

FLAIR Fluid-Attenuated Inversion Recovery

Used to suppress the signal from water (CSF).

Suppression of fluid signals allows enhanced visualization of lesions.

Diffusion-Weighted Imaging (DWI)

Following an ischemic stroke, there is increased signal on a DWI scan. Water diffusion is restricted as a result of cytotoxic edema (cellular swelling).

The DWI enhancement appears within 5-10 minutes of the onset of stroke symptoms.

MR ANGIOGRAPHY- MRA

MRA capitalizes on creating intensity differences between flowing tissue and stationary tissue. By suppressing background stationary tissue and focusing only on the high-signal flowing blood, one can obtain a data set that depicts only vascular structures. If employed with contiguous sections or three-dimensional volumetric acquisitions, one can produce very thin section MR angiograms that can be rotated in space to visualize the circle of Willis or the carotid bifurcation. MR is capable of detecting atherosclerotic narrowings or intracranial aneurysms in three-dimensional space.

CONCUSSION

A concussion is a traumatic brain injury that alters the way your brain functions. Effects are usually temporary, but can include problems with headache, concentration, memory, judgment, balance and coordination.

Although concussions usually are caused by a blow to the head, they can also occur when the head and upper body are violently shaken. These injuries can cause a loss of consciousness, but most concussions do not. Because of this, some people have concussions and don't realize it.

Concussions are common, particularly if you play a contact sport, such as football. But every concussion injures your brain to some extent. This injury needs time and rest to heal properly. Luckily, most concussive traumatic brain injuries are mild, and people usually recover fully.

Symptoms

The signs and symptoms of a concussion can be subtle and may not be immediately apparent. Symptoms can last for days, weeks or even longer.

The most common symptoms after a concussive traumatic brain injury are headache, amnesia and confusion. The amnesia, which may or may not be preceded by a loss of consciousness, almost always involves the loss of memory of the impact that caused the concussion.

Signs and symptoms of a concussion may include:

Headache or a feeling of pressure in the head

- Temporary loss of consciousness
- Confusion or feeling as if in a fog
- Amnesia surrounding the traumatic event
- Dizziness or "seeing stars"
- Ringing in the ears
- Nausea or vomiting
- Slurred speech
- Fatigue

Some symptoms of concussions may be immediate or delayed in onset by hours or days after injury:

- Concentration and memory complaints
- Irritability and other personality changes
- Sensitivity to light and noise
- Sleep disturbances
- Psychological adjustment problems and depression
- Disorders of taste and smell

Symptoms in children

Head trauma is very common in young children. But concussions can be difficult to recognize in infants and toddlers because they can't readily communicate how they feel. Nonverbal clues of a concussion may include:

- Listlessness, tiring easily
- Irritability, crankiness
- Change in eating or sleeping patterns
- Lack of interest in favorite toys
- Loss of balance, unsteady walking

Seek emergency care for a child who experiences a head injury and:

- Vomiting
- A headache that gets worse over time
- Changes in his or her behavior, including irritability or fussiness
- Changes in physical coordination, including stumbling or clumsiness
- Confusion or disorientation
- Slurred speech or other changes in speech
- Vision or eye disturbances, including pupils that are bigger than normal (dilated pupils) or pupils of unequal sizes
- Changes in breathing pattern
- Lasting or recurrent dizziness
- Blood or fluid discharge from the nose or ears
- Large head bumps or bruises on areas other than the forehead, especially in infants under 12 months of age

Seek emergency care for anyone who experiences a head injury and:

- A loss of consciousness lasting more than a minute
- Repeated vomiting
- Seizures
- Obvious difficulty with mental function or physical coordination
- Symptoms that worsen over time

Athletes

No one should return to play or vigorous activity while signs or symptoms of a concussion are present. Experts recommend that an athlete with a suspected concussion not return to play until he or she has been medically evaluated. Experts also recommend that child and adolescent athletes with a concussion not return to play on the same day as the injury.

Causes

Your brain has the consistency of gelatin. It's cushioned from everyday jolts and bumps by the cerebrospinal fluid that it floats in, inside your skull. A violent blow to your head and neck or upper body can cause your brain to slide back and forth forcefully against the inner wall of your skull. Sudden acceleration or deceleration of the head - resulting from events such as a car crash or being violently shaken (shaken baby syndrome) - also can cause brain injury.

These injuries affect brain function, usually for a brief period, resulting in signs and symptoms of concussion. A brain injury of this sort may even lead to bleeding in or around your brain causing symptoms, such as prolonged drowsiness and confusion, that may develop right away or even later. Such bleeding in your brain can be fatal. That's why anyone who experiences a brain injury needs to be monitored in the hours afterward and receive emergency care if symptoms worsen.

Risk factors

Factors that may increase your risk of a concussion include:

- Participating in a high risk sport, such as football, hockey, soccer or other contact sport; the risk is further increased if there's a lack of proper safety equipment and supervision
- Being involved in a motor vehicle collision
- Being a soldier involved in combat
- Being a victim of physical abuse
- Falling, especially in young children and older adults
- Having had a previous concussion

Complications

Potential complications of concussion include:

- **Epilepsy.** People who have had a concussion double their risk of developing epilepsy within the first five years after the injury.

- **Cumulative effects of multiple brain injuries.** Evidence exists indicating that people who have had multiple concussive brain injuries over the course of their lives may acquire lasting, and even progressive, cognitive impairment that limits functional ability.

Second impact syndrome. Sometimes, experiencing a second concussion before signs and symptoms of a first concussion have resolved may result in rapid and typically fatal brain swelling. After a concussion, the levels of brain chemicals are altered. It usually takes about a week for these levels to stabilize again. However, the time it takes to recover from a concussion is variable, and it is important for athletes never to return to sports while they're still experiencing signs and symptoms of concussion.

Tumors

Symptoms: Cerebral neoplasms produce subacute and progressive neurologic signs and symptoms. The patient's symptoms are determined by the size, location and rate of growth of the tumor, as well as the degree of peritumor cerebral edema.

1. Headache from increased intracranial pressure, at times with nausea and vomiting.
2. Focal clinical manifestations (hemiparesis, ataxia, aphasia, visual loss) depend on location and extent of surrounding brain edema. Tumors in relatively silent regions of the brain commonly present with changes in personality and behavior.
3. Seizures occur in about one-third of patients with tumors in the supratentorial compartment. Seizures are more likely to accompany slower-growing tumors than highly malignant ones.
4. Hemorrhage into a highly vascular tumor can produce a sudden change in neurological status that can be mistaken for a stroke.

Tumor-Related Brain Edema: Most brain tumors (primary or metastatic) cause edema in the surrounding brain parenchyma. If the edema is considerable or widespread, it can produce a marked increase in intracranial pressure, causing neurologic defects by compressing nearby structures.

Glioblastoma Multiforme (Spongioblastoma Multiforme)

Peak incidence between ages 50 and 60; twice as common in males; most common glioma. Unencapsulated, highly malignant; grows rapidly and infiltrates the brain extensively; may become enormous before diagnosed. Occurs most often in cerebral hemispheres, especially frontal and temporal lobes (rarely in brain stem and cerebellum). Occupies more than one lobe of affected hemisphere; may spread to opposite hemisphere by corpus callosum; may metastasize into cerebrospinal fluid (CSF), producing tumors in distant parts of the central nervous system.

Increased intracranial pressure (ICP), causing nausea, vomiting, headache, papilledema, mental and behavioral changes, altered vital signs (increased systolic pressure, widened pulse pressure, respiratory changes), speech and sensory disturbances. In children, irritability, projectile vomiting.

Astrocytoma

Second most common malignant glioma (30% of all gliomas). Occurs at any age, incidence higher in males. Occurs most often in white matter of cerebral hemispheres; may originate in any part of the central nervous system. Cerebellar astrocytomas usually confined to one hemisphere. Headache, mental activity changes, decreased motor strength and coordination, seizures, scanning speech, altered vital signs.

Oligodendroglioma

Third most common glioma (less than 5%). Occurs in middle adult years, more common in women. Slow growing. Mental and behavioral changes. Decreased visual acuity and other visual disturbances. Increased ICP.

Medulloblastoma

Rare glioma. Incidence highest in children ages 4 to 6. affects males more than females. Frequently metastasized via CSF. Increased ICP.

Ependymoma

Rare glioma. Most common in children and young adults. Locates most often in fourth and lateral ventricles. Similar to oligodendroglioma. Increased ICP and obstructive hydrocephalus, depending on tumor size.

Meningioma

Most common nongliomatous brain tumor, constituting 20% of primary brain tumors. Peak incidence among 50 year olds; rare in children; more common in females than males (ratio 3:2). Arises from the meninges. Common locations include parasagittal area, sphenoidal ridge, anterior part of the base of the skull, cerebellopontine angle, spinal canal. Benign, well-circumscribed, highly vascular tumors that compress underlying brain tissue.

Headache, seizures (two-thirds of patients), vomiting, changes in mental activity, similar to schwannomas.

Schwannoma (Acoustic Neurinoma, Neurilemoma, Cerebellopontine Angle Tumor)

Accounts for approximately 10% of all intracranial tumors. Higher incidence in women. Onset of symptoms between ages 30 and 60. Affects the craniospinal nerve sheath, usually cranial nerve VIII; also, V and VII and, to a lesser extent, VI and X on the same side as the tumor. Benign, but often classified as malignant because of its growth patterns; slow-growing, may be present for years before symptoms occur.

Unilateral hearing loss with or without tinnitus. Stiff neck and suboccipital discomfort. Secondary hydrocephalus. Ataxia and uncoordinated movement of one or both arms due to pressure on brain stem and cerebellum.

Neurofibromatosis Type I (von Recklinghausen Disease)

One of the most common inherited CNS disorders, most common autosomal dominant disorder, most common inherited tumor syndrome and occurs in 1:2,500 live births. Cutaneous lesions: Café-au-lait spots appear early childhood; axillary inguinal freckling, and subcutaneous or cutaneous NF.

Plexiform neurofibroma, optic nerve gliomas, scoliosis (30%), scalloped vertebrae (dural ectasia/lateral meningoceles) > underlying NFs.

Neurofibromatosis Type II

MISME: Multiple intracranial schwannomas, meningiomas, and ependymomas.

Bilateral vestibular schwannomas, multiple extra-axial tumors, schwannomas on cranial nerves and spinal nerve roots, and meningiomas on dural surfaces.

Hereditary syndrome. 50% known family history of NF2; 50% new mutations. 1 in 40,000-100,000. Most common kinds of symptoms: hearing loss, vertigo, multiple cranial neuropathies.

Pituitary Adenomas

Microadenomas are less than 10mm in diameter. Will be asymptomatic unless they are hypersecreting tumors.

Functional classification: 1. Non-functioning adenomas 2. Hypersecreting adenomas (prolactin, adrenocorticotrophic hormone (ACTH) or growth hormone)

Hyperprolactinemia: Prolactin levels greater than 100ng/mL almost always indicate a tumor. Hyperprolactinemia in women usually causes amenorrhea or galactorrhea. In men, the earliest symptoms are impotence and loss of libido.

Macroadenomas: Larger tumors compress the adjacent normal pituitary gland and produce hypopituitarism. Extension of the tumor above the sella turcica compress the optic chiasm. This causes progressive visual loss. Most macroadenomas come to attention because of visual loss or due to headache.

Cerebral Metastases

Metastasis to the brain parenchyma or meninges is a common complication of systemic cancer. Approximately 15-20% of patients dying of cancer will have brain metastasis at the time of autopsy. Bronchogenic carcinoma, breast carcinoma, renal cancer and malignant melanoma are the cancers most likely to metastasize to the brain.

The formation of a solid mass lesion is the most common type of cerebral metastasis. Metastases reach the brain by hematogenous spread. The frequency of metastasis in these structures is roughly in proportion to the blood flow to the region (cerebral hemispheres>cerebellum>brainstem). These lesions cause symptoms through increased intracranial pressure, destruction of brain tracts, cerebral edema, or seizures (similar to primary brain tumors). Headaches, intellectual or behavioral changes, focal weakness and unsteadiness are the most common presenting signs.

In about 40% of case of brain metastasis, a solitary lesion will be seen; in the remaining 60%, multiple tumors are seen. MRI with contrast is more sensitive for demonstrating small tumors and may show multiple lesions when only one is seen on contrast-enhanced CT.

White Matter Diseases in Adults

Multiple sclerosis (MS) and variants

Acute disseminated encephalomyelitis

Hurst hemorrhagic leukoencephalitis

Vascular disorders

Ischemic arteriolar disease

Boundary zone ischemia (unilateral carotid disease)

Arteritis (systemic lupus erythematosus , sarcoidosis)

Infectious/immune disorders

Acquired immune deficiency syndrome related disorders

Progressive multifocal leukoencephalopathy

Lyme disease

Vasogenic edema

Traumatic shear injury

Radiation injury/necrosis

Metabolic disorders

Central pontine myelinolysis

Marchiafava-Bignami disease

Adult leukodystrophies

Multiple sclerosis is the most common white matter disease and is related to focal areas of demyelination with reactive gliosis in the white matter of the brain, spinal cord, and the optic nerves. Clinical Presentation: Exacerbations and remissions of multifocal neurologic deficits. Impaired or double vision. Fatigue, weakness, numbness, tingling, and gait disturbances. Loss of sphincter control, blindness, paralysis and dementia. Onset 20-40. More common in persons of Western European lineage who live in temperate zones.

Individuals who migrate in early childhood from a low-risk to a high-risk area have the same risk of developing MS as those in the area they move to. If the same move is made after adolescence, the risk remains low.

- Familial incidence.
- HLA antigens: HLA-A3, HLA-B7, HLA-DR2.
- Cerebrospinal Fluid: Mild lymphocytosis, slightly elevated protein, and oligoclonal immunoglobulin bands of IgG on immunoelectrophoresis.
- Visual, auditory and somatosensory evoked responses.
- Lhermitte's Sign: Electric-like shocks spreading down the body on forceful flexion of the head and neck.

Intracranial Aneurysm

- Saccular aneurysms (berry)
- Tend to occur at arterial bifurcations
- Multiple aneurysms in 20% of adults
- Associated with polycystic kidney disease and coarctation of aorta

Symptoms

- Focal neurological deficit by compression of adjacent structures
- Most are asymptomatic or produce nonspecific symptoms until they rupture
- Subarachnoid hemorrhage

Saccular (berry) Aneurysm

- Most common type
- Secondary to congenital weakness of media
- Usually occurs at major vessel bifurcations
- Occurs at the circle of Willis
- Has a neck or stem
- Has a sac that may be partly filled with a blood clot

Fusiform (spindle-shaped) Aneurysm

- Occurs with atherosclerotic disease
- Characterized by irregular vessel dilation
- Develops on internal carotid or basilar arteries
- Rarely ruptures
- Produces brain and cranial nerve compression or CSF obstruction

Mycotic Aneurysm

- Rare
- Associated with septic emboli that occur secondary to bacterial endocarditis
- Develops when emboli lodge in the arterial lumen, causing arteritis; the arterial wall weakens and dilates

Dissecting Aneurysm

- Caused by arteriosclerosis, head injury, syphilis, or trauma during angiography
- Develops when blood is forced between layers of arterial walls, stripping intima from the underlying muscle layer

Traumatic Aneurysm

- Develops in the carotid system
- Associated with fractures and intimal damage
- May thrombose spontaneously

Giant Aneurysm

- Similar to saccular, but larger - 3cm or more in diameter
- Behaves like a space-occupying lesion, producing cerebral tissue compression and cranial nerve damage
- Associated with hypertension

Subarachnoid Hemorrhage

- Usually from rupture of an aneurysm or AVM
- No specific cause in 20%

Symptoms

- Sudden headache of a severity never experienced previously by patient
- Nausea and vomiting
- Loss or impairment of consciousness

Cerebrovascular Accident

Factors that increase the risk of CVA include history of transient ischemic attacks, atherosclerosis, hypertension, electrocardiogram changes, arrhythmias, rheumatic heart disease, diabetes mellitus, gout, postural hypotension, cardiac or myocardial enlargement, high serum triglyceride levels, lack of exercise, use of oral contraceptives, cigarette smoking, and family history of CVA. The major causes of CVA are thrombosis, embolism, and hemorrhage.

Thrombosis

In middle-aged and elderly, people, among whom there is a higher incidence of atherosclerosis, diabetes, and hypertension, thrombosis is the most common cause of CVA. Obstruction of a blood vessel causes the CVA. Typically, the main site of the obstruction is in extracerebral vessels, but sometimes it's intracerebral.

Thrombosis causes ischemia in brain tissue supplied by the affected vessel as well as congestion and edema. The latter may produce more clinical effects than thrombosis itself, but these symptoms subside with the edema.

Thrombosis may develop while the patient sleeps or shortly after he awakens; it can also occur during surgery or after a myocardial infarction. The risk increases with obesity, smoking, or the use of oral contraceptives. Cocaine-induced ischemic stroke is now being seen in younger patients.

Embolism

The second most common cause of CVA, embolism is an occlusion of a blood vessel caused by a fragmented cloth, a tumor, fat, bacteria, or air. It can occur at any age, especially among patients with a history of rheumatic heart disease, endocarditis, post-traumatic valvular disease, myocardial fibrillation and other cardiac arrhythmias, or following open-heart surgery.

The embolus usually develops rapidly . in 10 to 20 seconds . and without warning. When it reaches the cerebral vasculature, it cuts off circulation by lodging in a narrow portion of an artery, most often the middle cerebral artery, causing necrosis and edema.

Hemorrhage

The third most common cause of CVA is hemorrhage. Like embolism, it may occur suddenly, at any age. Such hemorrhage results from chronic hypertension or aneurysms, which cause sudden rupture of a cerebral artery. The rupture diminishes blood supply to the area served by this artery. In addition, blood accumulates deep within the brain, further compressing neural tissue and causing even greater damage.

Carotid Injury

The CCA bifurcates into the ICA and external carotid artery at approximately the level of the fourth cervical vertebral body near the superior border of the thyroid cartilage. Although the external carotid artery is initially anteromedial to the ICA, and ICA quickly courses medially at approximately the C1 or C2 level before entering the skull base. Unlike the lower cervical vertebrae, the lateral articular processes and pedicles of the first through third cervical vertebrae project more anteriorly, with the distal portion of each cervical ICA lying in close proximity just anterior to these.

The ICA enters the carotid canal as the petrous segment, where it is firmly fixed within the petrous bone. With extension of the neck, the carotid canal is elevated stretching and partially fixing the cervical ICA against the lateral masses of the upper cervical spine. Rotation, which largely occurs at the atlantoaxial joint, forces the contralateral lateral mass of C1 anteriorly, further stretching the ICA. Because of this relationship, the cervical ICA is the typical site of injury associated with hyperextension-rotation of the head and neck.

Type I injuries result from a direct blow to the neck. This is the characteristic mechanism in elderly persons with advanced atherosclerotic disease. Impingement of the ICA between the mandible and the cervical spine with acute hyperflexion would injure the vessel in a similar fashion and may account for some of the injuries seen in victims of motor vehicle accidents.

Type II injuries are due to hyperextension and contralateral rotation of the head and neck and are the characteristic injuries seen in victims of motor vehicle accidents.

Damage occurs as the ICA is stretched over the lateral masses of the first and second cervical vertebrae. This mechanism accounts for over 90% of blunt injuries to the ICA and tends to affect young patients more frequently, perhaps due to the protection afforded the elderly by their tortuous vessels and less mobile cervical spine.

Type III injuries result from intraoral trauma and are typically seen in a child who has fallen with a hard object, such as a pencil, in their mouth. Type IV injuries result from associated basilar skull fractures.

Carotid Artery Dissection

The disorder occurs in hypertensive individuals who have no evidence of atherosclerotic vessel disease. Other risk factors include smoking and fibromuscular dysplasia. Dissection may occur with hyperextension and lateral flexion of the neck as the artery is stretched over the transverse processes of the upper cervical vertebrae. Focal unilateral headache is the most common symptom in association with dissection. The headache is steady, non-throbbing, of variable intensity, and is located in the frontal, auricular, or periorbital area. Neurologic manifestations may include stroke (resulting in contralateral hemiparesis, paresthesias, aphasia, ipsilateral blindness, or abducens paralysis) or oculosympathetic palsy with ptosis and miosis without anhidrosis. Focal neurologic deficits may follow the onset of headache or neck pain within minutes of hours. Bruits may be heard over the carotid.

Vertebral Artery Dissection

Vertebral artery dissection occurs most commonly in middle-aged women. People with hypertension or fibromuscular dysplasia are at greater risk of dissection. Pain in the occiput or posterior neck is the presenting symptom in 80% of patients, preceding ischemic symptoms by minutes to 30 days. Most patients present with a completed stroke, with a minority presenting with transient ischemic attacks. The lateral medullary syndrome (pain, numbness, ipsilateral face [trigeminal], ataxia, vertigo, nystagmus, Horner's syndrome [descending sympathetic tract], dysphagia, numbness of ipsilateral appendages) is the most common neurologic manifestation. Severe cases may have basilar artery involvement with associated quadriparesis, dysphagia, diplopia, with preserved sensation.

Symptoms of TIA

Carotid Territory . paresthesia/weakness of hand, arm, and face; aphasia (dominant hemisphere); dysarthria; unilateral neglect.

Lacunar . hemibody sensory loss of paresthesia; pure motor hemiparesis.

Vertebrobasilar . dysarthria; vertigo, ataxia; diplopia; visual field loss; perioral paresthesias; acute confusional state; profound general weakness.

Transient Ischemic Attack

TIA is a sudden or rapid onset of neurological deficit caused by cerebral ischemia. It may last for a few minutes or up to 24 hours and clears without residual signs. Risk factors: hypertension, smoking, obesity, hyperlipidemias, advanced age.

Etiology

1. Cardiac emboli: atrial fibrillation, mitral valve disease, prosthetic heart valves, bacterial and marantic endocarditis, intracardiac defects with paradoxical embolism (patent foramen ovale, atrial septal defect)
2. Carotid or vertebral artery disease: arteriosclerosis, fibromuscular hyperplasia, traumatic and spontaneous carotid and vertebrobasilar artery dissection

Characteristics of Carotid Artery Syndrome

- Ipsilateral monocular vision loss (amaurosis fugax); the patient often feels as if a shade has come down over one eye
- Episodic contralateral arm, leg, and face paresis and paresthesias
- Slurred speech, transient aphasia
- Ipsilateral headache of vascular type
- Carotid bruit may be present over the carotid bifurcation
- Microemboli, hemorrhages, and exudates may be noted in the ipsilateral retina

Characteristics of Vertebrobasilar Artery Syndrome

- Binocular visual disturbances (blurred vision, diplopia, total blindness)
- Vertigo, nausea, vomiting, tinnitus
- Sudden loss of postural tone of all four extremities (drop attacks) with no loss of consciousness
- Slurred speech, ataxia, numbness around lips or face

Vertebrobasilar Injuries

Mechanisms of vertebral artery injury: cervical hyperextension, excessive contralateral rotation, and hyperextension and rotation.

Familial history of stroke or cardiovascular disease, hypertension, smoking, cervical spondylosis/arthrosis, bleeding disorders, medication, and/or anatomical anomaly/pathology.

Rotation of C1 on C2 between 30 and 45° causes the vertebral artery at the atlantoaxial junction to become compressed on the opposite side of head rotation, subsequently reducing blood flow to the basilar artery. In the normal patient, this diminution of blood flow caused by positional change of the cervical spine will not cause any neurological symptoms, such as dizziness, nausea, tinnitus, faintness, or nystagmus. This lack of symptoms is a result of the normal flow of collateral circulation by the opposite vertebral artery, common carotid arteries, and a communicating cerebral arterial circle (Circle of Willis).

Seven areas of possible compression: 1) between C1-2 transverse processes, where the vertebral arteries are relatively fixed at the C1 and C2 transverse foramina; 2) C2-3 at the level of the superior articular facet of C3 on the ipsilateral side to head rotation; 3) the C1 transverse process and the internal carotid artery; 4) the atlanto-occipital aperture by the posterior arch of atlas and the rim of foramen magnum, or anteriorly by folding of the atlanto-occipital joint capsule and posteriorly by the atlanto-occipital membrane; 5) C4-5 or C5-6 levels because of arthrosis of the joints of von Luschka with

compression on the ipsilateral side to head rotation; 6) at the transverse foramina of the atlas or axis between the obliquus capitis inferior and intertransversarii during rotatory movements; 7) before entering the C6 transverse process by the longus colli muscle or by tissue communicating between the longus colli and scalenus anterior muscles.

Vertebral artery pathological alterations: intimal disruption, subintimal hematoma, dissection, pseudoaneurysm, and thromboembolism.

Wallenberg's Syndrome: ipsilateral loss of cranial nerves V, IX, X, and XI cerebellar ataxia, Horner's syndrome, and contralateral loss of pain and temperature sensation. Sudden death, quadriplegia, and the locked-in syndrome (quadriplegia with loss of all lower cranial nerves).

Risk factors: age, hypertension, hyperlipidemia, family history of stroke or heart attacks, diabetes, smoking, heart and peripheral vascular disease, young adult females on birth control pills, cervical spondylosis, and cervical spine injury (hyperextension injury).

Clinical evaluation: blood pressure (both arms), palpate radial pulses (normal, feeble or absent), palpate carotid pulses, auscultate carotid arteries (bruit, hissing or squirting sound), and auscultate subclavian arteries. If pulsations or bruits are present at the carotid or subclavian arteries, do not perform the functional maneuver. A difference of 10 mm Hg between the two systolic blood pressures and a feeble or absent radial pulse is suggestive of subclavian artery stenosis.

Atherosclerosis

Pathologic degenerative process resulting from deposition of plasma lipids in arterial walls. Location: Internal carotid and basilar arteries most common sites in head and neck. Plaque surface irregularity associated with increased risk of stroke at all degrees of stenosis. Endarterectomy if symptomatic carotid stenosis is \geq 70%.