cranial nerves, possibly causing unilateral facial paralysis or visual problems. Unlike other forms of cancer, brain tumors do not cause the usual systemic signs of malignancy because they do not metastasize outside the CNS, and they will cause death before they are large enough to cause general effects.

Pituitary adenomas in the brain usually cause endocrinologic signs, depending on the type of excess secretion (see Chapter 16). Headaches and visual signs may result from increased ICP, and compression of the adjacent optic chiasm, nerves, or tracts, resulting in visual disturbances, is common.

Treatment

Surgery is the treatment of choice, if the tumor is reasonably accessible. Chemotherapy is often accompanied by radiation, and the prognosis for many types of tumors is improving as new drugs are developed. Some targeted drugs are being used to reduce blood flow to the tumor. It continues to be difficult to deliver drugs to CNS tumors because of the blood-brain barrier. In some cases, surgery and radiation may cause substantial damage to normal tissue in the CNS.

THINK ABOUT 14-9

a. Explain the specific signs of dysfunction that would be expected in a young child with a cerebellar tumor.
b. Choose a possible tumor site in one cerebral hemisphere and list the signs (focal and general) that would be expected as the tumor grows.
c. Explain why a tumor in the cerebral hemisphere may grow quite large before any signs appear, but a brain stem tumor causes signs in the early stages.
d. Explain why the general signs of cancer, such as weight loss and anemia, do not develop with brain tumors.
e. Explain why the brain is a common site of metastatic cancer from the lung.

Vascular Disorders

Vascular disorders may be hemorrhagic or ischemic in origin. Interference with blood supply to a specific area of the brain results in local damage and manifestations depending on the particular cerebral artery involved. If the deficit results from hemorrhage, the additional effects of increased intracranial pressure will cause local ischemia and generalized symptoms. Global cerebral ischemia, which may develop secondary to severe shock or cardiac arrest, occurs when impaired perfusion of the entire brain results in loss of function and generalized cerebral edema. In mild cases, confusion and neurologic dysfunction develop temporarily, followed by recovery with no permanent damage. If severe or prolonged ischemia occurs, significant diffuse necrosis or infarction results in deep coma. If death does not result, a vegetative state may ensue.

Transient Ischemic Attacks

A transient ischemic attack (TIA) results from a temporary localized reduction of blood flow in the brain. Recovery occurs within 24 hours. Transient ischemic attacks may occur singly or in a series.

Pathophysiology

A TIA may be caused by partial occlusion of an artery, caused by atherosclerosis, or from a small embolus, a vascular spasm, or local loss of autoregulation. Transient ischemic attacks are advantageous if they serve as a warning and lead to early diagnosis and treatment of a problem before the occurrence of a cerebrovascular accident (CVA, or stroke). The brain must have a constant source of glucose and oxygen or suffer permanent damage. Not all strokes are preceded by TIAs.

Signs and Symptoms

The manifestations of TIA are directly related to the location of the ischemia. The patient remains conscious. Intermittent short episodes of impaired function, such as muscle weakness in an arm or leg, visual disturbances, or numbness and **paresthesia** in the face, may occur. Transient aphasia or confusion may develop. The attack may last a few minutes or longer but rarely lasts more than 1 to 2 hours, and then the signs disappear. Repeated attacks are frequently a warning of the development of obstruction related to atherosclerosis.

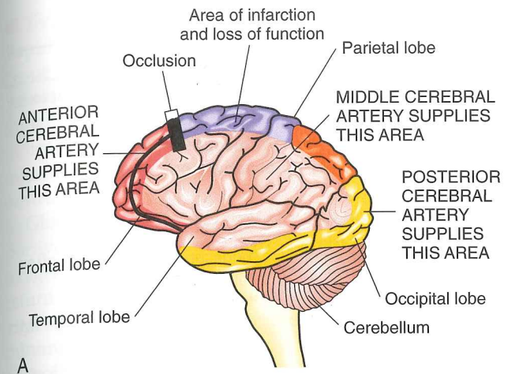
Such attacks should be investigated immediately and treatment instituted, depending on the cause, to prevent permanent brain damage.

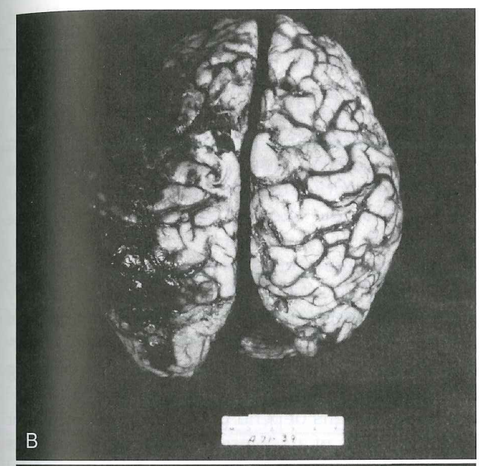
Cerebrovascular Accidents (Stroke)

It is estimated that in United States, someone has a stroke every 45 seconds. Of the approximate 700,000 strokes experienced annually, about 500,000 are first strokes and 200,000 are recurrent attacks. Cerebrovascular accidents (CVAs) account for more than 1 out of 15 deaths (about 160,000 per year). Incidence increases with age, most occurring in people who are over 65 years old. Strokes are considered a major cause of disability.

Pathophysiology

A CVA (stroke) is an infarction of brain tissue that results from lack of blood. Tissue necrosis may be an outcome of total occlusion of a cerebral blood vessel by atheroma or embolus, which causes ischemia, or may be the consequence of a ruptured cerebral vessel, which causes hemorrhage and increased intracranial pressure (see Fig. 14-13). Five minutes (or less) of ischemia causes irreversible nerve cell damage. A central area of necrosis develops, surrounded by an area of inflammation, and





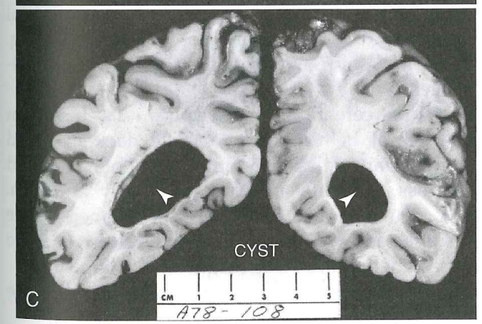


FIGURE 14-13 **A**, Effects of cerebrovascular accident (CVA). **B**, External superior surface of the brain showing acute hemorrhagic infarction. Note blood vessels on surface of brain. **C**, Cut surface of brain showing cyst from healed infarction. *(Courtesy of R.W. Shaw, MD, North York General Hospital, Toronto, Ontario, Canada.)*

function in this area is lost immediately. The tissue liquefies, leaving a cavity in the brain.

The development and effects of a stroke vary with the cause. There are three common categories (Table 14-8), described as follows:

- Occlusion of an artery by an atheroma is the most common cause of CVA. (*Atherosclerosis* is discussed in Chapter 12.) Atheromas often develop in the large arteries, such as the carotid arteries. This condition causes gradual narrowing of the arterial lumen by plaque and thrombus, leading to possible TIAs and eventually infarction.
- A sudden obstruction caused by an *embolus* lodging in a cerebral artery is the second type of stroke. Thrombi may break off of an atheroma, or mural thrombi may form inside the heart after a myocardial infarction and then break away. Emboli can also result from other materials, such as tumors, air, or infection (e.g., endocarditis).
- Intracerebral *hemorrhage*, usually caused by rupture of a cerebral artery in a patient with severe hypertension (see Chapter 12), is the third class of stroke. Hemorrhagic strokes are frequently more severe and destructive than other CVAs, because they affect large portions of the brain (see Fig. 14-13*B*). Because of the greater increase in ICP with hemorrhage, the effects are evident in both hemispheres and are complicated by the secondary effects of bleeding, in addition to the disrupted blood supply. The presence of free blood in interstitial areas affects the cell membranes and can lead to significant secondary damage as vasospasm, electrolyte imbalances, acidosis, and cellular edema develop.

An MRI can determine the cause of the stroke or illustrate other possible causes of the dysfunction.

Cerebral edema and an increasing area of infarction in the first 48 to 72 hours tend to increase the neurologic deficits. As the inflammation subsides, neurologic function increases. The inflammation and pressure in the brain must be minimized as quickly as possible and therapy instituted to dissolve thrombi and maintain adequate perfusion to limit the area of permanent damage. Collateral circulation may have already developed in areas gradually affected by atherosclerosis (see Chapter 12). Because neurons do not regenerate, an area of residual scar tissue and often cysts remains, with a permanent loss of neurons in that area (see Fig. 14-13*C*). In many cases, because specific functions result from integrated output from many areas, it is possible with intensive therapy for a person who has experienced a stroke to develop new neural pathways in the brain or to relearn a task, thus recovering some lost function.

Complications are common. These include recurrent CVA; secondary problems related to immobility such as pneumonia, aspiration, and constipation; or contractures related to paralysis.

TABLE 14-8 Types of Cerebrovascular Accidents

	Thrombus	Embolus	Hemorrhage
Predisposing condition	Atherosclerosis in cerebral artery	Atherosclerosis (carotid artery) or systemic source (e.g., heart)	Hypertension—arteriosclerosis
Onset	Gradual—may be preceded by transient ischemic attacks Often occurs often at rest	Sudden	Sudden—often occurs with activity
Increased ICP	Minimal	Minimal	Present; often high
Effects	Localized—may be less permanent damage if collateral circulation has been established	Localized unless multiple emboli are present	Widespread and severe—often fatal

Etiology

Risk factors for stroke include diabetes, hypertension, systemic lupus erythematosus, elevated cholesterol levels, hyperlipidemia, atherosclerosis, a history of TIAs, increasing age, obstructive sleep apnea, and heart disease. The risk factors for atherosclerosis (see Chapter 12) apply similarly to CVA. The combination of oral contraceptives and cigarette smoking has been well documented as an etiologic factor. Emboli may arise from atheromas in the large arteries, such as the carotids, or from cardiac disorders of the left ventricle, such as acute myocardial infarction, atrial fibrillation, or endocarditis or from an implant such as a prosthetic valve. Severe or long-term hypertension and arteriosclerosis in the elderly increase the risk of intracerebral hemorrhage.

WARNING SIGNS OF STROKE (CVA, OR BRAIN ATTACK)

1. Sudden transient weakness, numbness, or tingling in the face, an arm or leg, or on one side of the body
2. Temporary loss of speech, failure to comprehend, or confusion
3. Sudden loss of vision
4. Sudden severe headache
5. Unusual dizziness or unsteadiness

Immediate medical treatment may prevent permanent brain damage.

Signs and Symptoms

The National Institutes of Health has developed a diagnostic *stroke scale* that is designed to assist with rapid diagnosis of a cerebral vascular accident in an emergency situation. The stroke scale includes commands to determine capacity for speech, level of consciousness, motor abilities, and assessment of eye movements. The scale also identifies areas of damage based on resulting dysfunction.

Signs and symptoms depend on the location of the obstruction, the size of the artery involved, and the functional area affected (see Figs. 14-13*A* and 14-3). The presence of collateral circulation may diminish the size of the affected area. There are "silent" areas of the brain, in which dysfunction resulting from small infarctions is not obvious. Obstruction of small arteries may not lead to obvious signs until several small infarctions have occurred. In some cases, the effects of a stroke develop slowly over a period of hours (termed *evolving stroke*).

Initially flaccid paralysis is present; spastic paralysis develops several weeks later, as the nervous system recovers from the initial insult. Generally the functional deficits increase during the first 48 hours as inflammation develops at the site and then subside as some neurons around the infarcted area recover.

Occlusion of large arteries, such as the internal carotid artery or the middle cerebral artery, or a hemorrhage may cause severe, widespread effects, including coma, loss of consciousness, or death, almost immediately. Hemorrhagic strokes usually begin suddenly with a blinding headache and increasingly severe neurologic deficits.

Specific local signs depend on the area affected. For example, occlusion of an anterior cerebral artery affects the frontal lobe. Common signs include contralateral muscle weakness or paralysis, sensory loss in the leg, confusion, loss of problem-solving skills, and personality changes.

The middle cerebral artery supplies a large portion of the cerebral hemisphere; therefore, lack of blood supply to this artery leads to contralateral paralysis and sensory loss, primarily of the upper body and arm. Aphasia occurs when the dominant hemisphere of the brain is affected, whereas spatial relationships may be more severely impaired if the right side is damaged.

Because the posterior cerebral artery supplies the occipital lobe, visual loss is likely if it is occluded.

EMERGENCY FIRST AID FOR STROKE (CVA, OR BRAIN ATTACK)

1. Call 911 immediately and state the person has the symptoms of a stroke.
2. The patient should be transported to hospital as quickly as possible with a record of common drugs used and medical conditions being treated.
3. Time between onset of the stroke and treatment is directly related to the severity of the damage to the brain. Minutes count!

Treatment

Rapid treatment with "clot-busting agents," such as tissue plasminogen activator (tPA) (see Chapter 12), has reduced the effects of CVA in some individuals, but initial screening to rule out hemorrhage or other contraindications for anticoagulant drugs is essential. Surgical intervention may be possible to relieve carotid artery obstruction.

Glucocorticoids may reduce cerebral edema. Supportive treatment to maximize cerebral circulation and oxygen supply is usually initiated. Assisting the patient's return to a sitting or standing position as soon as the vital signs are stable helps to maintain muscle tone and minimize perceptual deficits.

A team approach to care, including occupational and physical therapists and speech-language pathologists, encourages recovery and minimizes complications in patients in whom many basic functions are impaired. Speech, mobility, swallowing, and other functions may be affected in one individual. Correct positioning, frequent changes of position, and passive exercises to prevent muscle atrophy, contractures, and skin breakdown are required (see Chapter 25). Rehabilitation programs should be instituted as soon as possible.

The underlying problem (hypertension, atherosclerosis, or thrombus) requires treatment to prevent recurrences. The prognosis varies considerably, depending on the underlying causative factors, the artery affected, and the general health status of the individual.

Approximately 20% of stroke patients die within the first few days. Complete recovery is rare. However, the sooner improvement and rehabilitation therapy begin, the more optimistic the prognosis can be. Newer therapies such as constraint induced therapy, which stimulates the use of the weaker side of the body, can be effective many years after the occurrence of a CVA. Ongoing assessment and rehabilitation are of value to any client who has experienced a CVA.

Cerebral Aneurysms

Pathophysiology

An aneurysm is a localized dilation in an artery. Cerebral aneurysms are frequently multiple and usually occur at the points of **bifurcation** on the circle of Willis (Fig. 14-14). These "berry" aneurysms develop where

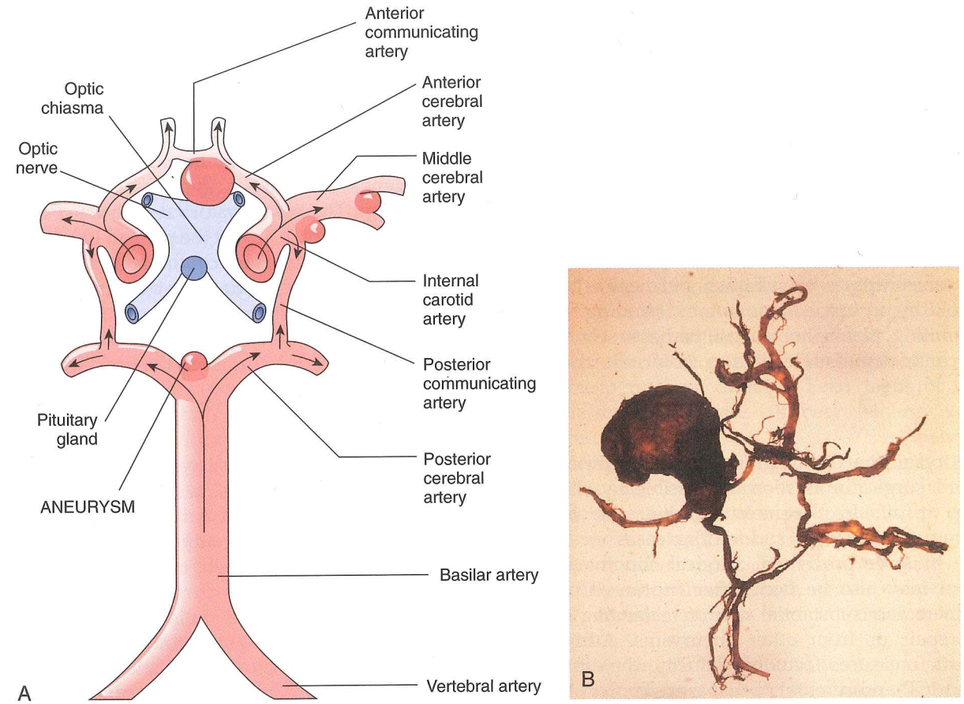


FIGURE 14-14 **A**, Cerebral aneurysm. **B**, Dissected circle of Willis showing a large cerebral aneurysm. *(From Kumar V, Abbas AK, Fausto M:* Robbins and Cotran Pathologic Basis of Disease, *ed 7, Philadelphia, 2005, Saunders.)*

there is a weakness in the arterial wall where branching occurs. The force of blood at this point leads to bulging in the wall, which is often aggravated by hypertension. Initially the aneurysms are small and *asymptomatic*, but they tend to enlarge over years, until compression of the nearby structures (e.g., a cranial nerve) causes clinical signs or rupture occurs.

Rupture often results from a sudden increase in blood pressure during exertion, and bleeding occurs into the subarachnoid space (the location of the circle of Willis) and the CSF. This rupture may be a small leak or a massive tear. Blood is irritating to the meninges and causes an inflammatory response and irritation of the nerve roots passing through the meninges. This free blood also causes *vasospasm* in the cerebral arteries, further reducing perfusion and leading to additional ischemia. Hemorrhage from the ruptured vessel causes increased ICP and its associated signs. No focal signs are present because the additional blood is dispersed through the system. Subarachnoid hemorrhages may be classified according to their clinical effects.

Signs and Symptoms

The enlarging aneurysm may cause pressure on the surrounding structures, such as the optic chiasm or the cranial nerves, leading to loss of the visual fields (see Figs. 14-7 and 14-14) or other visual disturbances. The mass may also result in headache as tension increases on the blood vessel wall and meninges.

A small leak is likely to cause headache, **photophobia** (increased sensitivity of the eyes to light), and intermittent periods of dysfunction, such as confusion, slurred speech, or weakness. **Nuchal rigidity**, or a stiff, extended neck, often develops because the escaped blood irritates the spinal nerve roots and causes muscle contractions in the neck.

A massive rupture or subarachnoid hemorrhage is manifested by an immediate, severe, "blinding" headache, vomiting, photophobia, and, perhaps, seizures or loss of consciousness. Death may occur shortly after rupture.

Treatment

An aneurysm that is diagnosed *before* rupture can be treated surgically as soon as possible by clipping or tying it off. In the interim, while the patient is waiting for surgery, sudden increases in blood pressure must be prevented. Surgical clipping of the aneurysm may also be done after rupture. Unfortunately, there is a substantial risk of rebleeding at the site of repair or from other aneurysms. Additional therapeutic measures focus on reducing the effects of increased ICP and cerebral vasospasm. Approximately 35% of patients die with the initial rupture, and an additional 15% die of a second rupture within several weeks.

THINK ABOUT 14-10

a. Differentiate a TIA from a CVA with regard to the cause of each and the effects of each on function.
b. Describe the three causes of CVAs and the characteristic onset of signs with each.
c. Describe several factors that influence the degree of functional recovery that is attained after a CVA.
d. List common signs of an expanding aneurysm and of a bleeding aneurysm.
e. Why does a headache occur with a subarachnoid hemorrhage?
f. Explain why skin breakdown or ulcers may occur in a person who has had a stroke and list the common sites of these problems.

Infections

Meningitis

Meningitis is an infection, usually of bacterial origin, in the meninges of the CNS. Many microbes can infect the CNS and all age groups are susceptible. Early diagnosis and treatment is essential to prevent deficits or death.

Pathophysiology

Microorganisms reach the brain via the blood, by extension from nearby tissue, or by direct access through wounds. Microbes such as meningococcus can bind to nasopharyngeal cells in an individual, cross the mucosal barrier, attach to the choroid plexus, and enter CSF.

Because the membranes are continuous around the CNS and CSF flows in the subarachnoid space, infection spreads rapidly through the coverings of the brain. Focal signs are absent because there is no localized mass of infection. The inflammatory response to the infection leads to increased ICP, and the pia and arachnoid layers become edematous. The common bacterial infections lead to a purulent exudate that covers the surface of the brain and fills the sulci, causing the surface to appear flat. The exudate is present in the CSF, and the blood vessels on the surface of the brain appear dilated (Fig. 14-15*D*).

Etiology

Different age groups are susceptible to different organisms that cause meningitis. In some categories, vaccines have reduced the risk of meningitis:

- In children and young adults, *Neisseria meningitidis*, or *meningococcus*, which is the classic meningitis pathogen, is frequently carried in the nasopharynx of asymptomatic carriers. It is spread by respiratory droplets. Epidemics are common in schools or institutions where close contact between the children is likely to spread the organism. Any close contacts of affected persons should be given prophylactic treatment. This type of meningitis occurs more frequently in late winter and early spring.
- In neonates, *Escherichia coli* is the most common causative organism; this form of meningitis is usually seen in conjunction with a neural tube defect, premature rupture of the amniotic membranes, or a difficult delivery.
- In young children, meningitis results most often from bacterial infections caused by *Haemophilus influenzae* and occurs more often in the autumn or winter.
- In elderly persons and young children, *Streptococcus pneumoniae* is a major cause of meningitis.

In any age group, meningitis may be secondary to other infections, such as sinusitis or otitis, or it may result from an abscess located where the infection can spread through the bone to the meninges (e.g., an abscessed tooth). Any form of head trauma or surgery can result in meningitis from a variety of microorganisms. Aseptic or viral meningitis results from an infection, such as mumps or measles.

Signs and Symptoms

Sudden onset of meningitis is common, with severe headache, back pain, photophobia, and nuchal rigidity (a hyperextended, stiff neck). These signs result from meningeal irritation. Two other clinical signs of meningeal irritation include *Kernig's sign* (resistance to leg extension when lying with the hip flexed) and *Brudzinski's sign* (neck flexion causes flexion of hip and knee). Vomiting, irritability, and lethargy progressing to **stupor** or seizures are common early indicators of increased ICP. Fever and chills with leukocytosis indicate infection.

Meningococcal infections result in a rose colored petechial rash or extensive ecchymoses over the body (see Fig. 14-15*B*). Different signs, including feeding problems, irritability, lethargy, a typical high-pitched cry, and bulging fontanelles, occur in the newborn.

Potential complications include hydrocephalus, if CSF flow is blocked by pus or adhesions, and cranial nerve damage. In some cases, damage to the cerebral cortex may occur, resulting in mental retardation, seizures, or motor impairment.

In **fulminant** (rapidly progressive, severe) cases caused by highly virulent organisms, frequently meningococcal, disseminated intravascular coagulation (see Chapter 10) develops, with associated hemorrhage of the adrenal glands, or meningococcal septicemia may directly cause adrenal hemorrhage (Waterhouse-Friderichsen syndrome) (see Fig. 14-15C). These cases usually result in vascular collapse or shock and death.

Diagnostic Tests

Examination of CSF, obtained by lumbar puncture, confirms the diagnosis. If meningitis is present, the CSF pressure is elevated; it will appear cloudy and usually contains an increased number of leukocytes. The causative organism in the CSF or blood must be identified to ensure adequate and effective treatment.

Treatment

Aggressive antimicrobial therapy (e.g., ampicillin) is required, along with specific treatment measures for ICP and seizures as needed. Glucocorticoids reduce cerebral inflammation and edema. With prompt diagnosis and treatment, the majority of patients survive. The mortality rate in neonatal meningitis is high, and there is some risk of permanent brain damage in young children. Between 10% and 20% of patients with meningitis are estimated to have some neurologic deficits as sequelae.

Vaccines are available as a preventive measure for some types of meningococcal, *S. pneumoniae* and *H. influenzae* meningitis, especially when outbreaks occur. Carriers should be identified in institutional epidemics, and contacts should be notified and treated. Vaccination may be undertaken as a preventive measure when cases occur in institutions such as schools.

Brain Abscess

An abscess is a localized infection, frequently occurring in the frontal or temporal lobes (see Fig. 14-16). There is usually necrosis of brain tissue and a surrounding area of edema. A medical history may be helpful in making the diagnosis. Abscesses usually result from the spread of organisms from ear, throat, lung, or sinus infections; multiple septic emboli from acute bacterial endocarditis; or directly from a site of injury or surgery. Common organisms are staphylococci, streptococci, and pneumococci.

The onset of a brain abscess tends to be insidious. Focal signs indicating neurologic deficits and increasing ICP develop. Both surgical drainage and antimicrobial therapy are required. The mortality rate is around 10%.

Encephalitis

Encephalitis is considered an infection of the parenchymal or connective tissue in the brain and cord, particularly the basal ganglia, although various viruses demonstrate an affinity for particular types of cells. The infection may include the meninges. Necrosis and inflammation develop in the brain tissue, often resulting in some permanent damage. Early signs of infection include severe headache, stiff neck, lethargy, vomiting, seizures, and fever.

Encephalitis is usually of viral origin but may be related to other organisms. In some cases, there may be considerable delay before signs appear. A few examples of specific diseases follow.

Western equine encephalitis is an arboviral infection spread by mosquitoes, which occurs more frequently in the summer months and is common in young children.

St. Louis encephalitis is found throughout the United States and affects older persons more seriously than younger ones.

West Nile fever is a form of encephalitis that originated in the northeastern United States but has now spread to

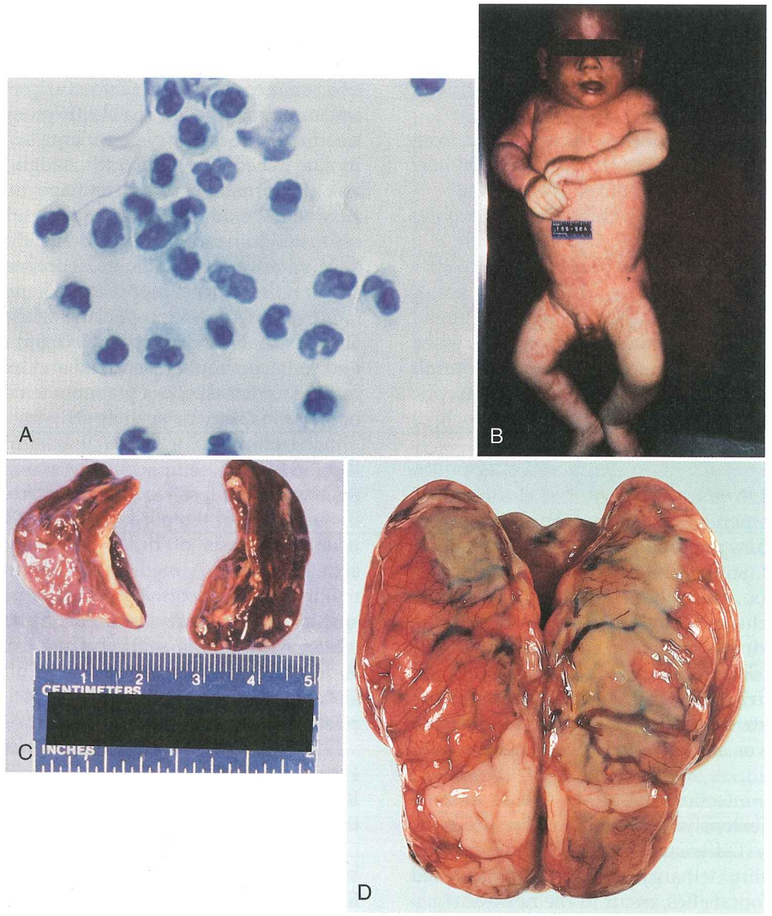


FIGURE 14-15 **A**, Meningitis—slide preparation of CSF showing many neutrophils with bacterial meningitis. **B**, Petechial rash associated with meningococcemia. **C**, Hemorrhage (dark areas) in the adrenal glands with Waterhouse-Friderichsen syndrome. **D**, Meningitis due to coliform microorganisms. The meninges are reddened from vascular congestion. Thick, greenish pus fills the subarachnoid space over both hemispheres. *(**A** from Stevens ML:* Fundamentals of Clinical Hematology, *Philadelphia, 1997, Saunders; **B** and **C** from Mahon CR, Manuselis G:* Textbook of Diagnostic Microbiology, *ed 2, Philadelphia, 2000, Saunders; **D** from Cooke RA, Stewart B:* Colour Atlas of Anatomical Pathology, *ed 3, Sydney, 2004, Churchill Livingstone.)*

a number of states across the country and into Canada. It is caused by a flavivirus, spread by mosquitoes, with certain birds as an intermediate host. The focus for control has been to track the spread and reduce the risk of mosquito bites in affected areas. The infection initially causes flulike symptoms with low-grade fever and headache, sometimes followed by confusion and tremors.

Neuroborreliosis (Lyme disease) is caused by a spirochete, *Borrelia burgdorferi*, transmitted by tick bites in summertime. The site of the tick bite is red with a pale center, gradually increasing in size to form the unique marker lesion, a "bull's eye" that may become quite large and persist for some time. The microbes then disseminate through the circulation, causing first, sore throat, dry cough, fever, and headache, followed by cardiac arrhythmias and neurologic abnormalities (e.g., facial nerve paralysis) related to meningoencephalitis. Last, pain and swelling may develop in large joints, sometimes progressing to chronic arthritis. The effects may persist for months. Prolonged therapy with antimicrobials such as doxycycline is prescribed.

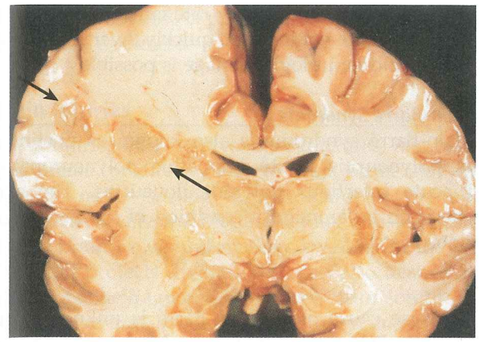


FIGURE 14-16 **Brain—Frontal Abscess** Infection causes central liquefactive necrosis and surrounding edema. *(From Kumar V, Abbas AK, Fausto M:* Robbins and Cotran Pathologic Basis of Disease, *ed 7, Philadelphia, 2005, Saunders.)*

Herpes simplex encephalitis occurs occasionally and is dangerous, arising from the spread of herpes simplex virus type 1 (HSV-1) from the trigeminal nerve ganglion. This virus causes extensive necrosis and hemorrhage in the brain, often involving the frontal and temporal lobes. Early treatment with an antiviral drug, such as acyclovir, may control the infection. Otherwise, treatment is supportive.

THINK ABOUT 14-11

a. List the signs of developing meningitis.
b. Describe the changes that occur in the CSF with meningitis.
c. What are the causes of death in meningococcal infection?
d. Why does an abscess cause focal signs, whereas meningitis does not?

Other Infections

Many other microorganisms specifically target the central and/or peripheral nervous systems. Brief descriptions of several of the diseases they cause are given here.

Rabies (hydrophobia) is caused by a virus that is transmitted by the bite of a rabid animal. The virus travels along peripheral nerves to the CNS, where it causes severe inflammation and necrosis, particularly in the brain stem and basal ganglia. The incubation period (often 1 to 3 months) depends on the distance between the bite and access to the CNS. Onset is marked by headache and fever; nervous hyperirritability, including sensitivity to touch; and seizures. The virus also travels to the salivary glands. Difficulty swallowing, caused by muscle spasm, and foaming at the mouth are typical. Respiratory failure causes death. Immediate cleansing of the bite area and prophylactic immunization are necessary treatments.

Tetanus (lockjaw) is caused by *Clostridium tetani*, a spore-forming bacillus. The spores survive for years in soil. The vegetative form is an anaerobe, thriving deep in tissues, for example, in a puncture wound. The exotoxin enters the nervous system, causing tonic muscle spasms. Symptoms of infection include jaw stiffness, difficulty swallowing, stiff neck, headache, skeletal muscle spasm, and eventually respiratory failure. The mortality rate is 50%. Immunizations are advised, with boosters as needed or after injury.

Poliomyelitis (infantile paralysis) is now rare in North America because of immunization but still occurs in other parts of the world. An immunization program is underway to combat an epidemic occurring in West and Central Africa, where large numbers of young children have been infected and threatened with paralysis in large numbers. The goal of the World Health Organization (WHO) is worldwide eradication through immunization. The polio virus is highly contagious through direct contact or oral droplet. It reproduces in lymphoid tissue in the oropharynx and digestive tract, and then enters the blood and, eventually, the CNS. The virus attacks the motor neurons of the spinal cord and medulla, causing minor flulike effects in many cases, but paralysis and respiratory failure in other cases, depending on the level of the destruction. Symptoms include fever, headache, and vomiting, followed by the typical stiff neck, pain, and flaccid paralysis.

Other microorganisms, such as *Candida albicans* or *Toxoplasma gondii*, may cause infection in the brain, most often in immune-suppressed individuals.

Infection-Related Syndromes

Herpes Zoster (Shingles)

Herpes zoster or shingles is caused by varicella-zoster virus (VZV) in adults. It is seen years after the primary infection of varicella or chickenpox, which usually occurs in childhood.

Shingles usually affects *one cranial nerve* or *one dermatome*, a cutaneous area innervated by a spinal nerve (see Fig. 14-22 for dermatomes) on *one* side of the body. Pain, paresthesia, and a vesicular rash develop in a line, unilaterally. This may occur on the face (e.g., following the trigeminal nerve) or along the path of a lumbar nerve from the spine extending around one side in the hip area. The lesions persist for several weeks and then clear in the majority of cases.

In some cases, particularly in older individuals, neuralgia or pain continues after the lesions disappear. In patients with immune deficiencies, the lesions tend to spread locally. Visual impairment has resulted from involvement of the ophthalmic division of the trigeminal nerve. Antiviral medications such as acyclovir or vidarabine have provided some relief from symptoms. A new shingles vaccine, Zostrix, is available for those 60

years of age or older. It prevents initial and recurrent outbreaks as well as reducing postherpetic nerve pain.

Postpolio Syndrome

Postpolio syndrome (PPS) has been occurring 10 to 40 years after recovery from the original infection, with progressive and debilitating fatigue, weakness, pain, and muscle atrophy. It is estimated that 25% to 50% of individuals infected with polio will develop PPS. Symptoms have developed in individuals who, as young children in the 1950s and earlier, were diagnosed with mild forms as well as paralytic forms of polio, and in those who were misdiagnosed at the time, but are now considered to have been infected. The more severe the original infection was, the more severe the effects of PPS. The syndrome does not appear to be a recurrence of latent infection, but the precise basis for the neuronal damage has not been determined. It appears that surviving motor neurons have now degenerated and died, possibly because they developed new additional axon branches to serve muscle cells as compensation for damage, but could not maintain them.

Reye's Syndrome

Pathophysiology

The cause of Reye's syndrome has not yet been fully determined, but it is linked to a viral infection, such as influenza, in young children that have been treated with aspirin (ASA). Depending on the particular virus, signs appear 3 to 5 days after the onset of the viral infection. The number of cases has decreased with awareness of this potential danger, and acetaminophen is now used to treat fever in children.

The major pathologic changes occur in the brain and the liver. A noninflammatory cerebral edema develops, leading to increased ICP. Brain function is severely impaired by cerebral edema and the effects of high ammonia levels in serum related to liver dysfunction.

The liver enlarges, develops fatty changes in the tissue, and progresses to acute failure. Jaundice is not present, but serum levels of liver enzymes are elevated. The resultant metabolic abnormalities include hypoglycemia and increased lactic acid in the blood and body fluids, which also contribute to acute encephalopathy. In some cases, the kidneys are also affected by fatty degenerative changes, leading to increases in serum urea and creatinine levels.

Signs and Symptoms

Manifestations vary in severity. Encephalopathy initially causes lethargy, headache, and vomiting, which are quickly followed by disorientation, hyperreflexia, hyperventilation, seizures, stupor, or coma.

Treatment

There is no immediate cure. Treatment is supportive and symptomatic, managing the metabolic imbalances and cerebral edema. The mortality rate is high if diagnosis and treatment are not initiated quickly. Average mortality is 30% and neurologic damage is possible.

Guillain-Barré Syndrome

Guillain-Barré syndrome is also known as postinfectious polyneuritis, acute idiopathic polyneuropathy, and acute infectious polyradiculoneuritis. The syndrome is an inflammatory condition of the peripheral nervous system.

Pathophysiology

The precise cause of Guillain-Barré is unknown, but evidence indicates that an abnormal immune response, perhaps an autoimmune response, precipitated by a preceding viral infection or immunization, may be responsible. Local inflammation, accompanied by accumulated lymphocytes, demyelination, and axon destruction, occurs. These changes cause impaired nerve conduction, particularly in the efferent (motor) fibers, although afferent (sensory) and autonomic fibers may also be involved. If the cell body remains alive through the acute period, the axon can regenerate. Initially the inflammatory and degenerative processes affect the peripheral nerves in the legs; then the inflammation ascends to involve the spinal nerves to the trunk and neck and frequently includes the cranial nerves as well. The critical period develops when the ascending paralysis involves the diaphragm and respiratory muscles. Recovery is usually spontaneous with the manifestations diminishing in reverse order; that is, motor function is regained first in the upper body and then gradually improves in the trunk and the lower extremities.

Signs and Symptoms

Progressive muscle weakness and areflexia, beginning in the legs, lead to an ascending flaccid paralysis, which may be accompanied by paresthesia, or pain and general muscle aching. As paralysis advances upward, vision and speech may be impaired. This process may occur rapidly over a few hours or several days. If swallowing and respiration are affected, a life-threatening situation develops. Many patients sustain autonomic nervous system impairment, manifested as cardiac arrhythmias, **labile** (fluctuating) blood pressure, or loss of sweating capability.

Treatment

Treatment is primarily supportive, and a ventilator is required in many cases. The use of immunoglobulin therapy or plasmapheresis, in which IgG is separated and removed from the patient's blood, in the early stage may shorten the acute period of the disease in some patients and hasten recovery. Physiotherapy, occupational therapy, and respiratory therapy throughout the recovery period are essential to maximize restoration of function. About 30% of patients experience some degree of residual weakness.

Brain Injuries

Brain injuries may involve skull fractures, hemorrhage and edema, or direct injury to brain tissue. An injury may be mild, causing only bruising of the tissue, or it can be severe and life-threatening, causing destruction of brain tissue and massive swelling of the brain. The skull protects the brain but can also destroy it by means of bone fragments that penetrate or compress the brain tissue and by its inability to expand to relieve pressure.

Types of Head Injuries

Various terms are used to classify and describe brain trauma, in some cases with overlap, as follows:

- *Concussion, also termed mild traumatic brain injury (MTBI)* is a reversible interference with brain function, usually resulting from a mild blow to the head, which causes sudden excessive movement of the brain, disrupting neurologic function and leading to loss of consciousness. **Amnesia**, or memory loss, and headaches may follow a concussion, but recovery with no permanent damage usually occurs within 24 hours. Recurrent concussions have been shown to cause progressive and permanent brain damage; thus at-risk individuals may need to change activities to prevent further damage.
- *Contusion* is a bruising of brain tissue with rupture of small blood vessels and edema that usually results from a blunt blow to the head. The possibility of residual damage depends on the force of the blow and the degree of tissue injury.
- *Closed* head injury occurs when the skull is not fractured in the injury, but the brain tissue is injured and blood vessels may be ruptured by the force exerted against the skull (Fig. 14-17). Extensive damage may occur when the head is rotated with considerable force.
- *Open* head injuries are those involving fractures or penetration of the brain by missiles or sharp objects.
- *Linear* fractures are simple cracks in the bone.
- *Comminuted* fractures consist of several fracture lines but may not be complicated.
- *Compound* fractures involve trauma in which the brain tissue is exposed to the environment and is likely to be severely damaged because bone fragments may penetrate the tissue and the risk of infection is high.
- *Depressed* skull fractures involve displacement of a piece of bone below the level of the skull, thereby compressing the brain tissue. With this type of fracture, the blood supply to the area is often impaired, and considerable pressure is exerted on the brain.
- *Basilar* fractures occur at the base of the skull and are often accompanied by leaking of CSF through the ears or nose. These fractures may occur when the forehead hits a car windshield with considerable force. Cranial nerve damage and dark discoloration around the eyes are common.
- *Contrecoup* injury occurs when an area of the brain contralateral to the site of direct damage is injured as the brain bounces off the skull. This injury may be secondary to acceleration or deceleration injuries, in which the skull and brain hit a solid object, which causes the brain to rebound against the opposite side of the skull, usually causing minor damage.

Pathophysiology

Primary brain injuries are direct injuries, such as lacerations or crushing of the neurons, glial cells, and blood vessels of the brain.

Secondary injuries result from the additional effects of cerebral edema, hemorrhage, hematoma, cerebral vasospasm, infection, and ischemia related to systemic factors.

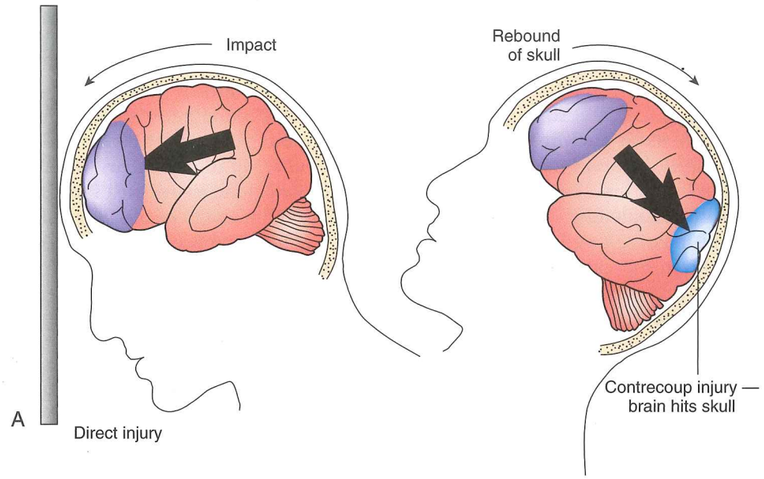
Primary injuries may involve a laceration or compression of brain tissue by a piece of bone or foreign object or rupture or compression of the cerebral blood vessels. Because the brain is not held tightly in place, the application of unusual force may rotate or shift it inside the skull. The brain tissue may be damaged by the rough and irregular inner surface of the skull or by the movement of the lobes of the brain against each other (shearing injury).

Any trauma to the brain tissue causes loss of function in the part of the body controlled by that specific area of the brain. Cell damage and bleeding lead to inflammation and vasospasm around the site of the injury, increased ICP and further general ischemia and dysfunction (Fig. 14-18). After the bleeding and inflammation subside, some recovery of the neurons in the area surrounding the direct damage may occur. The central area of damage undergoes necrosis and is replaced by scar tissue or a cyst.

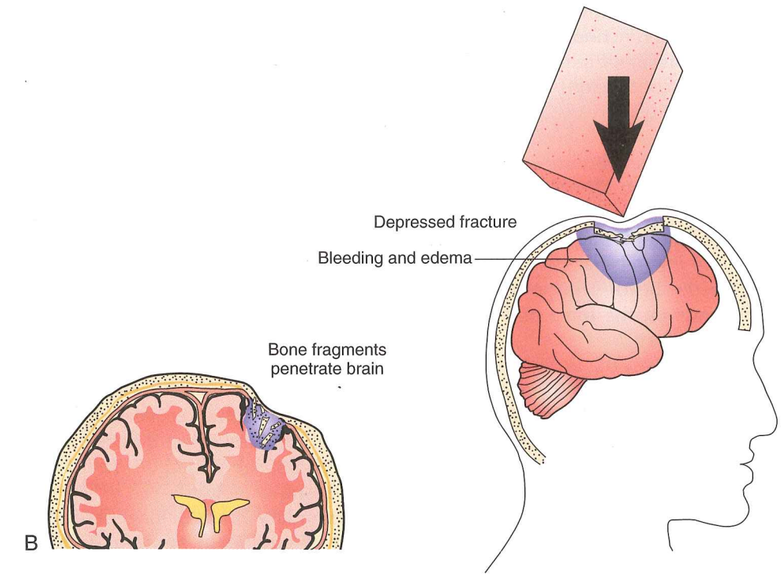
Secondary brain damage is caused by the development of additional injurious factors. A *hematoma* is a collection of blood in the tissue that develops from ruptured blood vessels, either immediately after the injury or after some delay (Fig. 14-19). Hematoma may also develop after surgery.

Hematomas and hemorrhages are classified by their location in relation to the meninges, as follows:

- *Epidural* (extradural) *hematoma* results from bleeding between the dura and the skull, usually caused by tearing of the middle meningeal artery in the temporal region. Signs of trouble usually arise within a few hours of injury, when the person loses consciousness after a brief period of responsiveness.
- *Subdural hematoma* develops between the dura and the arachnoid (Fig. 14-20). Frequently there is a small



Closed Injury — Direct and Contrecoup Injury



Open Injury

FIGURE 14-17 Types of head injury—closed and open.

tear in a vein, which causes blood to accumulate slowly. A hematoma may be acute (signs present in about 24 hours) or subacute (increasing ICP develops over a week or so). A chronic subdural hematoma may occur in an elderly person, in whom brain atrophy allows more space for a hematoma to develop. Also, a tear in the arachnoid can allow CSF to leak into the subdural space (hygroma), creating additional pressure.

- *Subarachnoid* hemorrhage occurs in the space between the arachnoid and pia and is associated with traumatic bleeding from the blood vessels at the base of the brain. Because the blood mixes with circulating CSF, a localized hematoma cannot form.

POSSIBLE EFFECTS OF HEAD INJURY

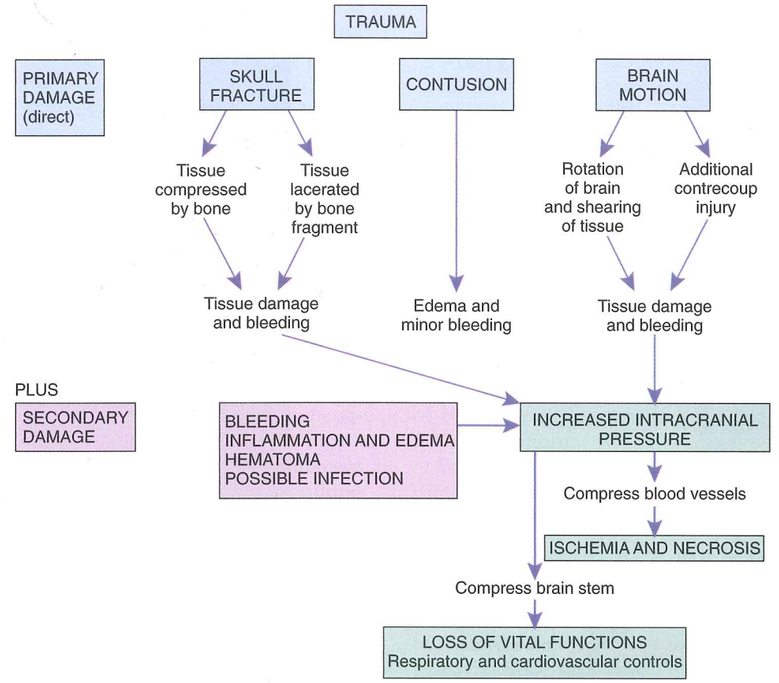


FIGURE 14-18 Possible effects of head injury.

- *Intracerebral hematoma* results from contusions or shearing injuries and may develop several days after injury.

In all types of hematomas, the bleeding leads to local pressure on adjacent tissue and a general increase in ICP. Blood may be partially coagulated, forming a solid mass. When the blood accumulates slowly, the blood cells undergo hemolysis. The fluid in this area of cell breakdown exerts osmotic pressure, drawing more and more water into the area, increasing the size and pressure of the mass, and raising the ICP. Herniation may result from an untreated mass. Any bleeding in the brain may precipitate cerebral vasoconstriction (vasospasm), leading to further ischemia and more damage to the neurons.

Other factors that may cause secondary brain damage include infection, which is usually a significant risk in persons with open head injuries, and hypoxia, which is related to systemic injury or shock. Respiratory or cardiovascular impairment may cause additional ischemia in the brain.

Etiology

The majority of head injuries occur in young adults as a result of sports injuries and accidents involving cars or motorcycles. In many of these accidents, excessive alcohol intake is a contributing factor. Unfortunately, a high blood alcohol level can impede neurologic assessment by masking the signs of injury. Alcohol, because of its dehydrating effects, tends to delay the onset of cerebral edema and elevation of ICP, but there may be a greater increase in ICP at a later time. Other systemic injuries, such as a chest injury or shock, can have the same effect.

Falls are a common cause of head injury in any age group, but more often in elderly persons. Boxers and other athletes engaged in contact sports are at risk for repeated head injury. Infants, when violently shaken, can experience severe damage to the brain and brain stem as the head swings. Other injuries may involve objects that fall on the head or a blow to the head.

Signs and Symptoms

The person with a head injury manifests the characteristic focal signs and the general signs of increased ICP. In addition, one or more of the following may develop:

1. Seizures, which are often focal but may be generalized, occur because of the irritating quality of blood.
2. Cranial nerve impairment may occur, particularly in persons who have sustained basilar fractures.

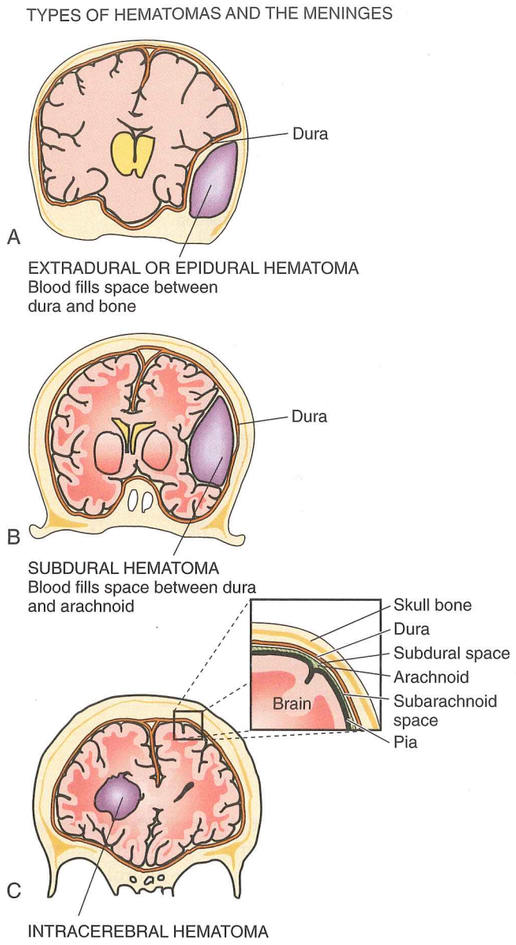


FIGURE 14-19 Types of hematomas.

3. Otorrhea or rhinorrhea (leaking of CSF from the ear or nose, respectively) occurs with fractures and tearing of the meninges, which allows fluid to pass out of the subarachnoid space. This type of trauma provides microbes with an entry point into the brain.
4. Otorrhagia is blood leaking from the ear through a fracture site with torn vessels and meninges.
5. Fever may be a sign of hypothalamic impairment or of cranial or systemic infection.
6. Stress ulcers may develop from increased gastric secretions.

If the individual is unconscious for a prolonged period of time, other problems may develop. Immobility may cause complications such as pneumonia or decubitus ulcers (see Chapter 25).

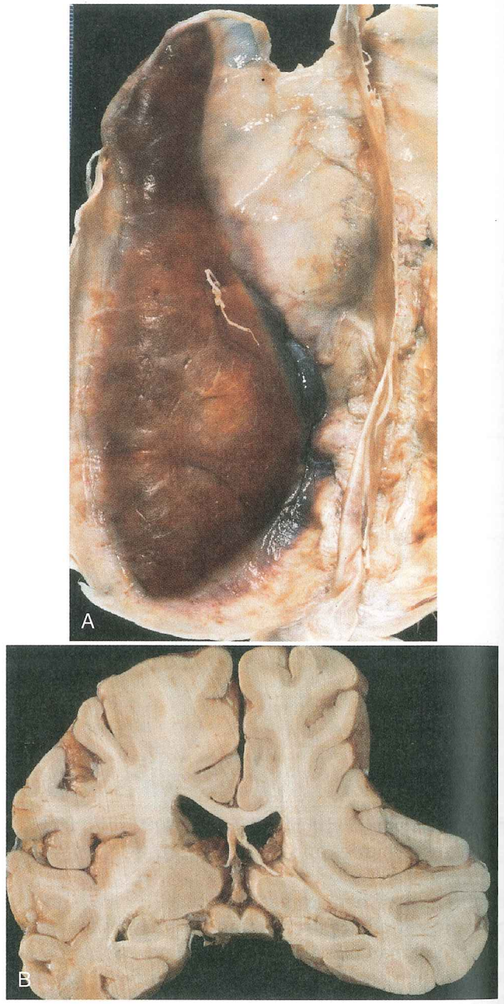


FIGURE 14-20 **A**, Large organizing subdural hematoma attached to the dura. **B**, Coronal section of the brain showing compression of the brain tissue under the hematoma. *(From Kumar V, Abbas AK, Fausto M:* Robbins and Cotran Pathologic Basis of Disease, *ed 7, Philadelphia, 2005, Saunders.)*

Treatment

Computed tomographic (CT) and MRI scans are useful in determining the extent of brain injury. Glucocorticoid agents, which decrease edema, and antibiotic agents, which reduce the risk of infection, are helpful. Surgery may be necessary to reduce ICP. Blood products and oxygen may be administered to protect the remaining brain tissue. Any other injuries must be treated promptly, particularly if they interfere with respiration or circulation.

Individuals with brain injuries may be examined and discharged from the hospital if no brain damage is apparent. The person's family or friends are usually asked to continue to perform a simplified head injury assessment for the next day or so to detect delayed hematoma formation. This routine involves awakening the person periodically to check the level of consciousness (i.e., response to questions and orientation to time, place, and people), checking for reactive pupils, and watching for vomiting or any change in movement, sensation, or behavior. Headache, irritability, and fatigue are often present for a few days in persons with minor injuries.

The prognosis for recovery from a brain injury is better now because of improved surgical techniques, monitoring devices, supportive rehabilitation and drug therapies. Physiotherapy is used to increase mobility. Occupational therapy addresses motor, visual and cognitive activity, whereas speech and language therapy address communication. There may be permanent residual damage in specific areas of the brain, resulting in motor or sensory deficits that may cause disability. Seizures, focal or generalized, are common sequelae because of the increased irritability of tissue around the scar. Often, general fatigue, frequent headaches, and memory loss are present for some time after recovery.

THINK ABOUT 14-12

a. Differentiate an open head injury from a closed head injury in terms of appearance and effects.
b. Describe the location, common source, and time of development of a subdural hematoma.
c. Describe three significant signs of an injury to the right occipital lobe, including one specific focal sign and two general signs.

Spinal Cord Injury

Approximately 11,000 Americans experience spinal cord injuries each year and 200,000 Americans live with ongoing disability due to spinal cord injury. Injury to the spinal cord usually results from fracture or dislocation of the vertebrae, which compresses, stretches, or tears the spinal cord (Fig. 14-21). The supporting ligaments and the intervertebral disc may be damaged also. Most injuries occur in areas of the spine that provide more mobility but less support (i.e., C1 to C7 and T12 to L2) (see Fig. 14-4). A few common types of injuries are described as follows:

- Cervical spine injuries may result from hyperextension or hyperflexion of the neck, with possible fracture. Usually damage to the disc and ligaments occurs, leading to dislocation, loss of alignment of the vertebrae, and compression or stretching of the spinal cord.
- Dislocation of any vertebra may crush or compress the spinal cord and compromise the blood supply.
- Compression fractures cause injury to the spinal cord when great force is applied to the top of the skull or to the feet and is transmitted up or down the spine. Diving into an empty pool, jumping from a height and landing on the feet, or an object falling on a standing person's head may cause this injury. The shattered bone is compressed and protrudes, exerting pressure horizontally against the cord. The sharp edges of bone fragments may lacerate or tear nerve fibers and blood vessels.
- Spinal cord damage also may result directly from penetration injuries, such as stab or bullet wounds.

Vertebral fractures may be classified as simple (single line break), compression (crushed or shattered bone in multiple fragments), wedge (a displaced angular section of bone), or dislocation (a vertebra forced out of its normal position).

Because spinal cord injuries are often unstable, immediate appropriate immobilization is essential to prevent secondary damage.

Pathophysiology

Damage to the spinal cord may be temporary or permanent. Nerves in the spinal cord do not undergo mitosis but axonal regrowth may occur. Laceration of nerve tissue by bone fragments usually results in permanent loss of conduction in the affected nerve tracts. Complete transection (severing) or crushing of the cord causes irreversible loss of all sensory and motor functions at and below the level of injury. Partial transection or crushing injuries may allow recovery of some function.

Bruising is reversible damage when mild edema and minor bleeding temporarily impair conduction of nerve impulses. Any compression of the cord must be relieved quickly to maintain adequate blood supply. Prolonged ischemia and necrosis lead to permanent damage. As with any trauma, bleeding and inflammation develop locally, creating additional pressure and further interfering with blood flow. Edema and hemorrhage extend for several segments above and below the level of injury. In addition, damaged tissue releases mediators such as norepinephrine, serotonin, and histamine in the area. These mediators cause vasoconstriction, leading to additional local ischemia and possible necrosis. Destructive enzymes are released as well, causing more inflammation and necrosis.

Initially the loss of function may appear to be extensive because of this additional compression, but as the edema subsides, there may be partial recovery of function. Regular assessment of movement and sensory response using the dermatome map (Fig. 14-22) can determine the degree of damage or recovery in the spinal cord. When injury occurs in the *cervical* region, the inflammation may extend upward to the level of C3

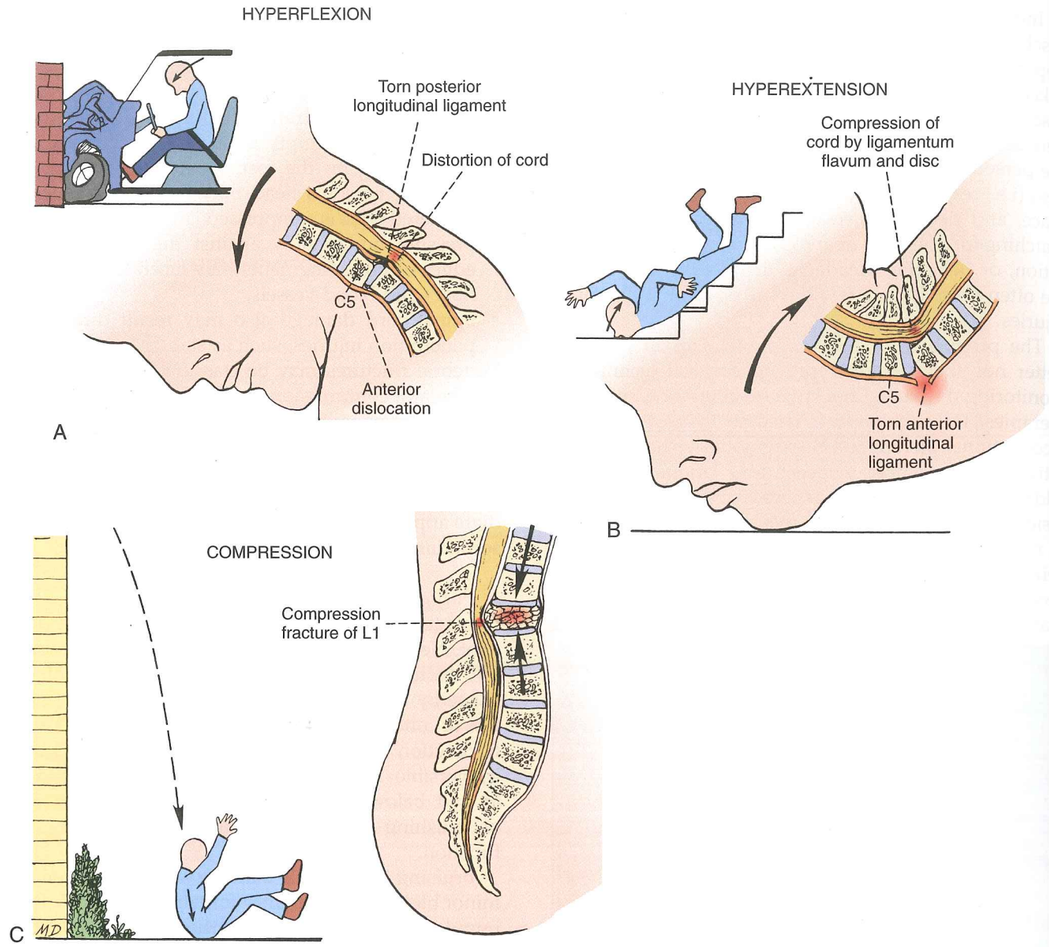


FIGURE 14-21 Types of spinal cord injuries. *(From Copstead LC:* Perspectives on Pathophysiology, *Philadelphia, 1995, Saunders.)*

to C5, interfering with *phrenic nerve* innervation to the diaphragm and therefore affecting respiration. Ventilatory assistance may be required.

In the initial period after the injury, conduction of impulses ceases in the nerve tracts and in the gray matter, a period known as *spinal shock* (which is a form of neurogenic shock). The extent of the injury, the amount of resultant bleeding, and the need for surgical intervention determine the rate and degree of recovery. The inflammation gradually subsides, damaged tissue is removed by phagocytes, and scar tissue begins to form. During this period, reflex activity resumes in the spinal cord below the level of injury, and any undamaged tracts continue to conduct impulses through the level of damage (Fig. 14-23).

Etiology

Most spinal cord injuries occur in young men and around 50% result from motorcycle or automobile accidents. The second most common cause is sports (e.g., diving, football). The other major cause of injury is falls, which elderly persons often experience. The average age for persons with SCI has increased over the past few years and now is 38 years.

Signs and Symptoms

There are two stages in the post-traumatic period, the early stage of spinal shock and increasing impairment, followed by recovery and recognition of the extent of functional loss. During the initial period of *spinal shock*, all neurologic activity ceases at, below, and slightly

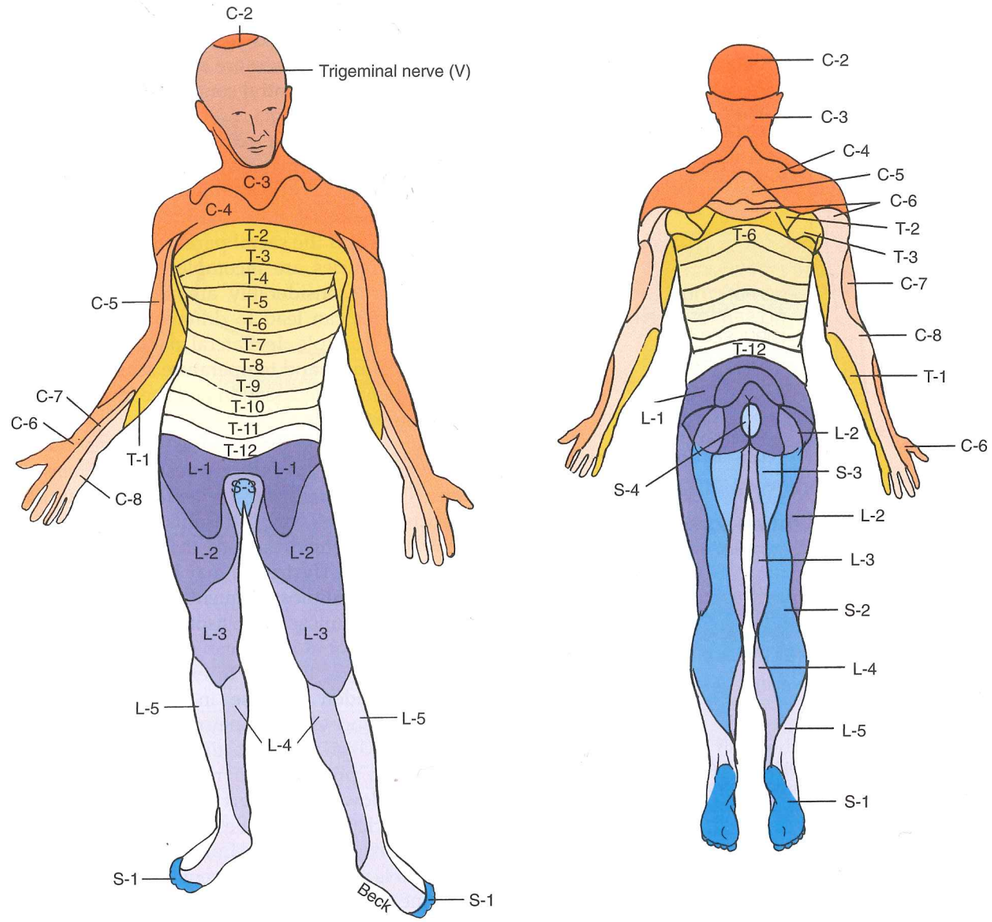


FIGURE 14-22 Dermatomes. *(From Thibodeau GA:* Anatomy and Physiology, *St. Louis, 1987, Mosby.)*

above the level of injury (see Fig. 14-23). No reflexes are present, including the skeletal muscle, sensory, and autonomic systems (bladder and bowel function). This condition may persist for days or weeks. During the period of spinal shock, signs include flaccid paralysis and sensory loss at and below the injured area, an absence of all reflex responses, and loss of central control of autonomic function. In patients with cervical injury, this includes loss of control of vasomotor tone, blood pressure, diaphoresis and body temperature, and bowel and bladder emptying. Blood pressure is low and labile. Urinary retention and paralytic ileus are present.

Recovery from spinal shock is indicated by the gradual return of reflex activity below the level of injury. No impulses, including reflexes, can pass through the specific area of damaged neurons. In most cases, *hyperreflexia* develops, because the normal inhibitory, or "dampening," impulses cannot reach the cord levels below the injury. Following recovery from spinal shock and the return of reflexes, spastic paralysis, sensory deficits, and reflex or neurogenic bladder and bowel function (urinary incontinence and reflex defecation) are present below the level of damage.

Gradually the extent of permanent damage is revealed. For example, a check of the dermatome response can assess sensory function. Voluntary motor activity and sensory impulses are blocked at the level of damage. The specific effects of permanent damage depend on the level at which the spinal cord trauma occurred (see Figs. 14-4 and 14-23). For example, cervical injuries affect motor and sensory function in the arms, trunk, and legs; respiratory function; SNS function (T1 to L2); and sacral parasympathetic fibers. In patients with cervical injuries, respiratory function may

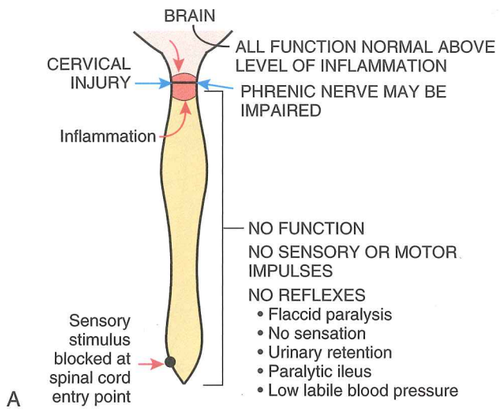


FIGURE 14-23 Effects of spinal cord damage.

continue to be a matter of concern owing to phrenic nerve impairment and the loss of intercostal muscle innervation. Blood pressure and body temperature may be labile, because central control of vasomotor tone and diaphoresis is lacking.

Paralysis of all four extremities is termed ***tetraplegia*** *(quadriplegia)*, whereas *paraplegia* refers to paralysis of the lower part of the trunk and legs. Trauma in the lumbar region interferes with function in the lower extremities and the sacral parasympathetic nerves.

Many injuries are incomplete, and the permanent effects vary considerably among individuals. Partial cord injuries can lead to different patterns of impairment, for example, ipsilateral paralysis and contralateral loss of pain and temperature sensation, depending on the point of decussation and the location of the specific injured tracts.

With injury of the cervical spine, stimulation of the sympathetic system may result in *autonomic dysreflexia* (Fig. 14-24). This is a potentially serious complication caused by a sensory stimulus that triggers a massive sympathetic reflex response that cannot be controlled from the brain. The trigger may be any noxious stimulus in the body, but most frequently is a distended bladder or decubitus ulcer. A sensory stimulus to the SNS below the level of injury can stimulate the entire chain of SNS ganglia, leading to excessive vasoconstriction, with a sudden increase in blood pressure, severe headache, and visual impairment. Bradycardia accompanies this syndrome as the baroreceptors sense the high blood pressure and respond through the vagus nerve by slowing the heart rate. Note that the excessive vasoconstriction cannot be reduced through the cardiovascular control center. Immediate resolution of this problem is necessary to prevent a stroke or heart failure. This means finding and removing the cause of the stimulus and administering medication to lower blood pressure.

Complications are common after spinal cord injury because of immobility and loss of function (see Chapter 25). Contractures may develop from muscle spasms and decubitus ulcers are common; respiratory and urinary infections are frequent.

Sexual function and reproductive capacity are likely to be affected. The sensory and psychological components of the sexual response are usually blocked by the injury. Men may have neurogenic reflex erections. Penetration depends on sustaining this reflex, which can be difficult. Many men, particularly those with high-level cord injuries, are infertile, because sperm production in the testes is impaired. Women usually resume menstrual cycles once they have recovered from the acute trauma period, and they can bear children. Close monitoring of the pregnancy is necessary, and vaginal delivery may be difficult. With counseling and supportive mates, many individuals with spinal cord injury can develop or maintain sexual relationships.

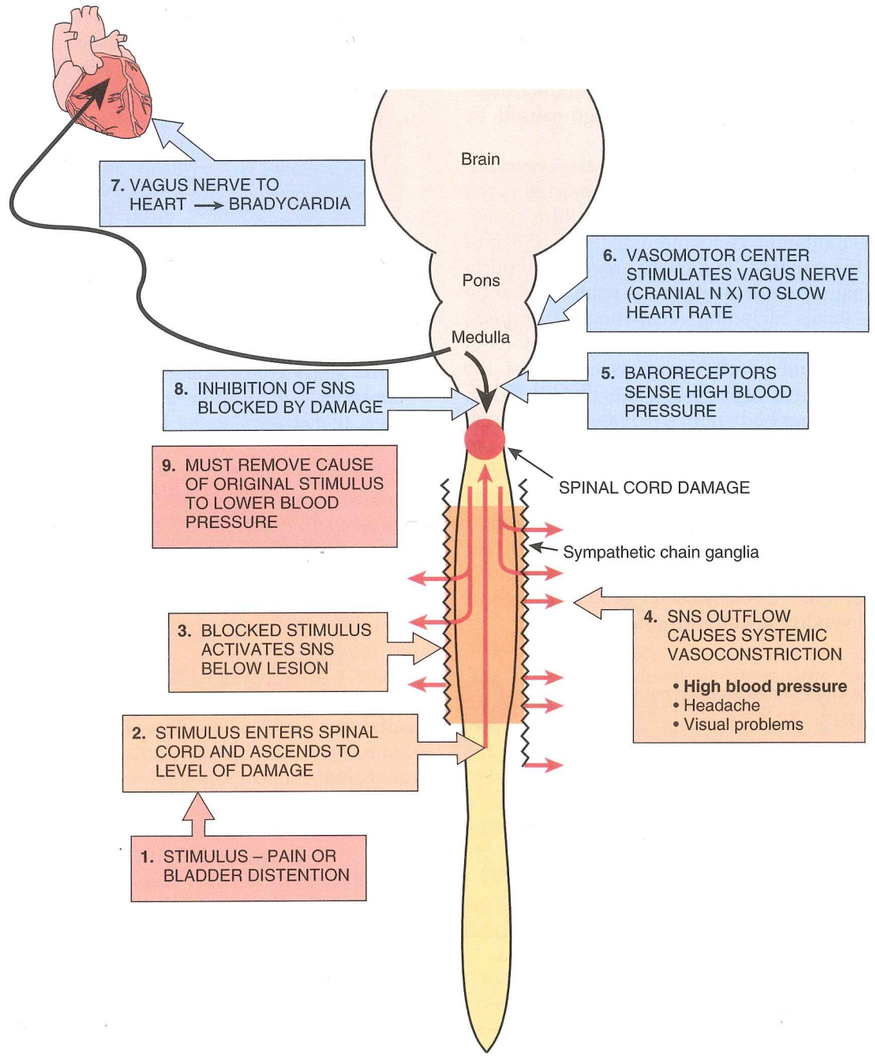


FIGURE 14-24 Autonomic dysreflexia following spinal cord damage.

Treatment

Assessment of damage is usually carried out using the American Spinal Injury Association (ASIA)* criteria:

- A = Complete: No motor or sensory function is preserved in the sacral segments S4-S5
- B = Incomplete: Sensory but not motor function is preserved below the neurologic level and includes the sacral segments S4-S5
- C = Incomplete: Motor function is preserved below the neurologic level, and more than half of key muscles below the neurologic level have a muscle grade less than 3
- D = Incomplete: Motor function is preserved below the neurologic level, and at least half of key muscles below the neurologic level have a muscle grade of 3 or more
- E = Normal: Motor and sensory function are normal.

Treatment and rehabilitation begin at the time of the injury. Care must be taken to immobilize the spine, maintain breathing, and prevent shock. In the hospital,

*From American Spinal Injury Association: International Standards for Neurological Classification of Spinal Cord Injury, reprint Chicago, IL.