic head.



Gastroduodenal Artery

The **superior mesenteric artery (SMA)** is the second branch of the aorta arising about 1 cm below the level of the celiac artery. It arises anteriorly off the aorta at a slight angle and then courses inferiorly to provide blood supply to the small bowel, portions of the large intestine and pancreatic head. The SMA is located posterior to the body of the pancreas.

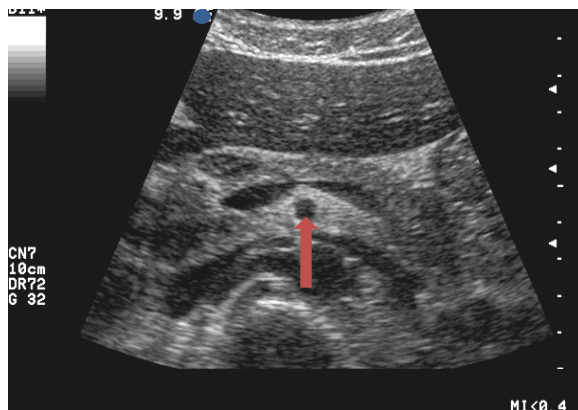
The SMA courses parallel to the aorta. Normally there should not be a separation of more than 1.1 cm between the anterior wall of the aorta and the posterior wall of the SMA. Increased separation or an angle of origination > 15 degrees is suspicious for adenopathy (Hagen-Ansert 110).



Superior Mesenteric Artery

The SMA has a classic sonographic appearance in the transverse scan plane seen as a round circular vessel surrounded by a bright white echogenicity. This has

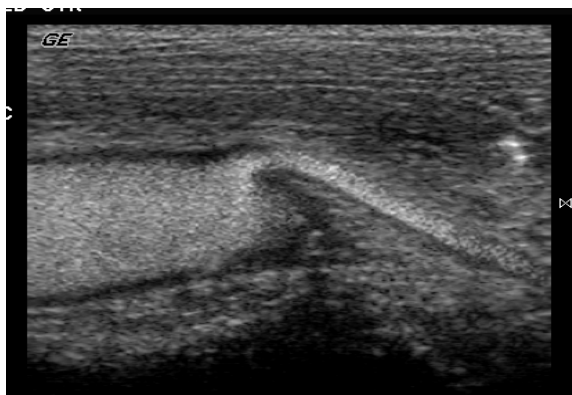
been referred to as the “bulls-eye sign”, “target-sign” and “donut sign”.



Transverse Superior Mesenteric Artery

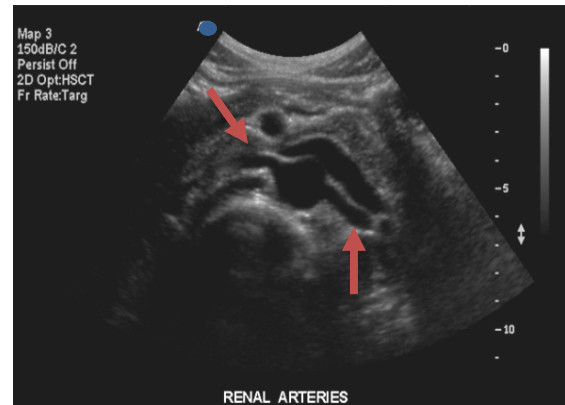
The **Inferior Mesenteric Artery (IMA)** arises from the anterior aspect of the abdominal aorta at the level of L-3 or L-4. It provides blood supply to the descending colon, sigmoid, and rectum. It also serves as a potential collateral pathway when mesenteric ischemia exists.

When seen, longitudinal scans will demonstrate a small vessel arising from the anterior aspect of the abdominal aorta just inferior to the level of the SMA.

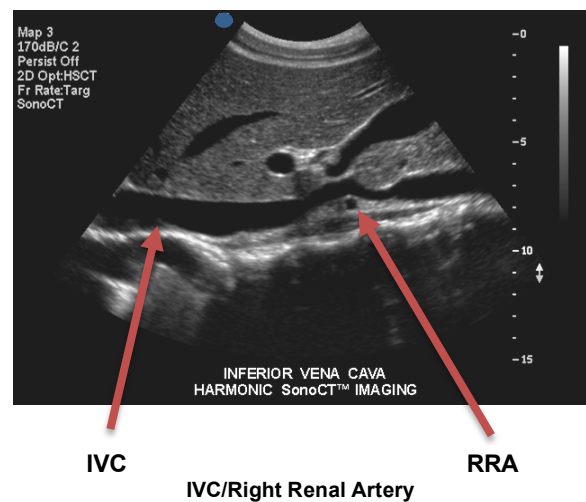


IMA

The **renal arteries** arise about 1 cm below the level of the SMA. The right renal artery courses posterior to the IVC before entering the right kidney.

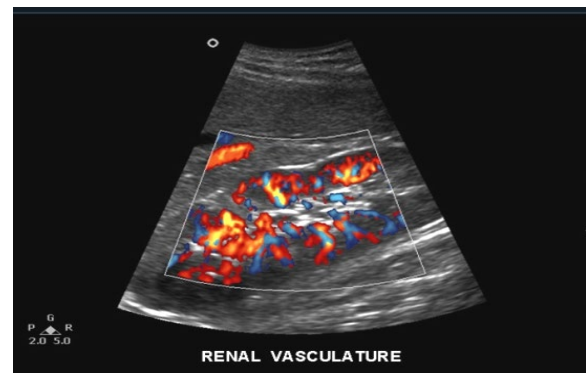


Transverse Aorta, SMA, RRA, LRA, LRV



IVC
IVC/Right Renal Artery
RRA

The left renal artery branches lateral off the aorta entering directly into the left renal hilum. Approximately 12 - 22% of patients will have duplicate renal arteries (Pellerito / Polak 527).



Renal Perfusion

After entering the renal parenchyma, the segmental arteries form the interlobar arteries. The **interlobar arteries** course toward the cortex and form the arcuate arteries. Tiny interlobular arteries branch from the arcuate arteries and course out to the periphery of the kidney.

MECHANISMS OF DISEASE: ARTERIAL

Risk Factors: Arterial Disease

Leading risk factors, which contribute to the development of arterial disease, include diabetes, hypertension, hyperlipidemia, and smoking.

Diabetics frequently have hardening of the arterial wall and a loss of elasticity. Diabetics also frequently have isolated occlusions of the vessels in the lower leg.

Hypertension causes an increase in intraluminal arterial wall stress and contributes to the development and/or progression of atherosclerosis.

Smoking causes an increase in the levels of carbon monoxide in the blood that irritates the endothelial lining of the vessel wall.

Hyperlipidemia also is a contributing factor to the development of atherosclerosis due to the high saturation of lipid fats in the blood.

Renovascular hypertension accounts for 5% - 10% of the hypertensive population. Renovascular hypertension is considered in patients who consistently have a diastolic blood pressure greater than 105 – 115 mm/hg without medication. It is most commonly due to atherosclerosis, but may also be due to fibromuscular dysplasia.

The kidneys have a primary role in blood pressure regulation by its influence on circulating plasma volume and vasomotor tone. The **renin-angiotensin system** is a

complex mechanism that normally maintains homeostasis by regulating the body's sodium potassium balance, fluid volume, and blood pressure under changing physiologic conditions. The juxtaglomerular apparatus is comprised of richly innervated smooth muscle cells that lie along the afferent arterioles. These cells are very sensitive monitors of perfusion pressure. Any condition that decreases this pressure will stimulate the release of renin into the renal vein and systemic circulation.

Renal artery stenosis can activate the renin-angiotension system that leads to renovascular hypertension.

Mesenteric ischemia is a life threatening condition that occurs when there is obstruction of any of the mesenteric vessels. The most serious conditions occur with occlusion of the SMA that results in ischemia of the small bowel and is considered to be a vascular emergency.

Mesenteric ischemia can be an acute or chronic condition. **Acute intestinal ischemia** is usually due to an embolic or thrombotic event. Chronic ischemia is due to progressive atherosclerosis, involving the origin of the celiac and/or SMA.

Acute embolic occlusion presents clinically with sudden onset of severe abdominal pain followed by vomiting or diarrhea. Abdominal distention may or may not be present. In addition, leukocytosis develops quickly.

Symptoms with chronic mesenteric occlusion develop more gradually and mimic a bowel obstruction. Instead of sudden severe pain, the patient usually presents with a history of post-prandial (after eating) pain, weight loss, and a change in bowel habits.

Chronic intestinal ischemia is due to a progressive development of atherosclerosis that allows for collateral channels to develop. If the collateral development is not