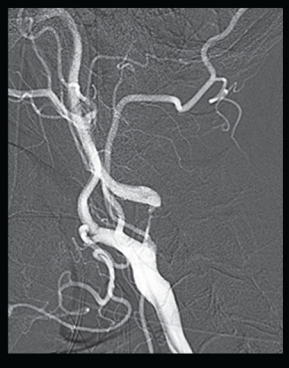
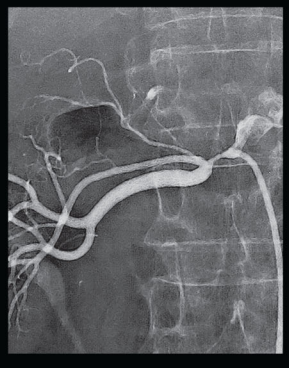
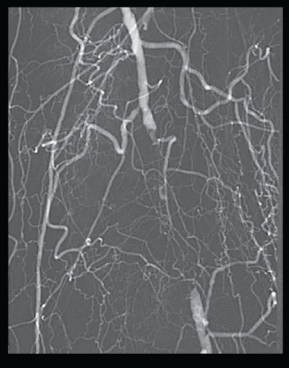
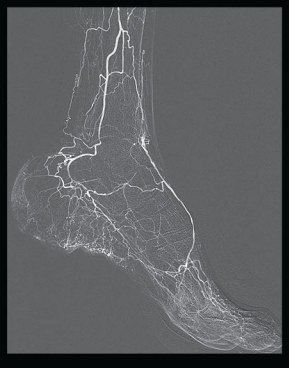
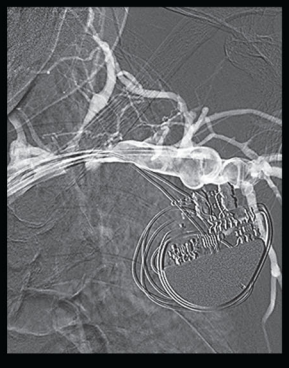
Carotid and Peripheral Vascular Interventions Textbook

Step-by-step technique

Thosaphol Limpijankit

CHAPTER 6

Carotid artery disease

Thosaphol Limpijankit

INTRODUCTION

Stroke is one of the main causes of mortality and morbidity worldwide, and causes substantial health burden. Approximately 15-20% of ischemic stroke is linked with atherosclerosis of the carotid arteries, typically stenosis which occurs at the bifurcation of the external and internal carotid arteries (1,2). Importantly, carotid atherosclerosis is frequently asymptomatic until the appearance of a fatal or disabling stroke occurs. Once patients develop symptoms [i.e., stroke or transient ischemic attack (TIA)], the risk of recurrent ipsilateral stroke is even higher, with the risk being highest immediately after the initial ischemic events. Moreover, nearly 80% of strokes occurs secondary to artery-to-artery embolization in the carotid distribution and may present as the initial event without warning (3). All of these reasons emphasize the need for early detection, treatment and prevention of carotid artery stenosis.

Medical, surgical, and endovascular treatments are commonly used to treat carotid artery stenosis. In symptomatic carotid stenosis, revascularization was found to have an incremental benefit over medical therapy in regards to preventing recurrent stroke (4,5). Carotid endarterectomy (CEA) was first performed by DeBakey in 1975 (6) and has been recommended as the standard treatment for carotid artery stenosis. However, there are limitations of CEA, such as patients' comorbidities, surgical complications and unfavorable neck anatomy. During the last decades, there has been a rise of endovascular techniques, more experienced operators, and new devices available. All of these components lead to a paradigm shift from CEA to carotid artery stenting (CAS).

ANATOMIC CONSIDERATION

It is essential that any operator contemplating CAS needs a thorough understanding about basic intracranial and extracranial circulation, as well as understanding about common anatomic variants.

AORTIC ARCH TYPES

It is important to identify the type of aortic arch as well as configuration concerning the great vessels, because these anatomic features affect procedure complexity. In normal anatomy, the most common aortic arch branching has separate origins for the brachiocephalic (or innominate), left common carotid artery (CCA), and left subclavian artery (Fig. 6-1 A). The second most frequent aortic arch branching uses a common origin for the brachiocephalic and left CCA (Fig. 6-1 B). In other variants, the left CCA begins directly at the brachiocephalic artery (Fig. 6-1 C). These last two patterns are also called a "bovine arch" (7). On rare occasions, a single brachiocephalic trunk beginning from the aortic arch can eventually split into bilateral subclavian arteries with a bicarotid trunk, though this aortic arch branching pattern is only found in cattle (Fig. 6-1 D).

Moreover, the aortic arch can be classified into 3 types, based on the relationship of the brachiocephalic (or innominate) artery to aortic arch. Type I aortic arch is characterized by all three great vessels originating from the outer aortic arch's curvature within the same horizontal plane (Fig. 6-2 A). For type II aortic arch, the brachiocephalic artery originates between the horizontal planes of the inner and outer of aortic arch curvatures (Fig. 6-2 B). For type III aortic arch, the brachiocephalic artery originates below the horizontal plane of the aortic arch's inner curvature (Fig. 6-2 C). The more inferior the origin of the treated artery (i.e., Type II or III aortic arch), the more difficult it is to obtain carotid artery access.

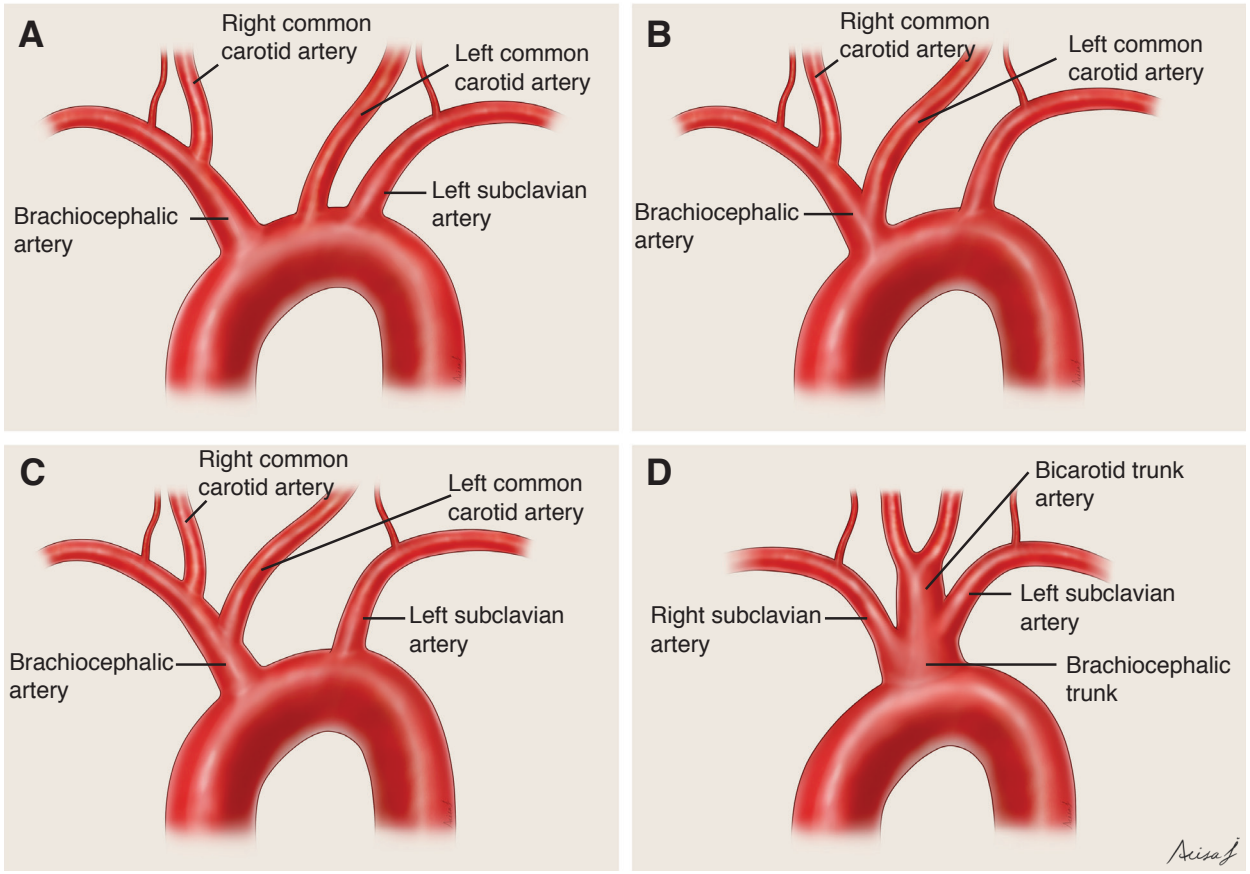


Figure 6-1. Types of aortic arch and variants. A: Normal aortic arch has separate origins for the brachiocephalic (innominate), left common carotid, and left subclavian arteries. B: Common origin for the brachiocephalic and left common carotid arteries. C: Left common carotid artery originates separately from the brachiocephalic artery. D: Single brachiocephalic trunk originates from the aortic arch that eventually splits into the bilateral subclavian arteries and a bicarotid trunk. (Redrawn from Layton KF, et al. Bovine Aortic Arch variant in Humans Clarification of a common misnomer . *AJNR Am J Neuroradiol*. 2006:27:1541-21, with permission from Williams & Wilkins co.)

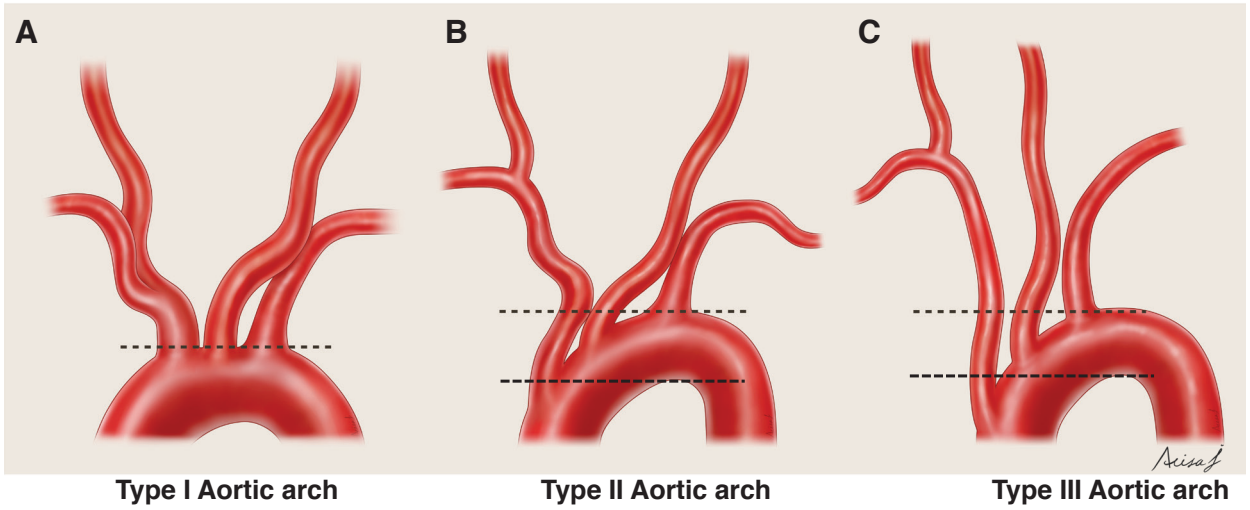


Figure 6-2. Three types of aortic arch. A: Type I aortic arch. The origins of all 3 great vessels are in the same horizontal plane (a dash line). B: Type II aortic arch. The brachiocephalic (or innominate) artery originates between the horizontal planes of the outer (a upper dash line) and inner curvatures (a lower dash line) of the aortic arch. C: Type III aortic arch. The brachiocephalic artery originates below the horizontal plane of the inner curvature (a lower dash line) of the aortic arch.

CAROTID ARTERY

The right CCA arise from the bifurcation of the brachiocephalic (innominate) artery, whereas the left CCA generally origins directly from the aortic arch. The CCAs ascend through the mediastinum, lying posterior to the sternoclavicular joints and medial to the internal jugular veins in the neck. The CCAs are in the carotid sheath, caudal (posterior) to the sternocleidomastoid muscle.

At the level of the superior border of the thyroid cartilage (normally at the C3-C4 vertebral level), each CCA bifurcates into an internal carotid artery (ICA) and external carotid artery (ECA) (Fig. 6-3). During diagnostic angiography, the mandible angle serves as a useful landmark for the carotid bifurcation, even though significant differences in the level of carotid bifurcation are common.

EXTERNAL CAROTID ARTERY

After dividing, the ECA leaves the sheath to provide blood supply to the neck and face, while the ICA proceeds in the carotid sheath to go into the carotid canal in the temporal bone. Understanding the anatomy of the ECA is essential because this vessel together with its branches are usually wired while performing a carotid intervention.

The ECA is readily identified, because of its many branches to the scalp, face, and thyroid (Fig. 6-4). These branches include:

- superior thyroid artery
- ascending pharyngeal artery
- lingual artery
- facial artery
- occipital artery
- posterior auricular artery
- internal maxillary artery
- superficial temporal artery.

Several of these ECA branches (i.e., occipital and internal maxillary) supply the dura of the lateral and basal brain surfaces. The ECA may supply essential intracranial flow if there is severe ICA stenosis or occlusion.

INTERNAL CAROTID ARTERY

The proximal ICA contains a bulbous dilatation, which is clinically significant because it functions as the place of the "carotid sinus," a baroreceptor , and the "carotid body," a chemoreceptor. The carotid body chemoreceptor can be affected by increased PCO_2, decreased PO_2, as well as decreased pH in the blood, and manages alerts to the brain to adjust respiratory rate.

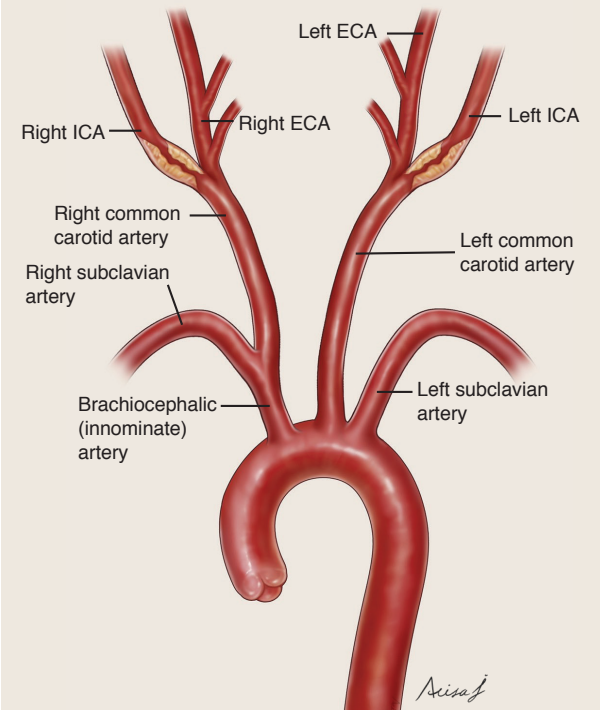


Figure 6-3. Anatomy of bilateral carotid artery with bifurcation into the internal carotid artery (ICA) and external carotid artery (ECA) at the C3-C4 vertebral level.

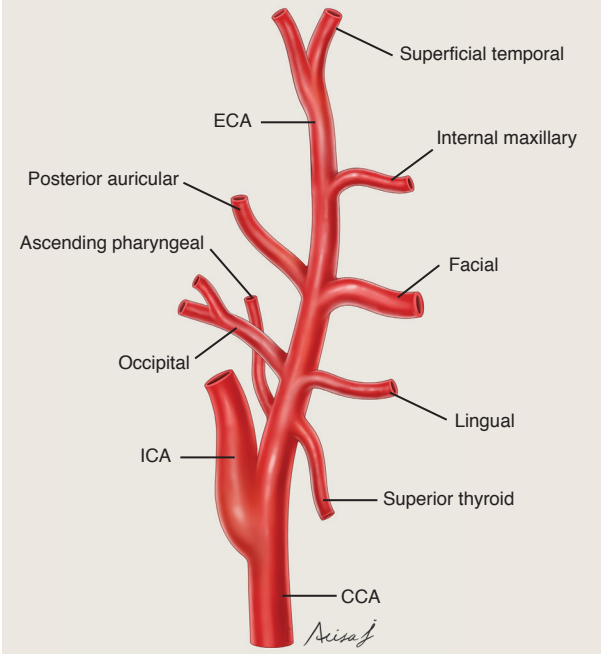


Figure 6-4. External carotid artery and its branches. CCA, common carotid artery; ECA, external carotid artery; ICA, internal carotid artery. (Adapted from Cho L, Mukherjee D. Basic cerebral anatomy for the carotid interventionalists: The intracranial and extracranial vessels. *Catheter Cardiovasc Interv.* 2006;68:104-111.)

The carotid sinus baroreceptors also reacts to changes in blood vessel stretching, as well as maintaining and detecting blood pressure (BP) changes.

It is necessary to recognize normal vascular physiology in order to understand potential cardiovascular reactions to carotid intervention. Stretching or compression of the carotid sinus may trigger a vasodepressor (hypotension without bradycardia), or vasovagal (hypotension and bradycardia) response with systemic hypotension. These reactions are mediated by stimulation of the carotid sinus nerve (a division of the glossopharyngeal nerve) in the carotid baroreceptor, together with vagus nerve activation, which leads to inhibiting sympathetic tone. The responsiveness of the carotid baroreceptors differs and can be affected by medication (e.g., beta-blockers and vasodilators may increase responsiveness), the occurrence of calcified plaque within the carotid bulb (increases responsiveness), or prior CEA (decreases sensitivity).

The ICA provides blood to the ipsilateral eye, cerebral hemispheres, and portions of the nose and forehead. In its proximal part, the ICA lies medial and caudal to the ECA. These connections might be recognized in the lateral and posteroanterior (PA) projections by angiography. Using the Bouthillier classification (8), the ICA is divided into seven parts (C1-C7) based on the angiographic presentation of the vessel, indicating each part providing branches into different vessels (Fig. 6-5).

- C1: Cervical
- C2: Petrous
  - Caraticotympanic artery
  - Vidian artery
- C3: Lacerum
- C4: Cavernous
  - Meningohypophyseal trunk
  - Inferolateral trunk
- C5: Clinoid
- C6: Ophthalmic
  - Ophthalmic artery
  - Superior hypophyseal trunk
- C7: Communicating
  - Posterior communicating artery
  - Anterior choroidal artery
  - Anterior cerebral artery (ACA)
  - Middle cerebral artery (MCA)

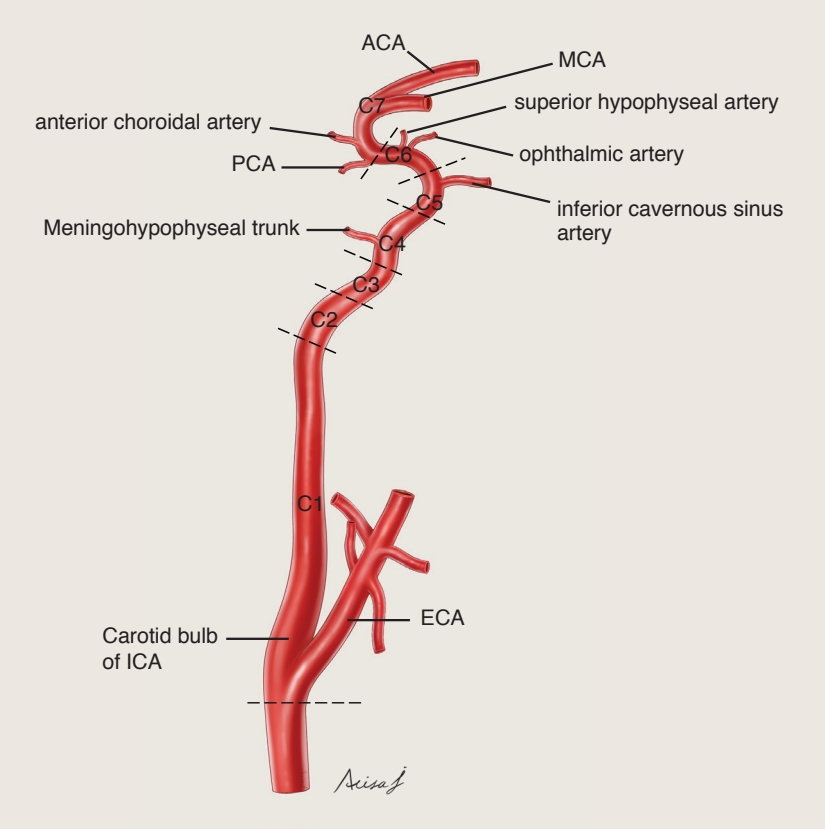


Figure 6-5. Internal carotid artery (ICA) and its branches. ACA, anterior cerebral artery; ECA, external carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery. (Adapted from Cho L, Mukherjee D. Basic cerebral anatomy for the carotid interventionalists: The intracranial and extracranial vessels. *Catheter Cardiovasc Interv*. 2006;68:104-111.)

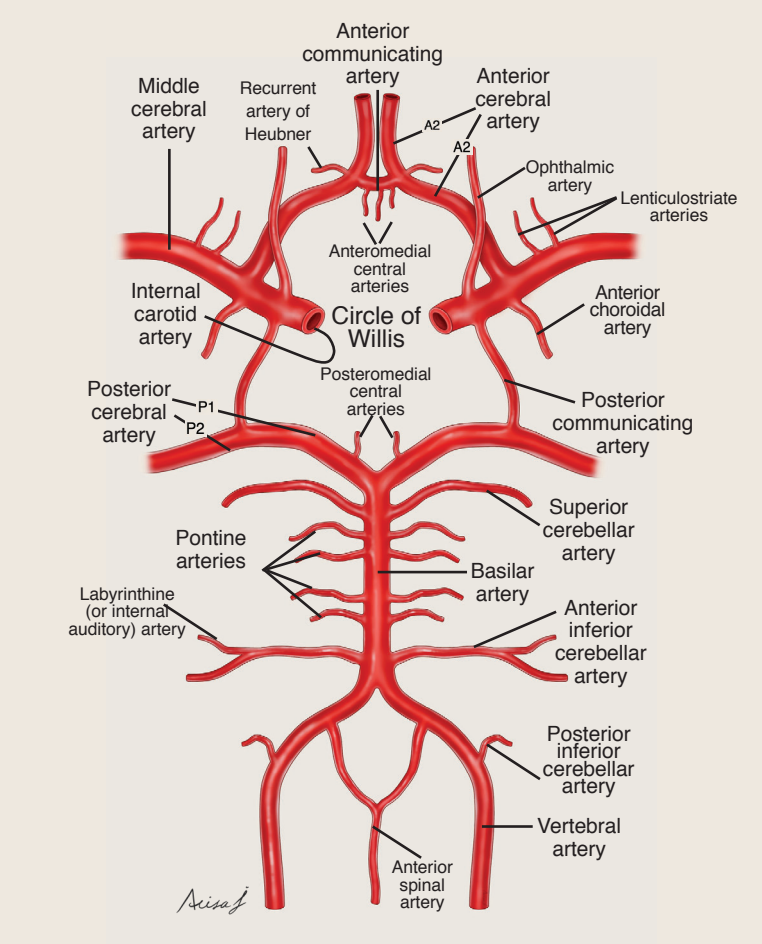


Figure 6-6. Anatomy of circle of Willis.

The branches of the ICA are normally small and unpredictable, and frequently may not exist. The ophthalmic artery is the most important and is the first intracranial division of the ICA. It appears soon after the ICA comes out of the cavernous sinus, traverses a short intracranial course, crosses the optic canal, and enters into the orbit. The ophthalmic artery provides blood supply to optic nerve and the ipsilateral retina, which is an essential route supplying collateral flow between the ICA and ECA (via maxillary branches).

Similarly, the posterior communicating artery, which arises from the ICA following the superior hypophyseal artery takes off, connects the posterior cerebral artery (PCA) and the branches of the basilar artery to complete the caudal portion of the 'circle of Willis' (Fig. 6-6), providing an important collateral link between the posterior and anterior cerebral circulations. At the circle of Willis, the distal ICA bifurcates into the anterior cerebral artery (ACA) and the middle cerebral artery (MCA).

The MCA is the largest of three major arteries and responsible for supplying the lateral (side) areas of the frontal, temporal, and parietal lobes. These areas control the sensory and motor cortices of the face and upper limb, as well as control speech from the Wernicke portion of the temporal lobe as well as Broca's area for the frontal lobe. The ACA is responsible for supplying the sensory and motor cortices for the lower limb.

COLLATERAL CIRCULATION

When one of the ICA is jeopardized by occlusive disease, then the cerebral collateral circulation performs an essential function to preserve cerebral perfusion (9). The main routes of collateral flow are extracranial anastomotic channels, the circle of Willis, and leptomeningeal communications that connect watershed areas between major arteries. However, the configuration of the circle of Willis widely differs, with a whole circle of Willis existing in fewer than 20-25% of individuals. The ECA may supply important intracranial flow if ICA stenosis is severe or occlusion via the ophthalmic

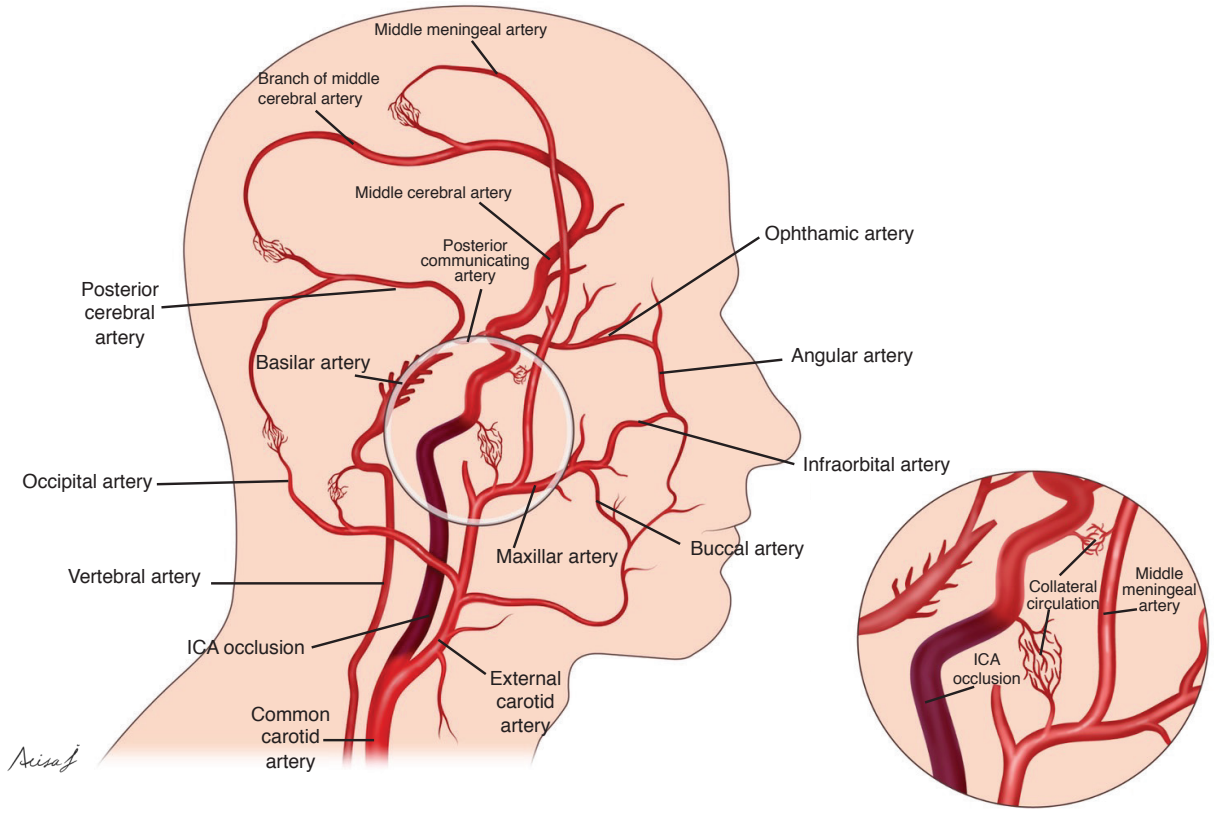


Figure 6-7. Collateral circulation between the internal carotid artery (ICA), external carotid artery, and vertebrobasilar artery supplies oxygenated blood to brain territories distal to an occluded ICA. (Redrawn from Erickson K, Cole D. Carotid artery disease: stenting vs endarterectomy. *British Journal of Anaesthesia*. 1010:105 (suppl 1): i34–i49, with permission from Elsevier)

artery (Fig. 6-7). The ophthalmic artery obtains blood flow from different collateral branches of the ECA, and the backward flow within the ophthalmic artery eventually fills the proximal subarachnoid part of the ICA. Additionally, the vertebral artery can interface with the carotid system by the ascending pharyngeal as well as the occipital ECA branches. Following vertebral artery blockage, the occipital artery can supply distal flow to the terminal vertebral artery segment via intramuscular communication.

Important intracranial and extracranial collateral pathways supply blood flow to brain territories which are distant to a stenosed ICA, including

- ICA to the contralateral ICA - via interhemispheric circulation via the anterior communicating artery
- Vertebrobasilar system to the ICA - via the trigeminal artery, posterior communicating artery, hypoglossal artery, otic artery, together with the proatlantal intersegmental artery (Fig. 6-8)
- ECA to the ICA - by the internal maxillary branch of the ECA to the ophthalmic branch of the ICA
- ECA to the vertebral artery - by the occipital branch of the ECA

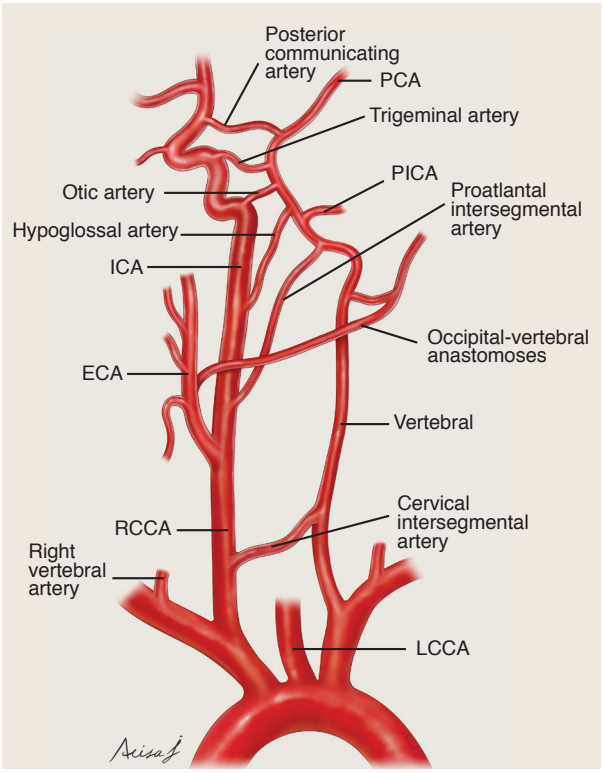


Figure 6-8. Carotid artery and vertebrobasilar collateral anatomoses. (Adapted from Cho L, Mukherjee D. Basic cerebral anatomy for the carotid interventionalists: The intracranial and extracranial vessels. *Catheter Cardiovasc Interv.* 2006;68:104-111.)

PATHOPHYSIOLOGY

The most common pathophysiology of carotid artery stenosis is the progressive narrowing of the carotid artery caused by development of atherosclerosis plaque, which is composed of lipid substances, calcium, and thrombus. These plaques have a tendency to develop at the points of carotid bifurcation and along vessel areas of curvature (10). Hemodynamic forces acting at carotid bifurcation locations have a function in restricting intimal thickening in these areas. Both in vitro and in vivo studies have reported that disrupted flow and low-shear conditions cause endothelial dysfunction and cause stenosis (11,12).

The two primary mechanisms of stroke in extracranial ICA disease are distal embolization and low-flow phenomenon due to insufficient collateral circulation distant from a hemodynamically significant occlusion or stenosis (13). A diffuse-weight imaging MRI study showed that an acute ischemic injury in ICA occlusive disease is mostly multiple (14). These results reinforce the fact that embolism from an atherosclerotic plaque rupture is the principal mechanism of ischemic stroke. In addition, thrombosis and low cerebral blood flow are other possible mechanisms which are caused by gradual plaque growth and luminal narrowing. Hemodynamic insufficiency can occur when any conditions that interfere with cerebral perfusions such as orthostatic, hypotension or volume depletion are added on to the carotid occlusion, particularly if the contralateral carotid disease has severe stenosis. This low flow mechanism can explain the infarction in the border zone areas or so called 'watershed infarction'.

EPIDEMIOLOGY

The prevalence of atherosclerotic carotid disease differs between studies, depending on the definition used, race, population risk factors, and investigative tool used for diagnosis (15,16). From a recent study using ultrasonography on people aged 30-79 years (17), there were substantial variations in prevalence between each region worldwide. The Western Pacific region experienced the biggest share of global cases, followed by the regions of South-East Asia, Europe, the Americas, the Eastern Mediterranean, and Africa. Current smoking habits, hypertension and diabetes were confirmed as general risk factors for both carotid plaque buildup and greater carotid intima-media thickness. The global prevalence of carotid disease, defined as greater carotid intima-media thickness by 1.0 mm or greater in people ages 30-79 years, was estimated to be 27.6%. However, the prevalence of carotid stenosis, defined as 50% or greater stenosis, was only 1.5%. These numbers are consistent with another study that revealed the incidence of carotid stenosis in the general population to be about 3% (18). This finding suggested that routine screening to detect carotid stenosis should not be recommended for general adults.

ETIOLOGY

The most common etiology of carotid artery stenosis is atherosclerosis. The other causes are fibromuscular dysplasia (FMD), vascular injury, and carotid dissection or radiation therapy. Patients that have vascular disease at other territories as well as risk factors such as hypertension, diabetes mellitus, hyperlipidemia and smoking have greater risk for developing carotid artery atherosclerosis. The lesion is normally unifocal and 90% are identified within 2 cm from the ICA origin.

FMD is a rare disease that mainly involves medium-sized arteries. The vessels involved are usually at the mid and distal ICA and renal arteries (19). This disease most commonly affects middle-aged females. The clinical manifestation varies from asymptomatic to TIAs or cerebral infarction, associated with stenosis, dissections, aneurysms, or complete occlusion.

Vascular injury can cause subsequent carotid stenosis. Torsion and transient occlusion have the effect of creating a temporary functional arterial stenosis. Sometimes this vascular injury results in an intimal tear and thrombus formation.

Dissection of the extracranial ICA can happen spontaneously or linked with trauma, such as penetrating neck injuries, intraoperative lacerations and percutaneous carotid angiography. The etiology of carotid artery dissection results from an intimal tear. That tear may cause complete or partial occlusion, traumatic pseudoaneurysm, cerebral emboli, or dissection. Cystic medial necrosis with deterioration of muscle fibers together with internal elastic laminae have also been identified.

Radiation therapy is normally used to treat malignant neck tumors and can affect extracranial carotid vessels. Such radiation-induced carotid stenosis has been linked

to cerebrovascular insufficiency. Three patterns of large vessel injury are described as; 1) intimal damage with mural thrombosis occurring within 5 years of therapy, 2) fibrotic occlusion presenting within 10 years, as well as 3) predisposition to development of atheroma together with periarterial fibrosis that has a latency of about 20 years (20). Radiation-induced atheromatous lesions are similar to usual atherosclerotic lesions. However, the lesions are clearly confined to irradiated vessel segments, sparing close nonirradiated segments, which present in unusual locations of atherosclerosis (Fig. 6-9). The specific process of occlusion and vascular stenosis after radiation therapy is unknown.

NATURAL HISTORY

To determine the best preventive therapy for carotid artery stenosis, the natural history of stroke in these patients must first be elucidated. It is well established that stroke risk is greater in symptomatic patients compared with asymptomatic patients. Previous studies have published an annual stroke risk of about 2% to 5% for patients suffering from severe asymptomatic carotid stenosis (ACAS) (3,21). On the opposite side, among symptomatic patients (i.e., patients who have had previous stroke or TIA), the risk of a subsequent stroke increases to 12% - 15% during the first year and 30% - 35% within 5 years (22). Besides patient symptoms, annual stroke risk is dependent on stenosis severity, rate of disease progression, plaque morphology, contralateral disease, silent cerebral infarction, collaterals' extent, and concurrent medical treatment. Therefore, the natural history of carotid disease is quite uncertain. Some patients have disease progression and develop a carotid total occlusion without any symptoms, while other patients may develop a disabling or fatal stroke (23). However, proper treatment of asymptomatic ICA stenosis, especially for moderate to severe (70% - 80%) disease, is still controversial. Although that patient population usually has a low rate of stroke or TIA, there is still a high stenosis progression rate, supporting the need to observe those patients closely.

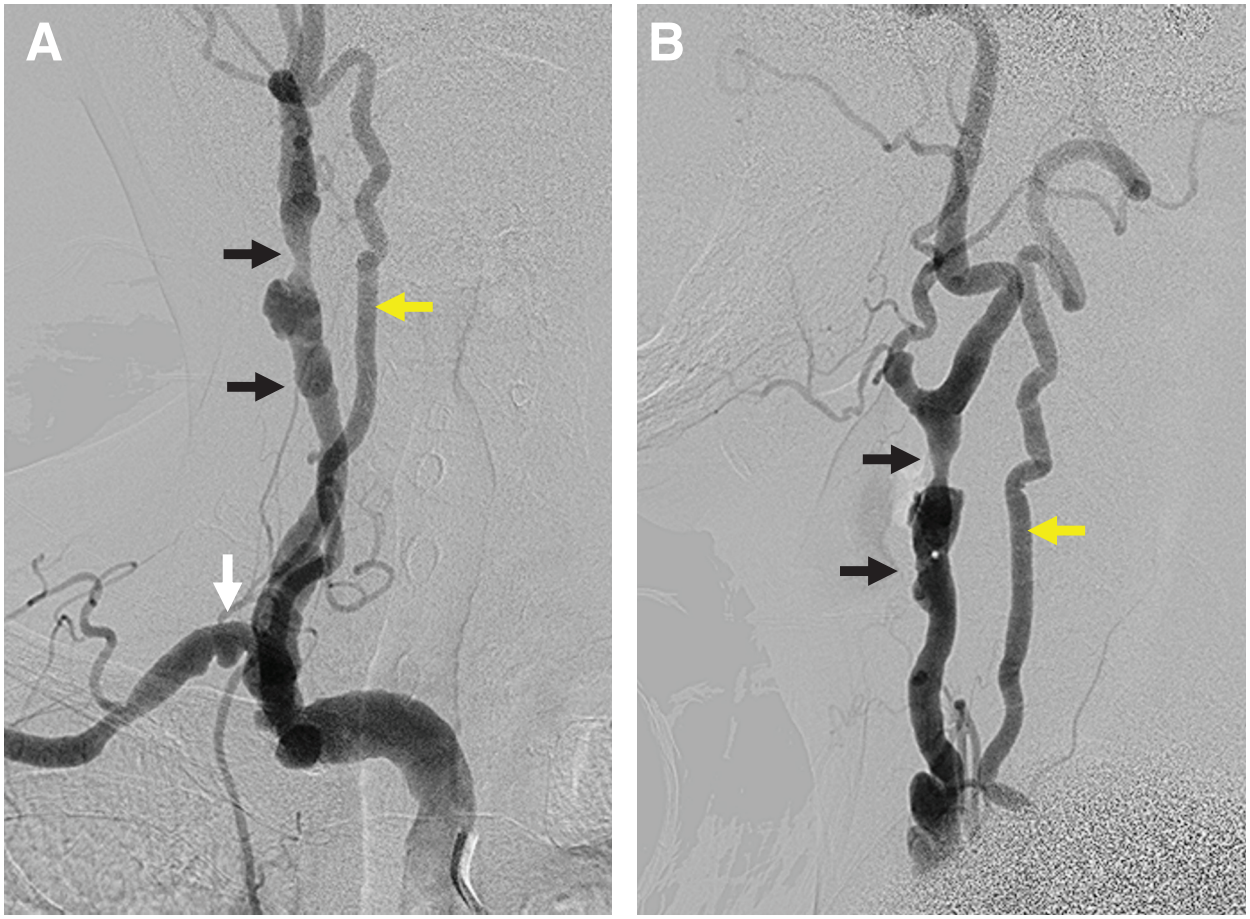


Figure 6-9. Radiation-induced carotid artery stenosis. Digital subtraction angiography in anteroposterior (A) and lateral (B) views of the right carotid artery showing long diffuse stenoses of the right common carotid artery (black arrows) and involving proximal right subclavian artery (white arrow) with no stenosis of right vertebral artery (yellow arrow).

CLINICAL PRESENTATION

A careful consideration of patient history is important in determining whether symptoms are attributable to carotid stenosis. Most patients that have carotid artery stenosis are asymptomatic and only diagnosed following auscultation of a carotid bruit or routine carotid ultrasound screening. In symptomatic patients, the clinical presentation is either TIA or ischemic stroke. TIA is defined as a brief neurological dysfunction episode arising from focal temporary cerebral ischemia, which is not linked with an acute cerebral infarction and resolves within 24 hours. If not treated, 11% of TIA patients develop a stroke within 90 days, with about one-half occurring within the first 2 days (24). Ischemic stroke is designated as a neurological dysfunction episode resulting from retinal or focal cerebral infarction that persists longer than 24 hours.

The typical symptoms of extracranial carotid stenosis are transient monocular blindness (amaurosis fugax), hemiparesis, hemiplegia, and speech disorders (aphasia). Patients that have both hemispheric and retinal symptoms often suffer severe extracranial carotid disease. The clinical manifestations associated with ICA branch stenosia or occlusions are summarized in Table 6-1.

Amaurosis fugax or transient monocular blindness is traditionally expressed as, 'a shade coming down over one's eye'. Hemispheric symptoms comprise sensory loss, unilateral motor weakness, language or speech disruptions, and visual field disruptions. It is important to discriminate between vertebrobasilar and hemispheric symptoms, because patients may have asymptomatic carotid stenosis and/or vertebrobasilar insufficiency. Signs and symptoms which are caused by infarction or ischemia in the vertebrobasilar system include cranial nerve deficits (dysarthria, diplopia, dysphagia), ataxia, imbalance, dizziness, visual field loss, and incoordination, which are atypical for the circulation of the carotid artery. The accurate designation of symptoms will greatly help with clinical management and appropriate revascularization timing.

Table 6-1. Clinical manifestations associated with ICA branch stenosia or occlusion (37).

Occluded artery	Clinical manifestation
Ophthalmic artery	• Transient monocular blindness (amaurosis fugax)
Anterior choroidal artery	• Contralateral dense hemiparesis: face, arm, leg • Contralateral hemisensory loss (if lateral geniculate is involved, a contralateral hemianopsia)
Recurrent artery of heubner	• Mild weakness in the contralateral limb with dysarthria • Abulia with apathy and inertia of movement
Anterior cerebral artery	• Contralateral weakness of the legs and shoulder • Cortical sensory deficit with poor touch localization and extinction with bilateral stimuli (left arm apraxia only)
Middle cerebral artery (MCA) - M1 Segment - M2 Segment	 • Contralateral spastic hemiplegia, visual deficit • Hemiparesis affecting the face and arm more than the legs • Visual deficits
Left hemisphere MCA (superior branch)	• Motor aphasia (Broca's aphasia) • Apraxia-both upper extremities • Oral buccal apraxia
Left hemisphere MCA (inferior branch)	• Receptive aphasia (Wernicke's)
Nondominant hemisphere MCA (superior branch)	• Neglect—left side of space • Apraxia in left upper extremity only
Nondominant hemisphere MCA (inferior branch)	• Constructional apraxia and difficulty with shape • Confusion and delirium

MCA, middle cerebral artery; ICA, internal carotid artery

To confirm the diagnosis, a complete physical examination and neurological assessment is important, including the examination of heart rhythm, auscultation for carotid bruits and heart murmur (to rule out cardiac emboli), fundoscopic examination (for detecting retinal embolization), together with a focused neurologic examination (to associate with an ischemic territory). The National Institute of Health Stroke Scale (NIHSS) may be applied for quantification of the neurological deficit and speculate the outcome after ischemic stroke (25,26). Again, clinical findings have to be associated with vascular and brain imaging in order to decide if a carotid stenosis is symptomatic.

Carotid artery stenosis is defined as 'asymptomatic' if no previous symptoms can be determined or if symptoms happened >6 months ago, and 'symptomatic' if linked with symptoms in the previous 6 months.

IMAGING STUDIES

In patients with stroke or TIA, an urgent imaging study is crucial to evaluate the structural pathology and anatomy for the brain and carotid artery, and to guide treatment. A duplex ultrasound study (DUS), computed tomographic angiography (CTA) and/or magnetic resonance angiography (MRA) are recommended methods for assessing the severity of extracranial carotid stenoses (27).

A DUS is normally the first-choice imaging modality for detecting, grading, and monitoring of extracranial ICA stenoses. The key variables to determine the severity of carotid stenosis are the ICA peak systolic velocity (PSV), ICA end diastolic velocity (EDV), and the ratio of PSV of the ICA to that of the CCA. As the stenosis at the bifurcation becomes more severe, the velocity of the ICA velocity increases, which in turn, leads to a higher ICA/CCA PSV ratio (Fig 6-10). To date, there is a wide range and no internationally accepted standard for the gradation of carotid stenosis. The Society of Radiologists in Ultrasound (SRU) consensus criteria are still widely used and recommend PSV cutoff values

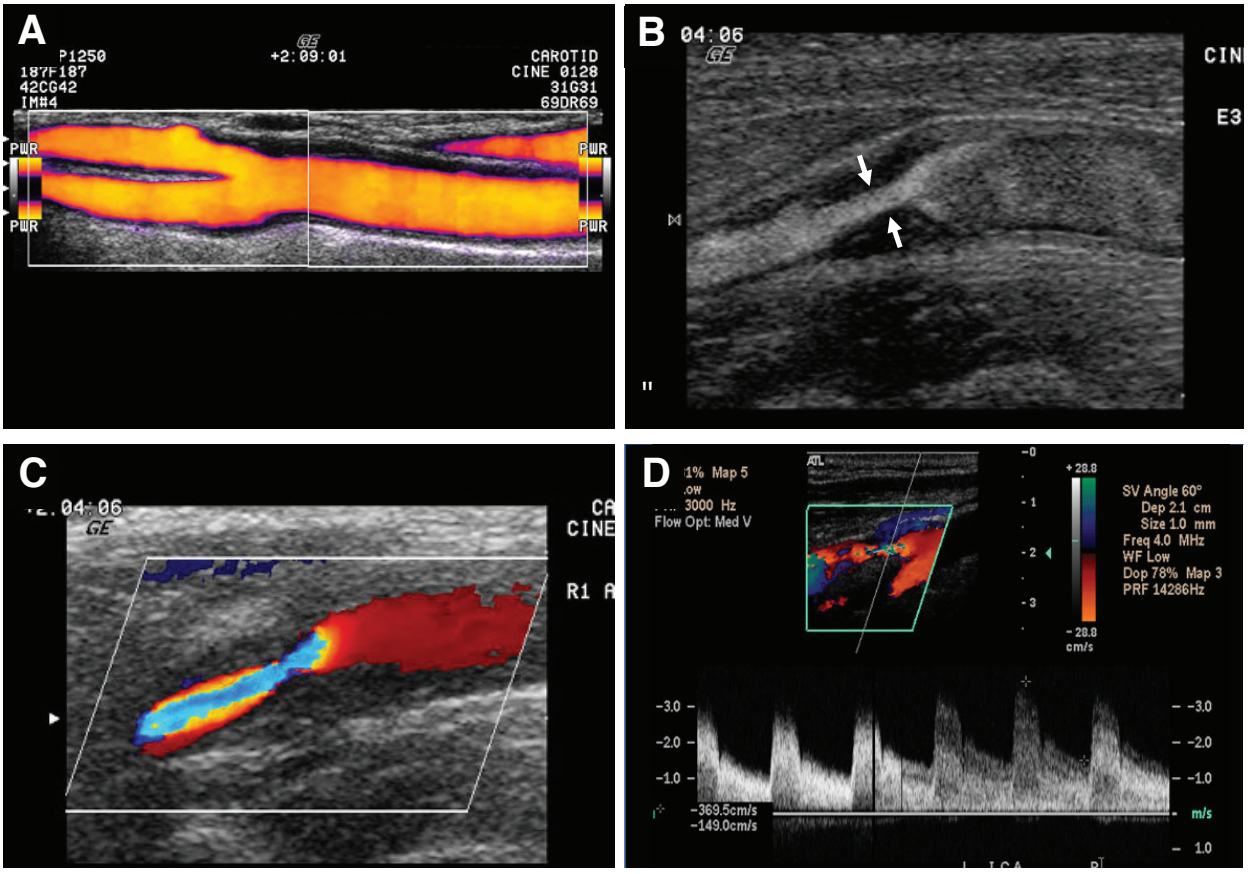


Figure 6-10. Carotid duplex ultrasonography. A: Longitudinal image of the normal carotid artery and bifurcation into the internal and external carotid arteries. B: B-mode imaging of the internal carotid artery showing the plaque morphology of the vessel wall as well as the area of narrowing (arrows). C: Color Doppler in the same patient demonstrating the area of narrowing with increased (aliased) flow denoted by the blue/yellow pattern. D. B-mode imaging reveals severe turbulence flow in the narrowing area. Doppler waveforms demonstrating increase in peak systolic flow velocity (369 cm/sec) and end-diastolic velocity (149 cm/sec).