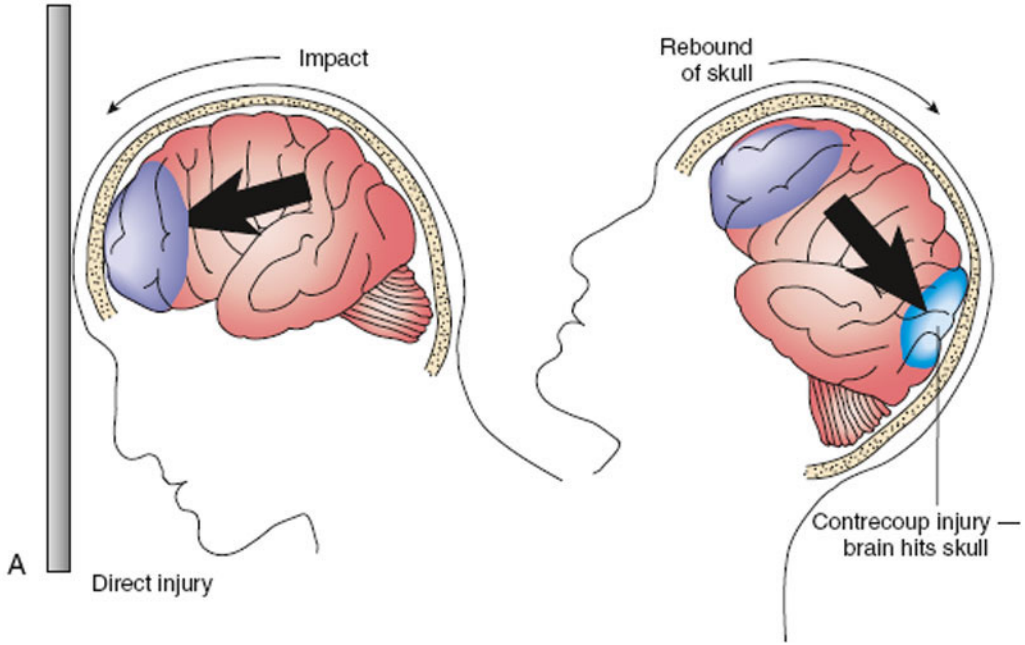
Nervous System Disorders



Closed Injury — Direct and Contrecoup Injury

General Effects of Neurologic Dysfunction

Local (Focal) Effects

- Signs related to the specific area of the brain or spinal cord in which lesion is located
- Examples
  - Paresis or paralysis of the right arm // Results from damage to a section of the left frontal lobe
  - Expanding lesions // Caused by growing tumor or hemorrhage - Additional impairment is noted as adjacent areas become involved.

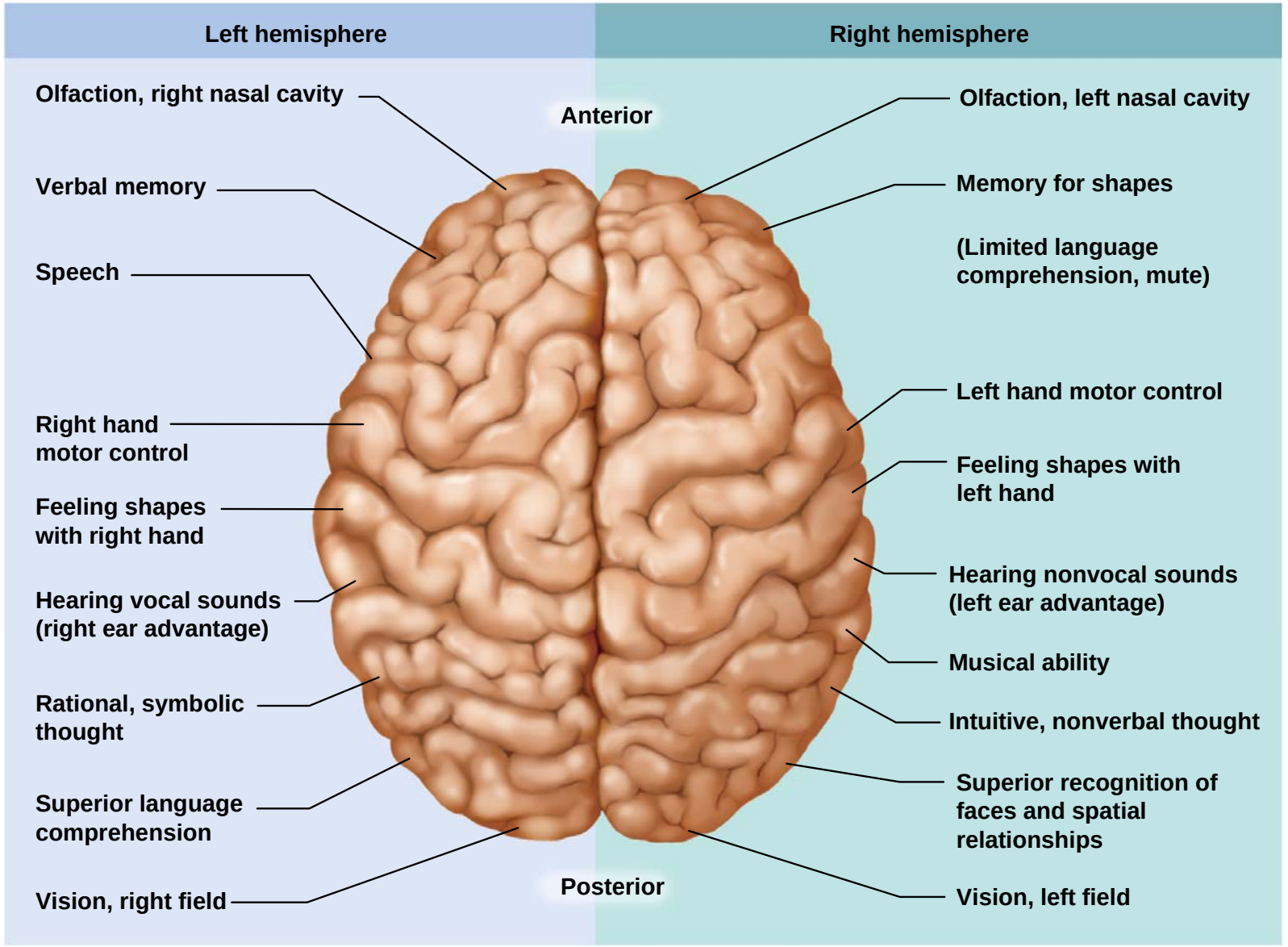
Supratentorial and Infratentorial Lesions

- Supratentorial lesions
  - Occur in the cerebral hemispheres above the tentorium cerebelli // Lead to specific dysfunction in a discrete area
- Infratentorial lesions
  - Located in the brainstem or below the tentorium
  - May affect many motor and sensory fibers // Results in widespread impairment
  - Respiratory and circulatory function may be impaired.
  - Level of consciousness may be impaired.

Left and Right Hemispheres

- Damage to left hemisphere
  - Loss of logical thinking ability, analytical skills, other intellectual abilities, communication skills, language skills
- Damage to right hemisphere
  - Impairs appreciation of music and art
  - Causes behavioral problems
  - Spatial orientation and recognition of relationships may be deficient
  - Self-care deficits common

Cerebral Lateralization



Level of Consciousness

- Decreased level of consciousness or responsiveness
  - Early changes with acute brain disorders
- Levels of reduced consciousness may lead to:
  - Confusion and disorientation
  - Memory loss
  - Unresponsiveness to verbal stimuli
  - Difficulty in arousal
  - Loss of consciousness or coma

TABLE 14-5 Glasgow Coma Scale and Use in Assessment

Criteria	Maximum	Example—0700Hours	Example—0900Hours	Example—1100Hours
Eye opening				
Spontaneous	4			
Response to speech	3	×	×	
Response to pain	2			
None	1			×
Motor response				
Obeys commands	6	×		
Localizes pain	5		×	
Normal flexion (to pain)	4			
Abnormal flexion (decorticate)	3			
Abnormal extension (decerebrate)	2			
None (flaccid)	2			×
Verbal response				
Oriented to time and place	5			
Confused	4	×		
Inappropriate words	3		×	
Incomprehensible	2			
None	1			×
Score	15 (good, normal)	13	11	4

Level of Consciousness

- Vegetative state // Loss of awareness and mental capabilities
  - Result of diffuse brain damage
  - Brainstem function continues.
  - Appearance of a sleep-wake cycle
  - Person unresponsive to external stimuli
- Locked-in syndrome
  - Individual is aware and capable of thinking but is paralyzed and cannot communicate

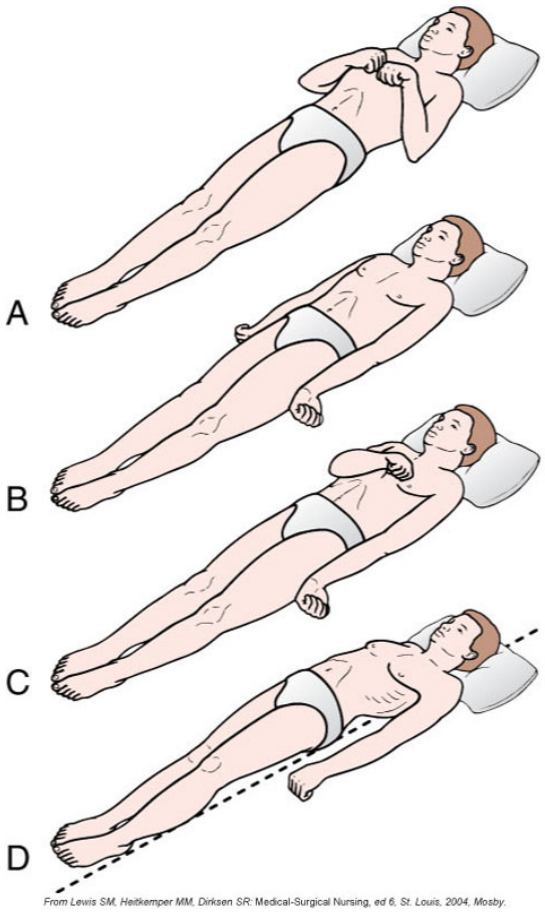
Level of Consciousness

- Criteria for brain death
  - Cessation of brain function // Including function of the cortex and the brainstem // Flat or inactive electroencephalogram (EEG)
  - Absence of brainstem reflexes or responses
  - Absence of spontaneous respirations when ventilator assistance is withdrawn
  - Establishment of the certainty of irreversible brain damage by confirmation of cause of the dysfunction
  - Evaluation twice by different physicians

Motor Dysfunction

- Damage to upper motor neurons
  - Interference with voluntary movements
  - Weakness or paralysis on the contralateral side of the body
- Damage to lower motor neurons
  - Weakness or paralysis on the same side of the body
  - At and below the level of spinal cord damage
- Decorticate and decerebrate posturing
  - Severe brain damage

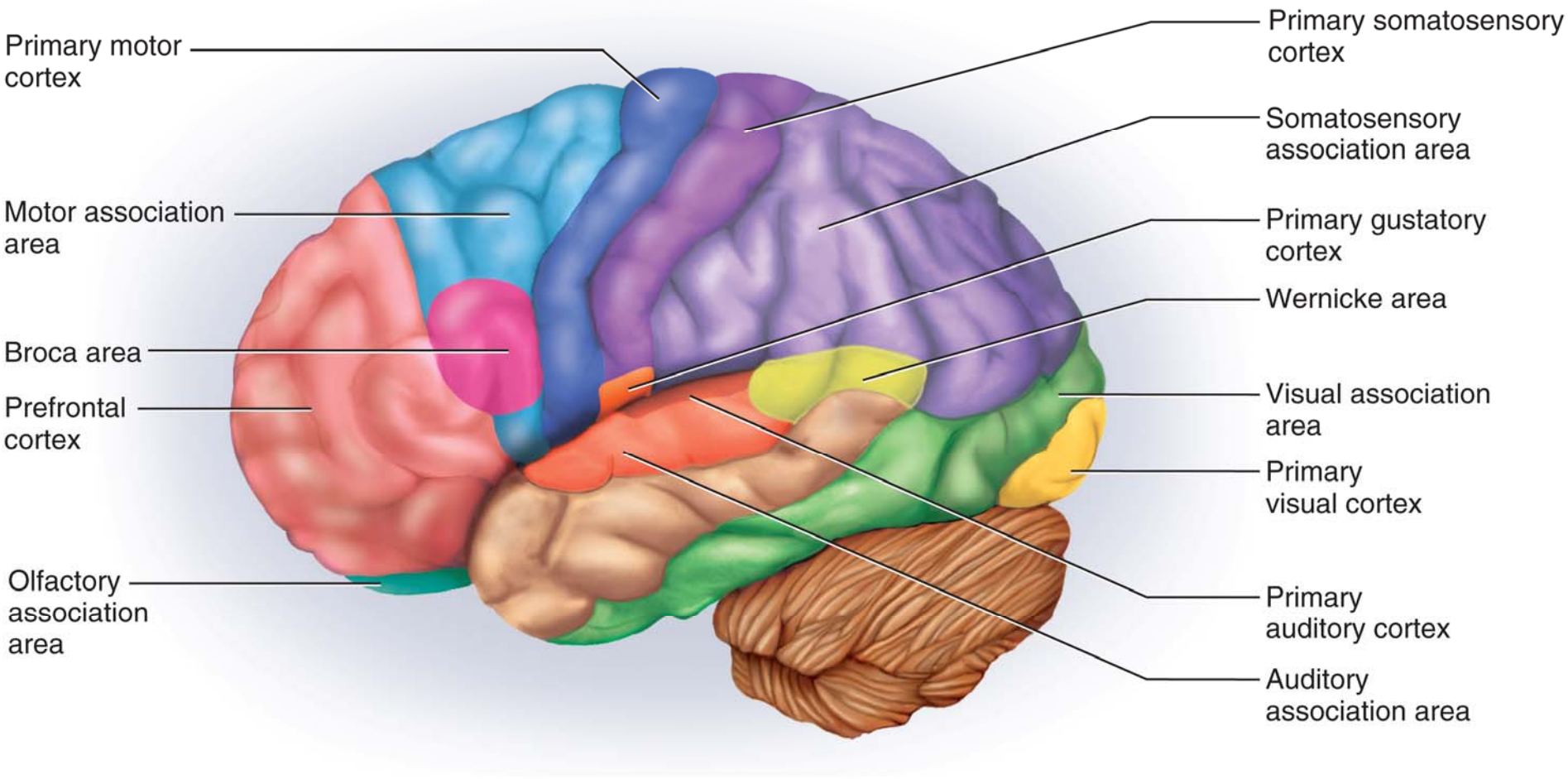
Decorticate and Decerebrate Posturing



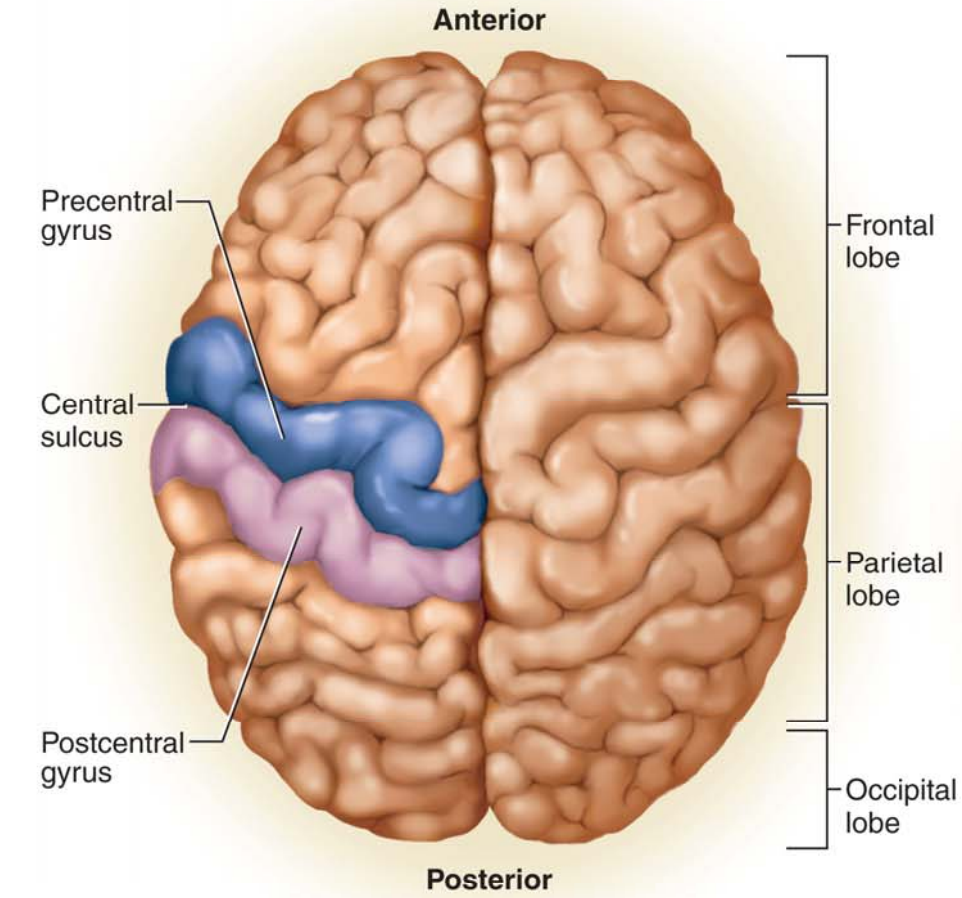
From Lewis SM, Heitkemper MM, Dirksen SR: Medical-Surgical Nursing, ed 6, St. Louis, 2004, Mosby.

Sensory Deficits

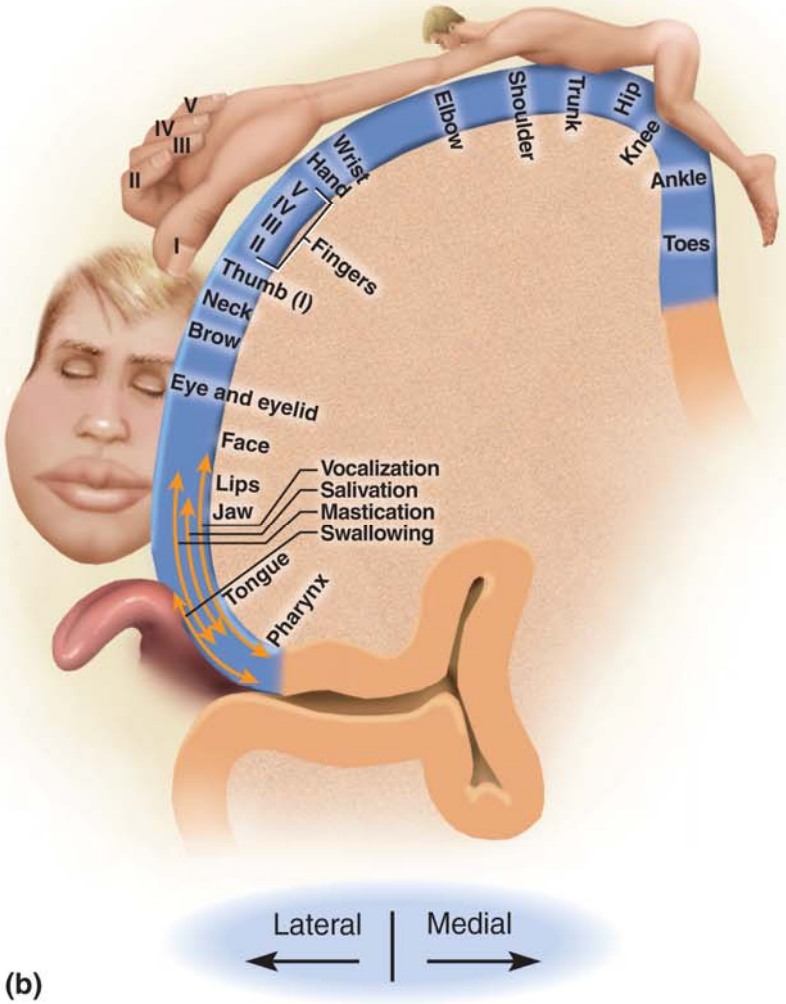
- Somatosensory cortex in the parietal lobe receives and localizes basic sensory input
  - Mapped by dermatomes // Assists in evaluation of spinal core lesions
  - Involves touch, pain, temperature, position
  - Involves special senses of vision, hearing, taste, smell



Primary Motor and Sensory Cortex



(a)



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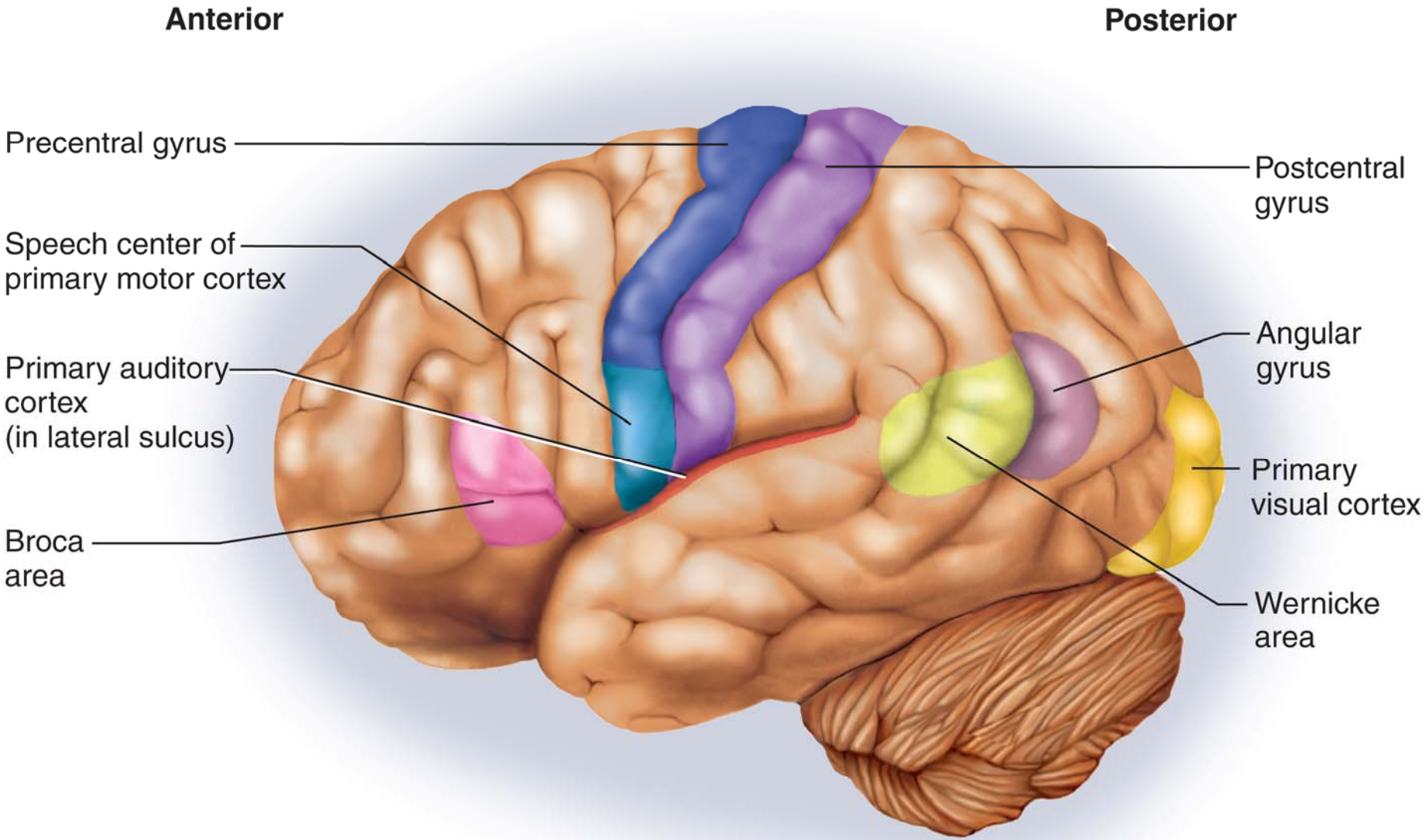
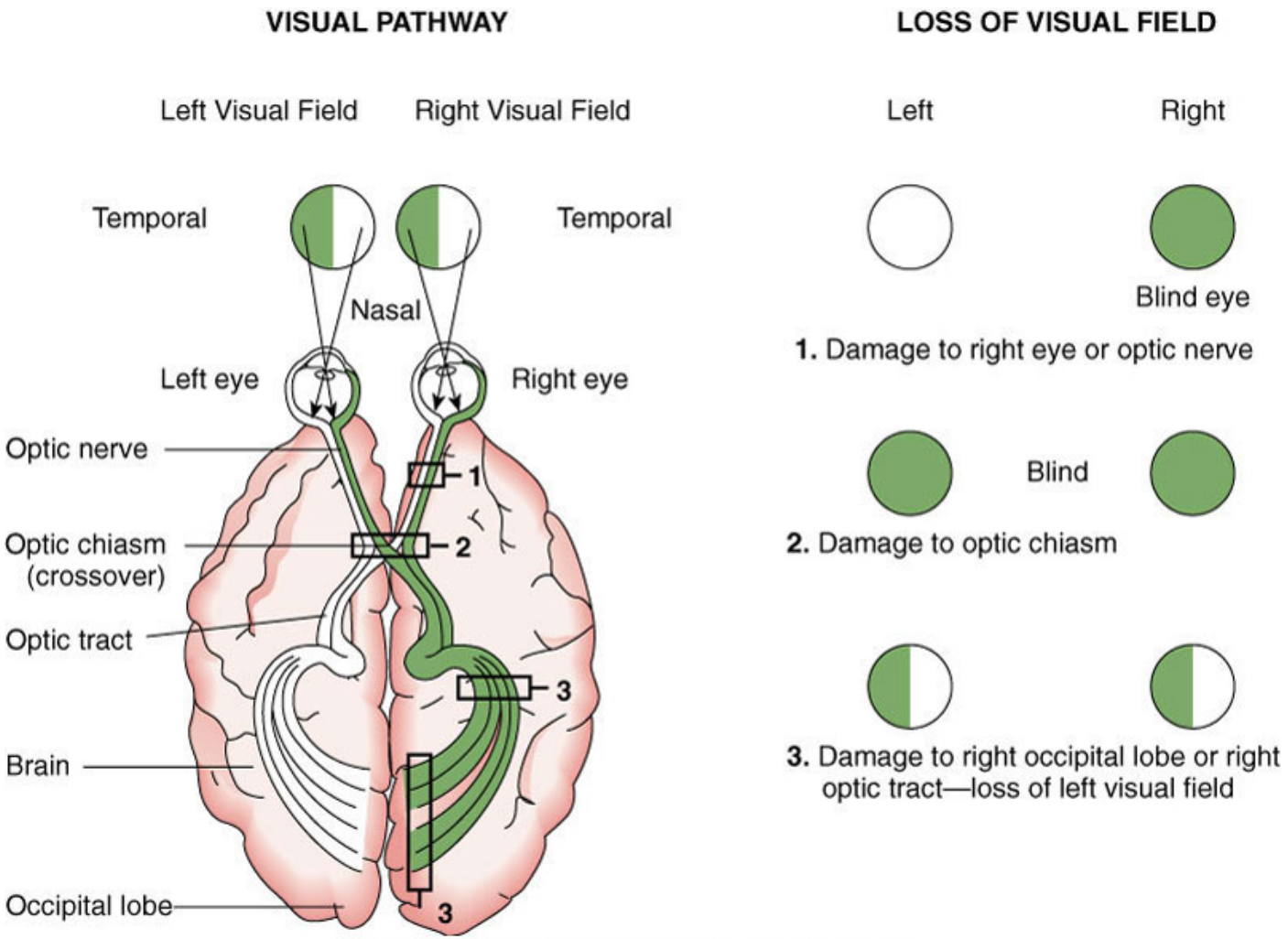


Visual Loss: Hemianopia

- Depends on site of damage in visual pathway
- Optic chiasm damage
  - Vision lost in both eyes if chiasm is totally destroyed
  - Partial loss // Depends on particular fibers damaged
- Optic tract or occipital lobe damage
  - Loss of the visual field on side opposite to that of the damage

The Visual Pathway



Language Disorders

- Aphasia
  - Inability to comprehend or express language
  - Receptive - damage to Wernicke's area
  - Expressive - damage to Broca's area
  - Mixed, global - damage to both areas or to the fibers and tracts between them
- Dysarthria // Motor dysfunction affecting muscles used in speech

(Next slide)

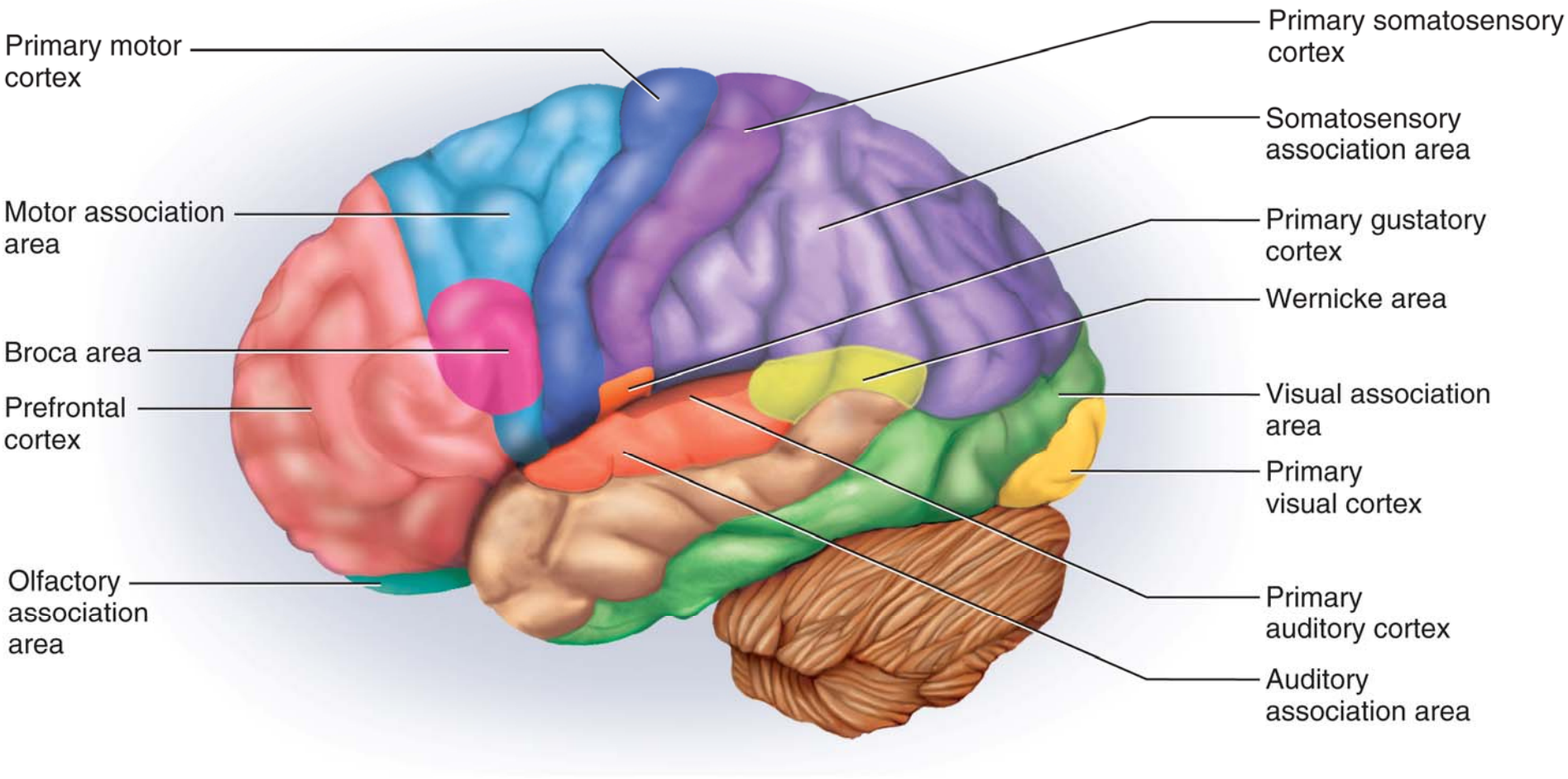


TABLE 14-6 Aphasia

Type	Site of Damage	Effect
Expressive (motor)	Broca's area Left frontal lobe	Cannot speak or write fluently or appropriately
Receptive (sensory)	Wernicke's area Left temporal lobe, prefrontal	Unable to understand written or spoken language
Global	Broca's and Wernicke's areas and connecting fibers	Cannot express self or comprehend others' language

- Expressive, or motor aphasia
  - Impaired ability to speak or write fluently or appropriately
  - Occurs when Broca's area in dominant frontal lobe is damaged
- Receptive or sensory aphasia
  - Inability to read or understand the spoken word
  - Source—inability to process information in the brain
  - Result of damage to Wernicke's area in the left temporal lobe
  - Usually also affects expression

- Global aphasia
  - Combination of expressive and receptive aphasia
  - Major brain damage, including Broca's area, Wernicke's area, and many communicating fibers
- Fluent aphasia // Pace of speech relatively normal
  - Includes made-up words (word salad)
  - Associated with damage to Wernicke's area
- Nonfluent aphasia // Slow and labored, with short phrases
  - Associated with damage to Broca's area

Language Disorders

- Dysarthria // Words cannot be articulated clearly
  - Motor dysfunction—usually results from cranial nerve damage or muscle impairment
- Agraphia // Impaired writing ability
- Alexia // Impaired reading ability
- Agnosia // Loss of recognition or association

Seizures

- Seizures or convulsions
  - Caused by spontaneous, excessive discharge of neurons in the brain
  - Causes // Inflammation – Hypoxia - Bleeding in the brain
  - Focal // Related to the particular site of the irritation
  - May become generalized
  - Often manifested by involuntary repetitive movements or abnormal sensations (aura)

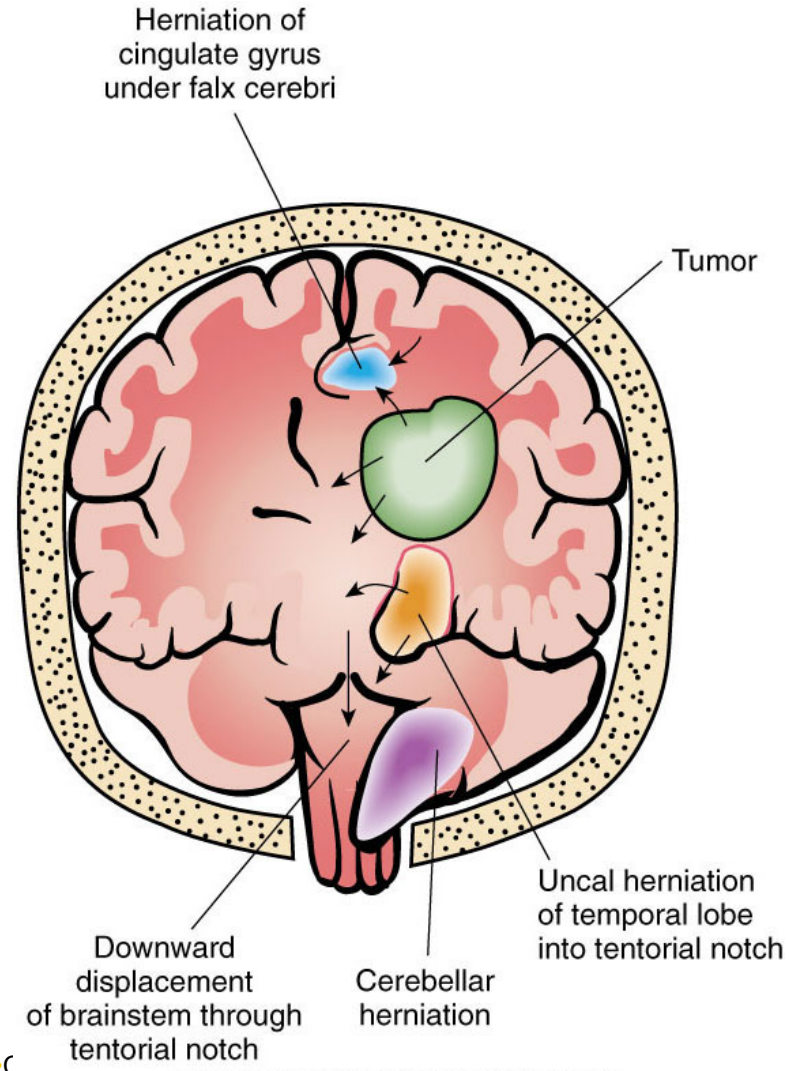
Seizures

- Generalized // Absence seizures (petit mal)
  - Tonic-clonic
  - Myoclonic
- Partial // Simple partial
  - Complex partial (psychomotor)
- Continuous seizures (status epilepticus)
  - Increased metabolism of glucose and oxygen
  - May be life-threatening

Increased Intracranial Pressure

- Brain is encased in rigid, nonexpendable skull.
- Fluids, blood, and CSF are not compressible.
- Increase in fluid or additional mass causes increase in pressure in the brain
  - Ischemia and eventual infarction of brain tissue
- Increased ICP is common in many neurological problems.
  - Brain hemorrhage, trauma, cerebral edema, infection, tumors, abnormal circulation of CSF

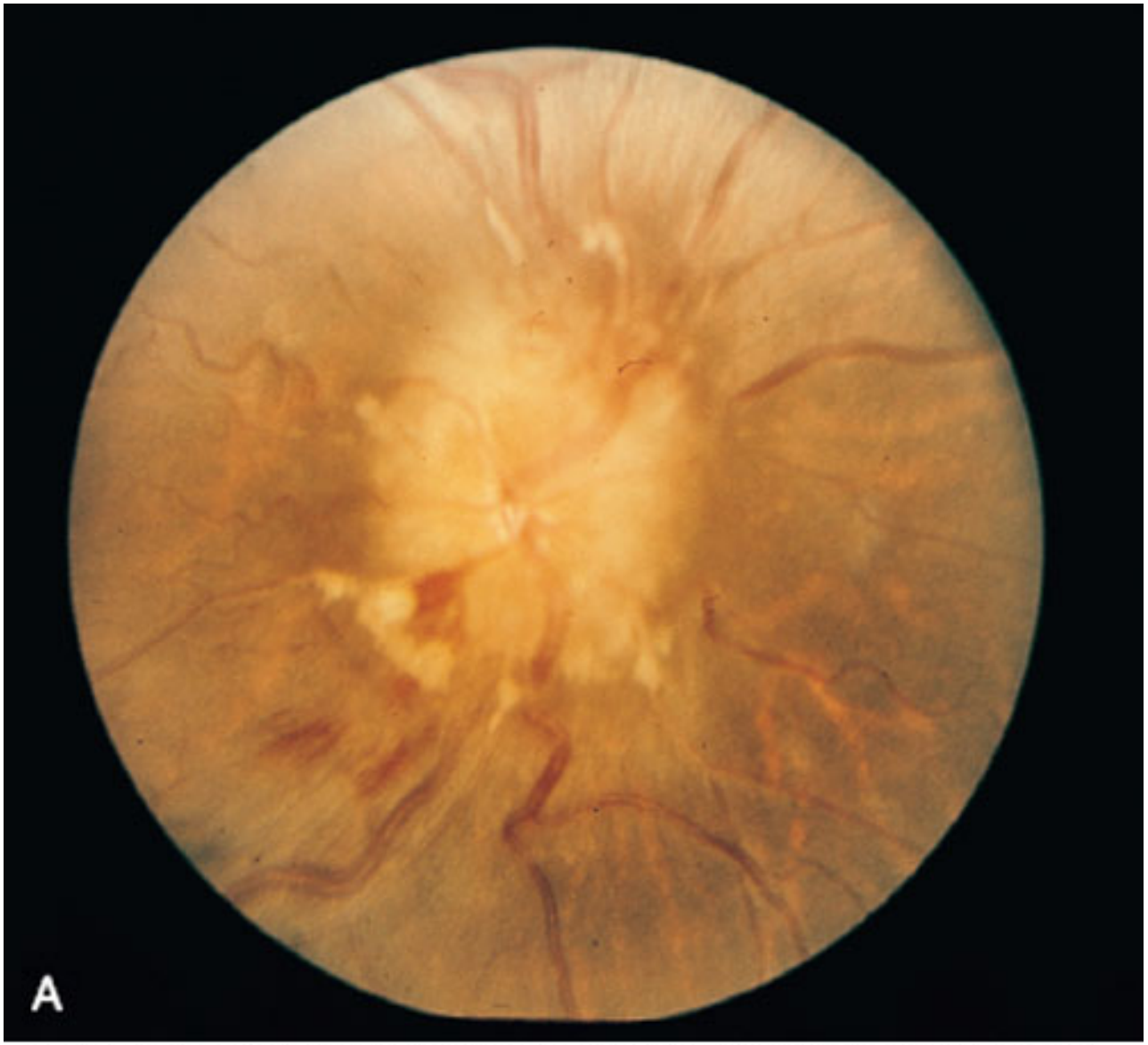
Increased Intracranial Pressure and Possible Herniations



Increased Intracranial Pressure

- Early signs — if cause is not removed
  - Decreasing level of consciousness or decreased responsiveness (lethargy)
  - Decreased pupillary responses
  - Severe headache // From stretching of dura and walls of large blood vessels
  - Vomiting // Often projectile, not associated with food intake // Result of pressure stimulating the emetic center in the medulla
  - Papilledema // Caused by increased ICP and swelling of the optic disc (see next slide)

Papilledema



A, Courtesy John W. Payne, MD, The Wilmer Ophthalmological Institute, The Johns Hopkins University and Hospital, Baltimore, MD, from Seidel HM, Ball JW, Dains JE, et al: *Mosby's Guide to Physical Examination,* ed 5, St. Louis, 2003, Mosby; B, From Cotran RS, Kumar V, Collins T: *Robbins Pathologic Basis of Disease,* ed 6, Philadelphia, 1999, Saunders.

TABLE 14-7 Effects of Increased Intracranial Pressure

General Signs	Rationale
Decreasing level of consciousness	Pressure on RAS (brain stem) or cerebral cortex
Headache	Stretching or distortion of meninges or walls of large blood vessels
Vomiting	Pressure on emetic center in medulla
Vital Signs	
Increasing blood pressure with increasing pulse pressure	Cushing's reflex; response to cerebral ischemia causes systemic vasoconstriction
Slow heart rate	Response to increasing blood pressure
Signs Affecting Vision	
Papilledema	Increased pressure of CSF causes swelling around the optic disc
Pupil, fixed and dilated	Pressure on cranial nerve III (oculomotor)

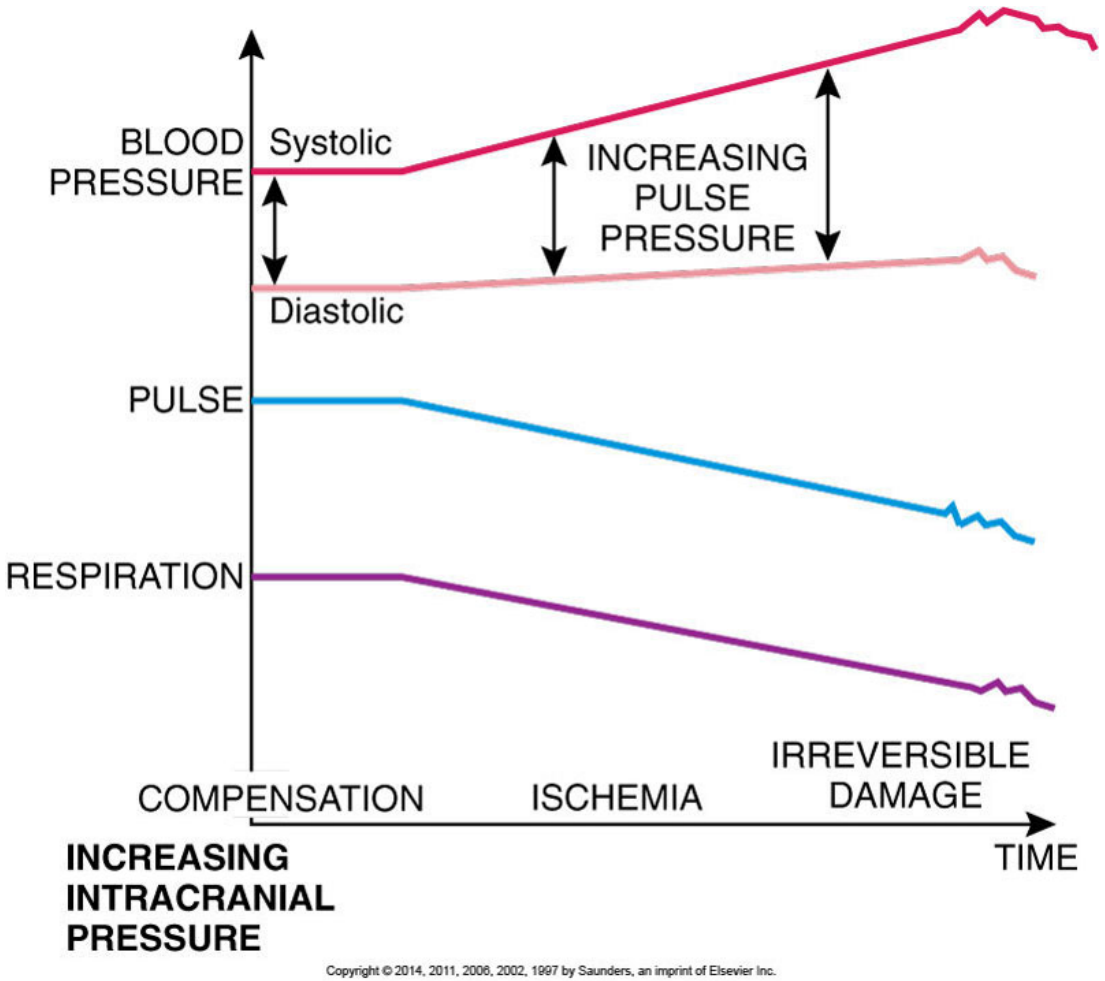
CSF, cerebrospinal fluid; RAS, reticular activating system.

Increased Intracranial Pressure

- Vital signs
  - Development of cerebral ischemia // Vasomotor centers respond in attempt to increase arterial blood supply to brain (Cushing reflex)
  - Systemic vasoconstriction // Increase of systemic blood pressure—more blood to brain to relieve ischemia
  - Baroreceptor response // In carotid arteries // Increased blood pressure by slowing heart rate
  - See next slide

- Chemoreceptor response
  - Respond to low carbon dioxide levels
  - Reduction of respiratory rate
- Improved cerebral circulation
  - Relieves ischemia for short time
  - Increasing ICP causes ischemia to recur; cycle will repeat
- ICP continues to rise, blood pressures rises
  - Increased pulse pressure is significant in people with ICP.

Vital Signs with Increased Intracranial Pressure



Visual Signs of ICP

- Pressure on oculomotor nerve (cranial nerve III) affects size and response of pupils
- Pupil ipsilateral to lesion becomes fixed and dilated
- As pressure increases, shift of contents across the midline → both pupils become fixed and dilated.
- Ptosis (droopy eyelid) may occur. // Effect of pressure on CN III (oculomotor)

Herniation

- Transtentorial herniation
  - Cerebral hemispheres, diencephalon, midbrain are displaced downward
  - Resulting pressure affects flow of blood and CSF, RAS, and respiration
- Uncal herniation
  - Uncus of the temporal lobe is displaced downward
  - Creates pressure on CN III, posterior cerebral artery, and RAS

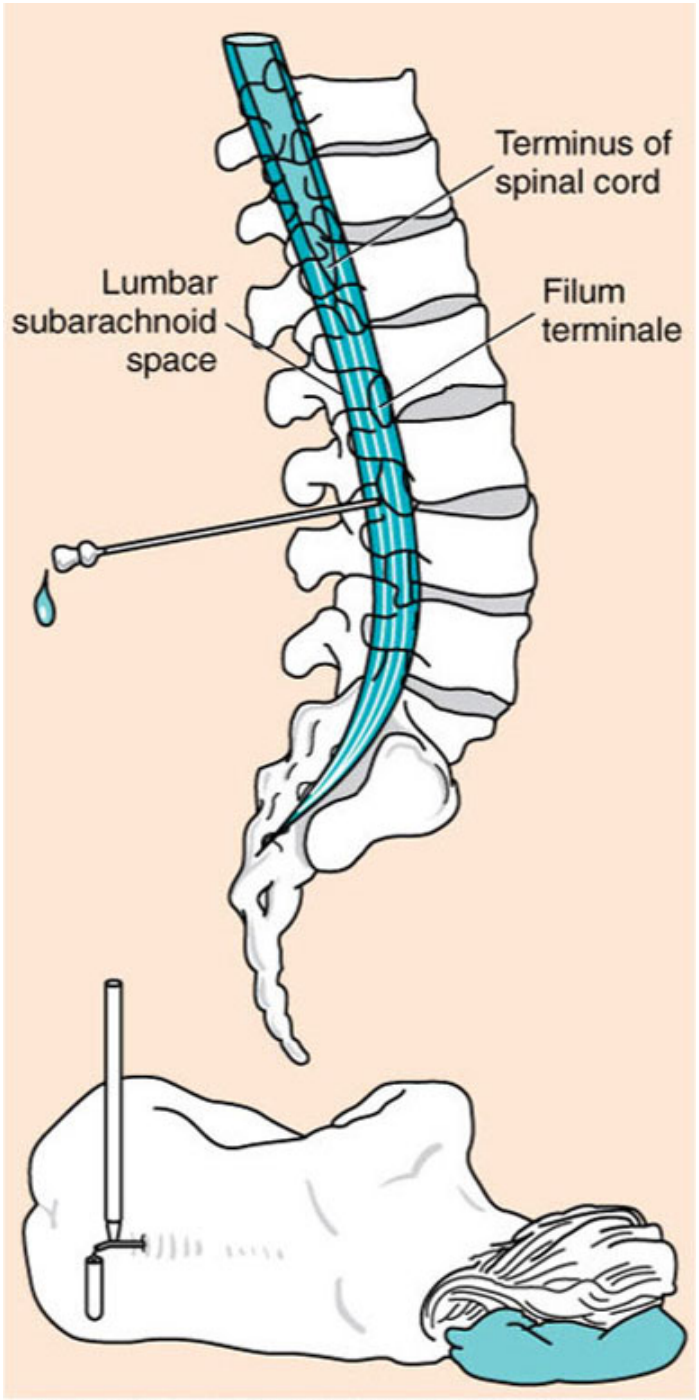
Herniation (Cont.)

- Infratentorial (cerebellar, or tonsillar) herniation
  - Cerebellar tonsils are pushed downward through the foramen magnum.
  - Compresses brainstem and vital centers → infarction
  - Causes death

Diagnostic Tests

- Computed tomography (CT) scans
- Magnetic resonance imaging (MRI)
- Cerebral angiography
- Doppler ultrasound
- Electroencephalography
- Radionuclide may be used to track perfusion in CNS
- Lumbar puncture used to check pressure and analyze CSF

Lumbar Puncture



From Mahon CR, Manuselis G: Textbook of Diagnostic Microbiology, ed 2. Philadelphia, 2000, Saunders.

Specific Acute Neurologic Problems

Brain Tumors

- Space-occupying lesions that cause increased ICP
- Benign and malignant tumors can be life-threatening. // Unless accessible and removable
- Gliomas (glial cell cancer) form the largest category of primary malignant tumors. // Classified according to cell derivation and location of the tumor

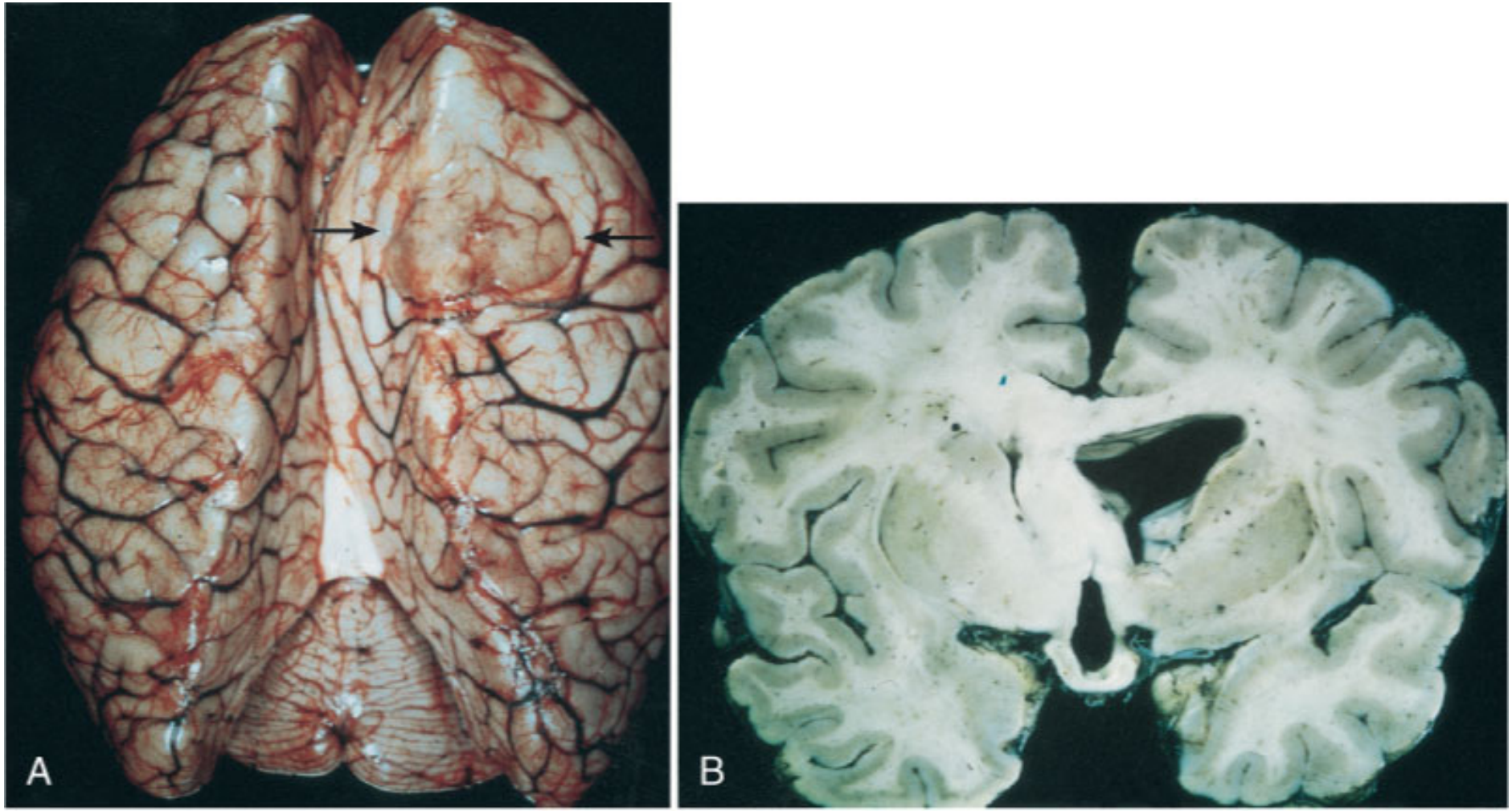
Brain Tumors

- Tumors in the meninges or pituitary gland cause similar neurological effects.
- Primary malignant tumors rarely metastasize outside the CNS.
- Secondary brain tumors
  - Metastasize from breast or lung tumors
  - Cause effects similar to those of primary brain tumors

Brain Tumors

- Pathophysiology
  - Primary malignant brain tumors // Usually no well-defined margins
    - Invasive and have irregular projections into adjacent tissue // Difficult to remove completely
  - Usually inflammation around the tumor
- Occurrence
  - Brainstem and cerebellar tumors common in young children
  - Adults - occur more frequently in the cerebral hemispheres

Brain Tumors



From Kumar V, Abbas AK, Fausto M: Robbins and Cotran Pathologic Basis of Disease, *ed 7, Philadelphia, 2005, Saunders.*

Brain Tumors

- Signs and symptoms
  - Site of tumor determines focal signs
  - Seizures often first sign
  - Headaches (increased ICP), vomiting, lethargy, irritability, personality and behavioral changes, possible unilateral facial paralysis or visual problems
  - Do not cause systemic signs of malignancy // Will cause death before they cause general effects
- Treatment—may cause damage to normal CNS tissue
  - Surgery if tumor is accessible
  - Chemotherapy and radiation (many are radioresistant)

Vascular Disorders

- Interference with blood supply // Local damage and manifestations depend on cerebral artery involved
- Hemorrhage // Increased ICP will cause local ischemia and generalized symptoms.
- Global cerebral ischemia // Impaired perfusion of entire brain
  - Loss of function and generalized cerebral edema
  - Brain death if not reversed quickly

Transient Ischemic Attacks (TIAs)

- May occur singly or in a series
- Result from temporary localized reduction of blood flow in the brain
  - Partial occlusion of an artery
  - Atherosclerosis
  - Small embolus
  - Vascular spasm
  - Local loss of autoregulation

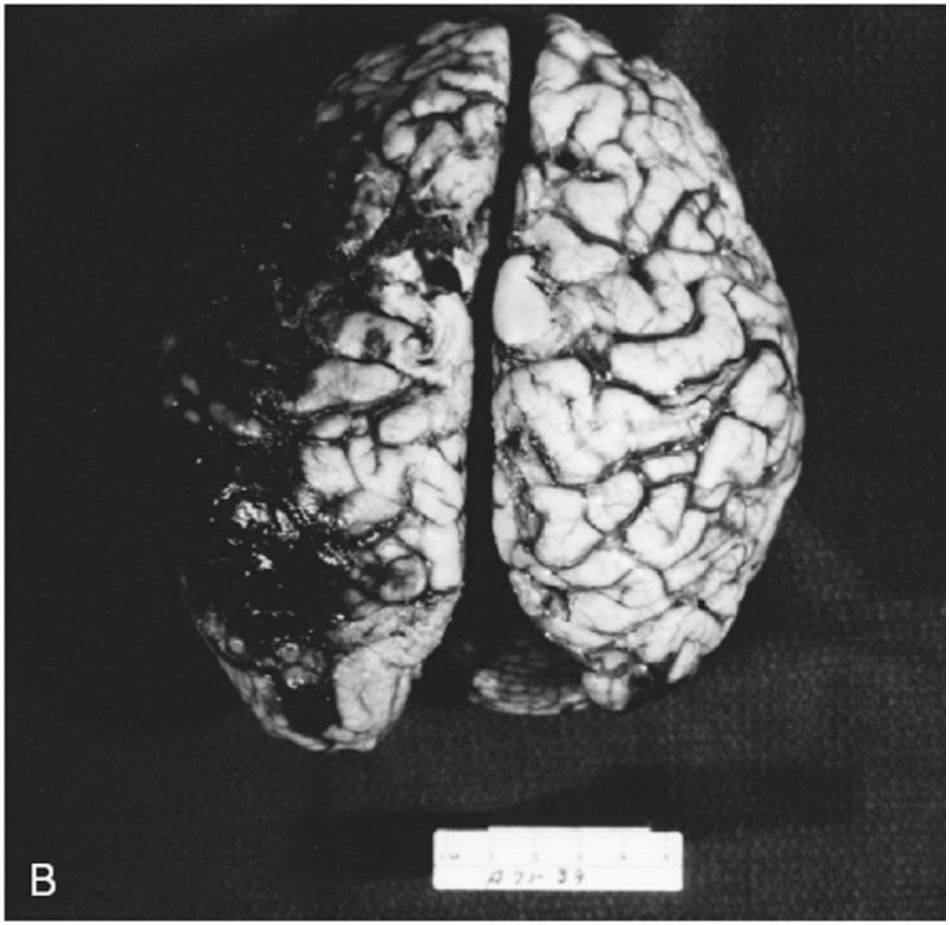
TIAs Signs and Symptoms

- Difficult to diagnose after the attack
- Directly related to location of ischemia
- Intermittent short episodes of impaired function // e.g., muscle weakness in arm or leg
- Visual disturbances
- Numbness and paresthesia in face
- Transient aphasia or confusion may develop. // Repeated attacks may be a warning sign for obstruction related to atherosclerosis.

Cerebrovascular Accidents (CVAs)

- A CVA (stroke) is an infarction of brain tissue that results from lack of blood.
  - Occlusion of a cerebral blood vessel
  - Rupture of cerebral vessel
- 5 minutes of ischemia causes irreversible nerve cell damage.
  - Central area of necrosis develops
  - All function lost
  - Surrounded by an area of inflammation. this zone will regain function following healing.

Acute Hemorrhagic Infarction



Courtesy of R.W. Shaw, MD, North York General Hospital, Toronto, Ontario, Canada.

Types of CVAs

- Occlusion of an artery by an atheroma // Often develop in large arteries
- Sudden obstruction caused by an embolus // Lodging in a cerebral artery
- Intracerebral hemorrhage // Caused by rupture of a cerebral artery in patient with severe hypertension
  - Effects are evident in both hemispheres.
  - Complicated by secondary effects of bleeding

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

MRI Can Determine Cause of the Stoke

Risk Factors of CVAs

- Diabetes, hypertension, systemic lupus erythematosus, atherosclerosis
- History of TIAs, increasing age, obstructive sleep apnea, heart disease, smoking, sedentary lifestyle
- Combination of oral contraceptives and cigarette smoking
- Congenital malformation of blood vessels
- Increasing age

Signs and Symptoms of CVAs

- Depend on the location of obstruction
- Size of artery involved // Presence of collateral circulation may diminish size of affected area
- Lack of voluntary movement or sensation on opposite side of the body.
- Initially flaccid paralysis
- Spastic paralysis develops weeks later
- NIH Stroke Scale (NIHSS) // Developed by the National Institutes of Health (NIH) // Designed to assist with rapid diagnosis in an emergency situation

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!

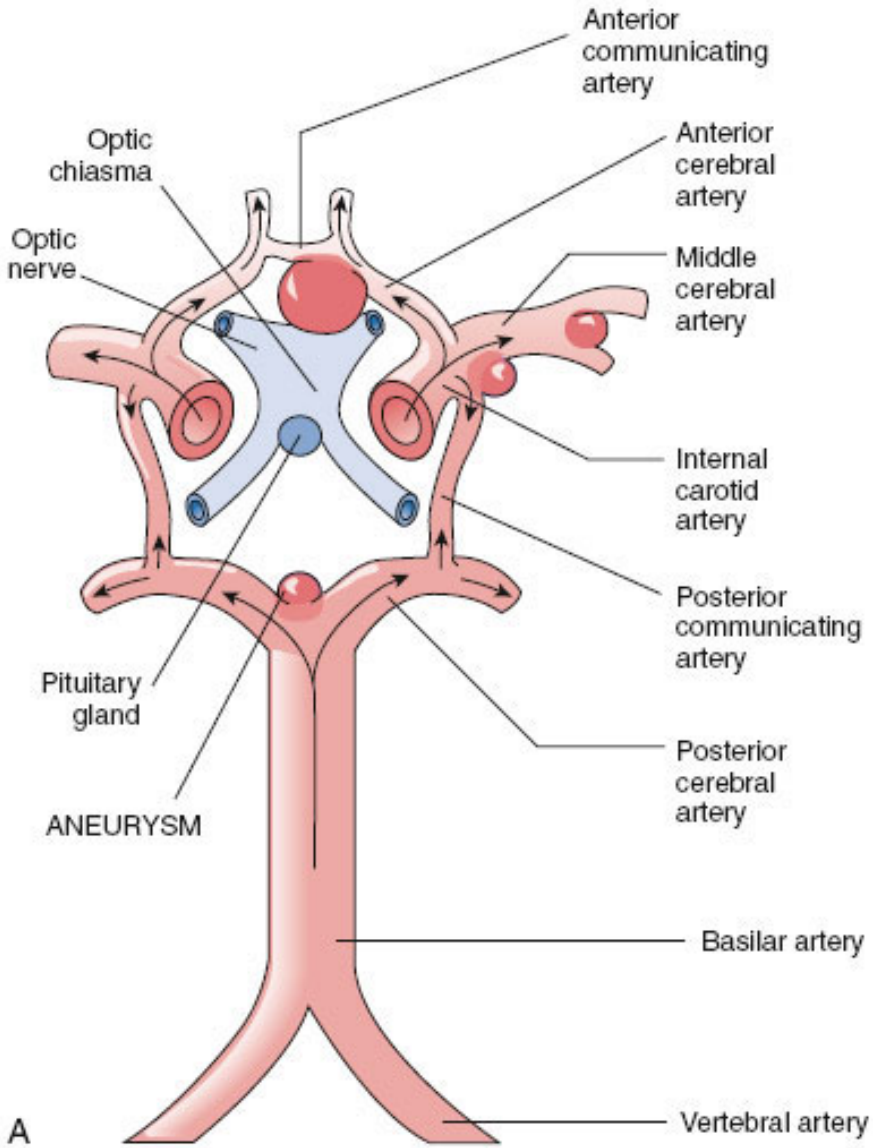
Treatment of CVAs

- Clot-busting agents
- Surgical intervention
- Glucocorticoids
- Supportive treatment
- Occupational and physical therapists; speech-language pathologists
- Treat underlying problem to prevent recurrences.
- Rehabilitation begins immediately.

Cerebral Aneurysms

- Localized weakness in the wall of an artery
- Cerebral aneurysms frequently multiple. // Usually at the points of bifurcation on the Circle of Willis // Berry aneurysms
  - Often aggravated by hypertension
  - Initially small and asymptomatic
  - Slow bleed causes vascular type headache.
  - Rupture leads to sudden fatal increase in ICP and death.

Circle of Willis



From Kumar V, Abbas AK, Fausto M: *Robbins and Cotran Pathologic Basis of Disease*, ed 7, Philadelphia, 2005, Saunders.

Signs and Symptoms of Cerebral Aneurysms

- Loss of visual field or visual disturbances
- Headache and photophobia
- Intermittent periods of dysfunction
- Nuchal rigidity caused by meningeal irritation
- Vomiting, seizures, loss of consciousness in case of massive rupture; rapidly followed by death

- Treatment // Surgical treatment before rupture // Antihypertensive drugs

Infections

- Different age groups are susceptible to infection by different causative organisms. // May be secondary to other infections

- Children and young adults

  - *Neisseria meningitidis* or meningococci

  - Classic meningitis pathogen

  - Frequently carried in the nasopharynx of asymptomatic carriers

  - Spread by respiratory droplets

  - Occurs more frequently in late winter and early spring

Infections

- Neonates // *Escherichia coli* most common causative organism
  - Usually in conjunction with a neural tube defect, premature rupture of the amniotic membranes, difficult delivery
- Young children // Most often caused by *Haemophilus influenzae*
    - More often in the autumn or winter
- Older adults // *Streptococcus pneumoniae* - major cause

Signs and Symptoms of Infections

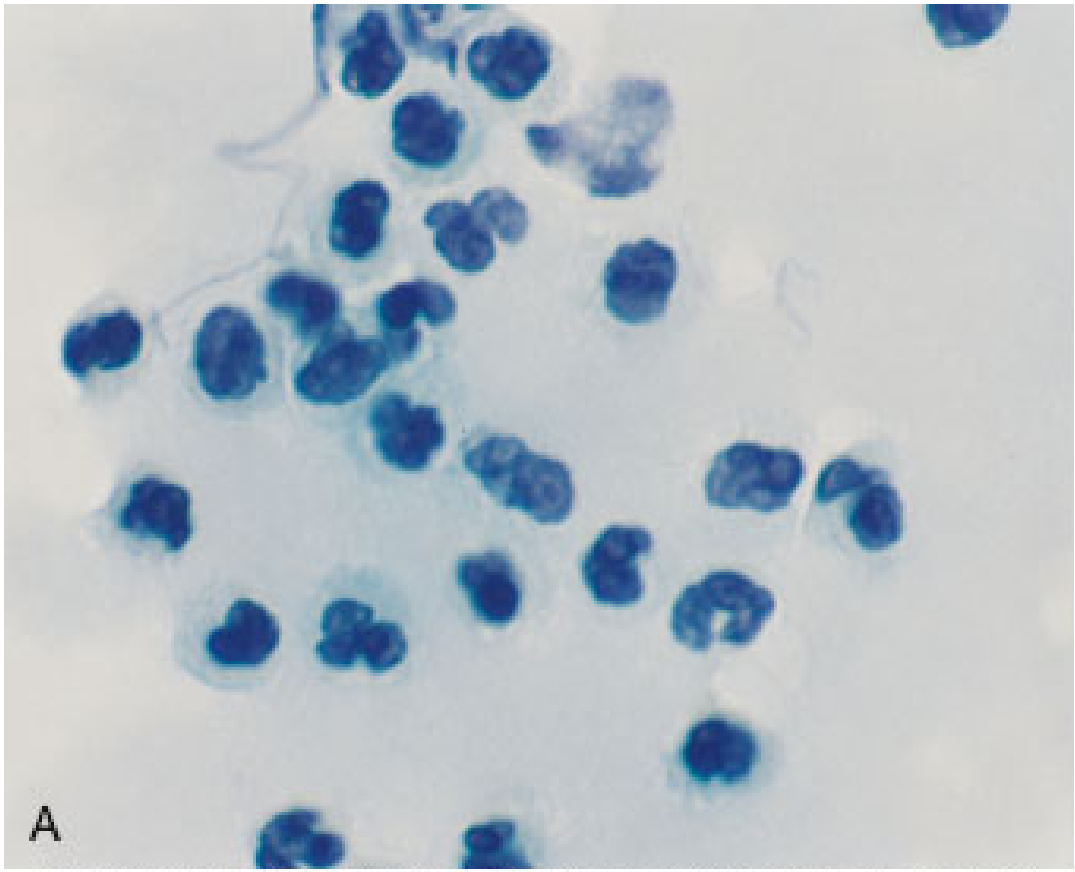
- Sudden onset is common with severe headache
- Photophobia
- Nuchal rigidity
- Kernig sign
- Brudzinski sign
- Vomiting, irritability, lethargy, fever, chills with leukocytosis
- Progression to stupor or seizures

Infections

- Diagnostic tests
  - Examination of CSF (obtained by lumbar puncture)
  - Identification of causative organism
- Treatment
  - Aggressive antimicrobial therapy
  - Specific treatment measures for ICP and seizures
  - Glucocorticoids // Reduction of cerebral inflammation and edema
  - Vaccines are available for some types of meningitis.

Meningitis / Infection of the CT Surrounding CNS

Slide preparation of CSF showing many neutrophils with bacterial meningitis

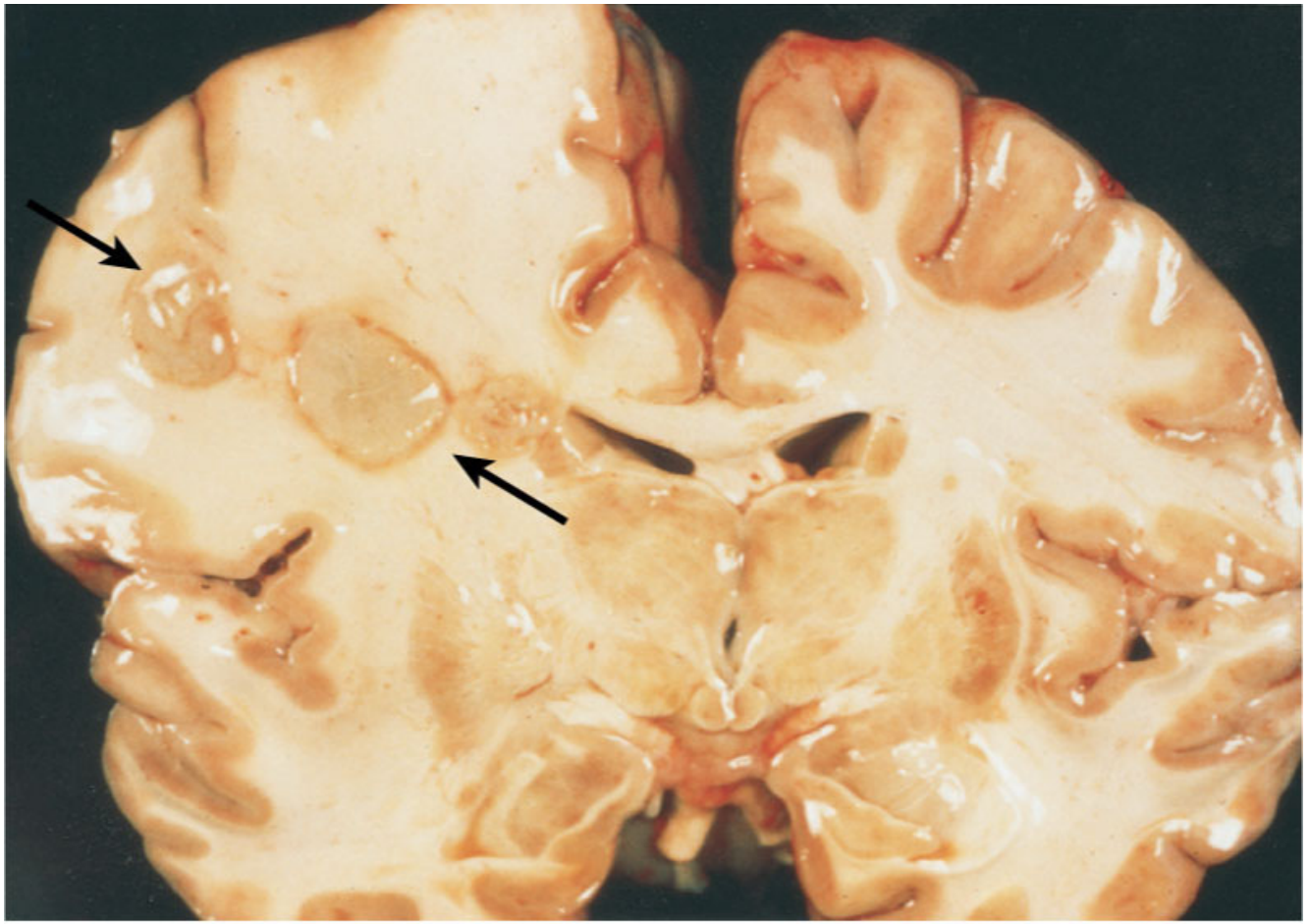


A from Stevens ML: *Fundamentals of Clinical Hematology*, Philadelphia, 1997, Saunders; **B** and **C** from Mahon CR, Manuselis G: *Textbook of Diagnostic Microbiology*, ed 2, Philadelphia, 2000, Saunders; **D** from Cooke RA, Stewart B: *Colour Atlas of Anatomical Pathology*, ed 3, Sydney, 2004

Brain Abscess

- Localized infection // Frequently in frontal or temporal lobes
- Usually necrosis of brain tissue and surrounding area of edema
- May spread from organisms in ear, throat, lung, sinuses
- May result from septic emboli, acute bacterial endocarditis, site of injury, or surgery
- Surgical drainage and antimicrobial therapy

Brain: Frontal Abscess



From Kumar V, Abbas AK, Fausto M: Robbins and Cotran Pathologic Basis of Disease, *ed 7, Philadelphia, 2005, Saunders.*

Encephalitis

- Infection of the parenchymal or connective tissue in the brain and spinal cord (i.e. CNS)
  - Necrosis and inflammation develop in brain tissue. // Result in some permanent damage
- Infection may include meninges.
- Usually of viral origin // May be caused by other organisms
- Early signs // Severe headache, stiff neck, lethargy, vomiting, seizures, fever

Types of Encephalitis

- Western equine encephalitis
  - Arboviral infection spread by mosquitoes
  - More frequent in summer months
  - Common in young children
- St. Louis Encephalitis // Affects older persons more seriously than younger individuals
- West Nile fever // Caused by a flavivirus - Spread by mosquitoes

Types of Encephalitis

- Neuroborreliosis (Lyme disease)
  - Caused by *Borrelia burgdorferi* // Transmitted by ticks
  - Typical bull's-eye lesion—sore throat, dry cough, fever, headache, cardiac arrhythmias, neurological abnormalities
  - Antimicrobial therapy
- Herpes simplex encephalitis // Occurs occasionally
  - Spread from herpes simplex I
  - Extensive necrosis and hemorrhage in the brain

Other Infections

- Rabies
  - Viral—transmitted by: // Bite of rabid animal // Transplantation of contaminated tissues
  - Virus travels along peripheral nerves to CNS
  - Headache and fever, nervous hyperirritability, sensitivity to touch, seizures
  - Virus also travels to salivary glands // Difficulty swallowing // Fear of fluids // Foaming effect
  - Respiratory failure, death

Other Infections

- Tetanus
  - Caused by *Clostridium tetani // produce spores that* can survive in soil (years). // also in honey
  - Wound
  - Exotoxin enters nervous system
    - Tonic muscle spasms // Jaw stiffness
    - Difficulty swallowing // Stiff neck
    - Headache and skeletal muscle spasm
    - Respiratory failure

Poliomyelitis Infections (infantile paralysis)

- Polio virus
- Immunization available
- Endemic in West and Central Africa
- Highly contagious // Direct contact via Oral droplets
- Attacks motor neurons of spinal cord and medulla
- Fever, headache, vomiting, stiff neck, pain, flaccid paralysis

Infection-Related Syndromes
Post-Polio Syndrome

- Occurs 10 to 40 years after recovery from original infection
- Progressive and debilitating fatigue, weakness, pain, muscle atrophy
- The more severe the original infection, the more severe are the effects of PPS.

Infection-Related Syndromes
Herpes Zoster (Shingles)

- Caused by varicella-zoster virus in adults // Can occur years after primary infection of varicella (chickenpox)
- Usually affects cranial nerve or one dermatome
- Pain, paresthesia, vesicular rash // If antiviral drugs started within 48 hours of onset, pain is significantly reduced
- Lesions and pain persist for several weeks. // Post-herpetic pain may persist for months to years in some cases.
- Vaccine available for those 60 years or older

Infection-Related Syndromes
Reye's Syndrome

- Cause not fully determined
- Linked to *viral infection in children treated with aspirin*
- Pathological changes in brain and liver
- Brain // Function severely impaired by cerebral edema
- Liver // Enlarged, fatty changes develop in tissue // Can result in acute failure
- Manifestations vary in severity.
- No immediate cure

Infection-Related Syndromes

- Guillain-Barre syndrome
  - Postinfection polyneuritis, acute idiopathic polyneuropathy, acute infectious polyradiculoneuritis
  - Inflammatory condition of the PNS
  - Exact cause unknown
  - Local inflammation with accumulated lymphocytes, demyelination, axon destruction
  - Changes cause impaired nerve conduction.

Infection-Related Syndromes
Guilain-Barre Syndrome

- Critical period develops // Ascending paralysis involves diaphragm and respiratory muscles
- Progressive muscle weakness, lack of reflex response, ascending flaccid paralysis, pain, general muscle aching
  - Paralysis may move upward—vision and speech may be impaired.
  - Process may occur rapidly over a few hours or several days.
  - Life-threatening situation may develop.
- Treatment primarily supportive

Head Injuries

- May involve skull fractures
- Hemorrhage and edema
- Direct injury to brain tissue
- Injury may be mild. // Bruising of the tissue
- Can be severe and life-threatening
  - Destruction of brain tissue
  - Massive swelling of the brain

Types of Head Injuries

- Concussion (minimal brain trauma)
  - Reversible interference with brain function // Causes sudden excessive movement of the brain
  - Result of mild blow to the head or whiplash-type injury
  - Amnesia and headaches may follow. // Recovery usually within 24 hours, without permanent damage
- Contusion // Bruising of brain tissue, rupture of small blood vessels, and edema
  - Blunt blow to the head
  - Possible residual damage

Types of Head Injuries

- Closed head injury
  - Skull is not fractured in injury.
  - Brain tissue is injured and blood vessels may be ruptured.
  - Extensive damage may occur when head is rotated.
- Open head injuries // Involve fractures or penetration of the brain

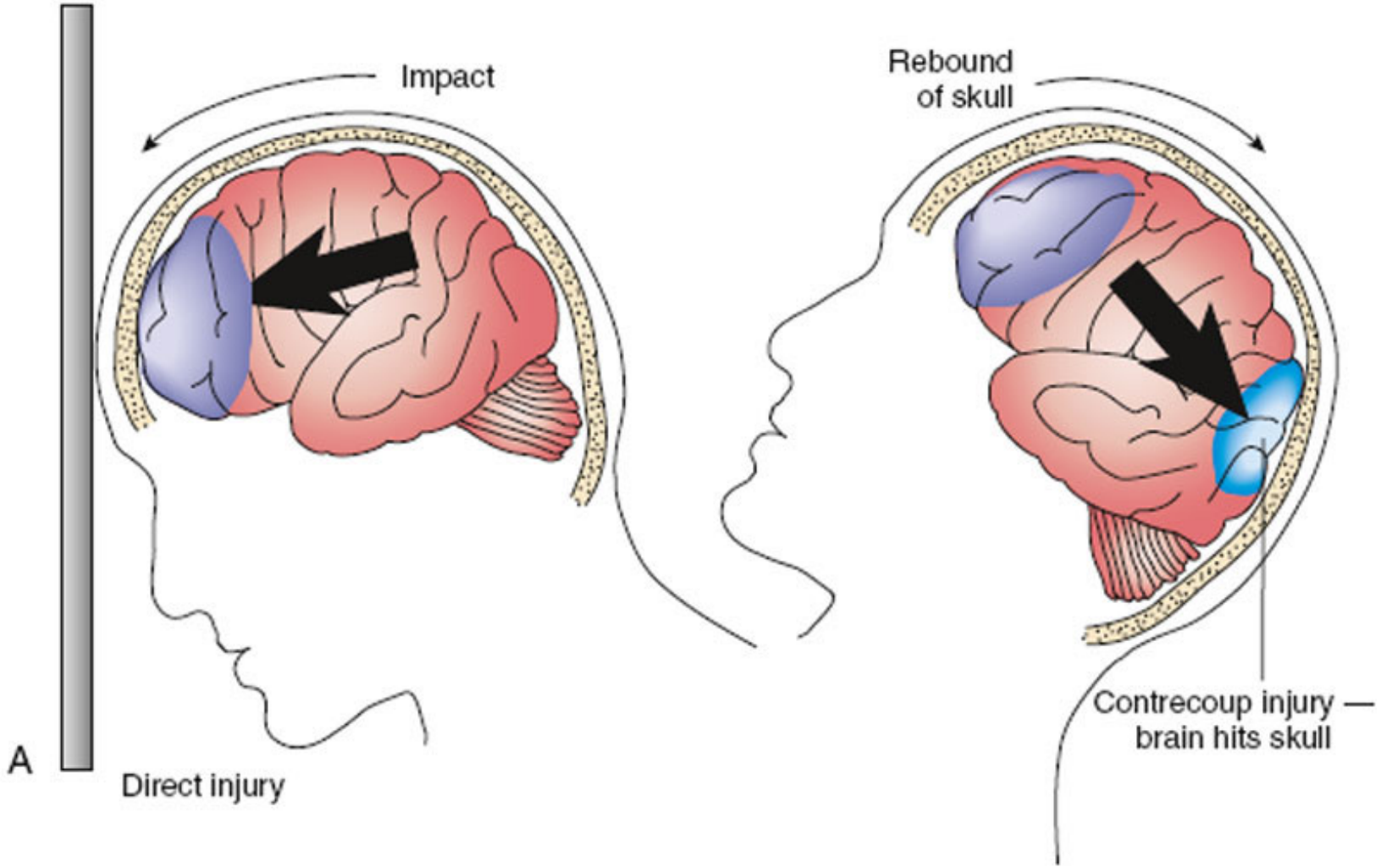
Types of Head Injuries

- Depressed skull fractures
  - Involve displacement of a piece of bone below the level of the skull
    - Compression of brain tissue
    - Blood supply to area often impaired // pressure to brain

Types of Head Injuries

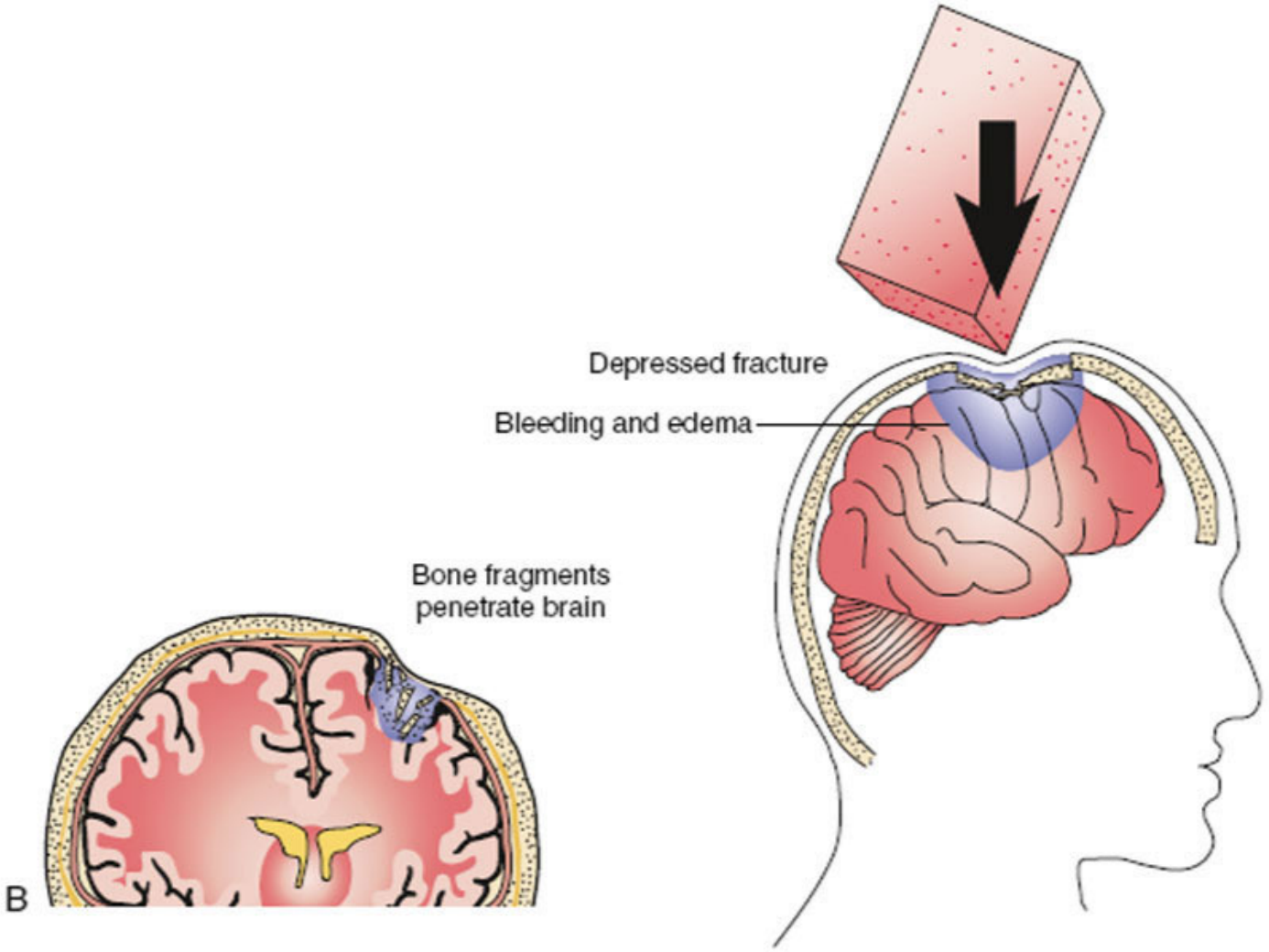
- Basilar fractures // Occur at the base of the skull
  - Leakage of CSF through ears or nose is possible
  - May occur when forehead hits windshield
- Contrecoup Injury // Area of the brain contralateral to the site of direct damage is injured - As brain bounces off the skull
  - May be secondary to acceleration or deceleration injuries

Closed Head Injury



Closed Injury — Direct and Contrecoup Injury

Open Head Injury

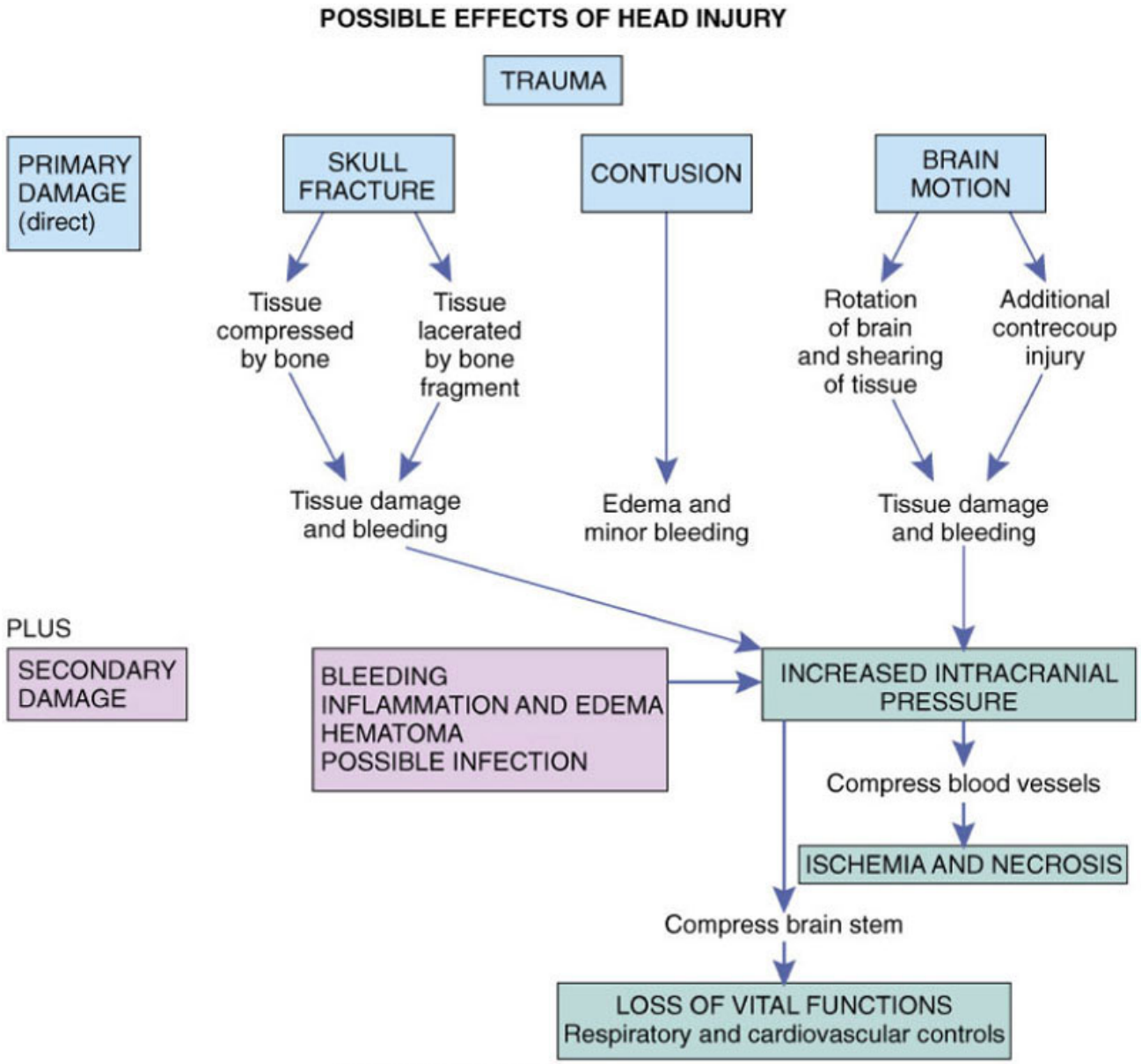


Open Injury

Types of Head Injuries

- Primary brain injuries
  - Direct injuries
    - Laceration or compression of brain tissue
    - Rupture or compression of cerebral blood vessels
    - Damage because of rough or irregular inner surface of the skull
    - Movement of lobes against each other
- Secondary injuries
  - Result from additional effects of cerebral edema, hemorrhage, hematoma, cerebral vasospasm, infection, ischemia related to systemic factors

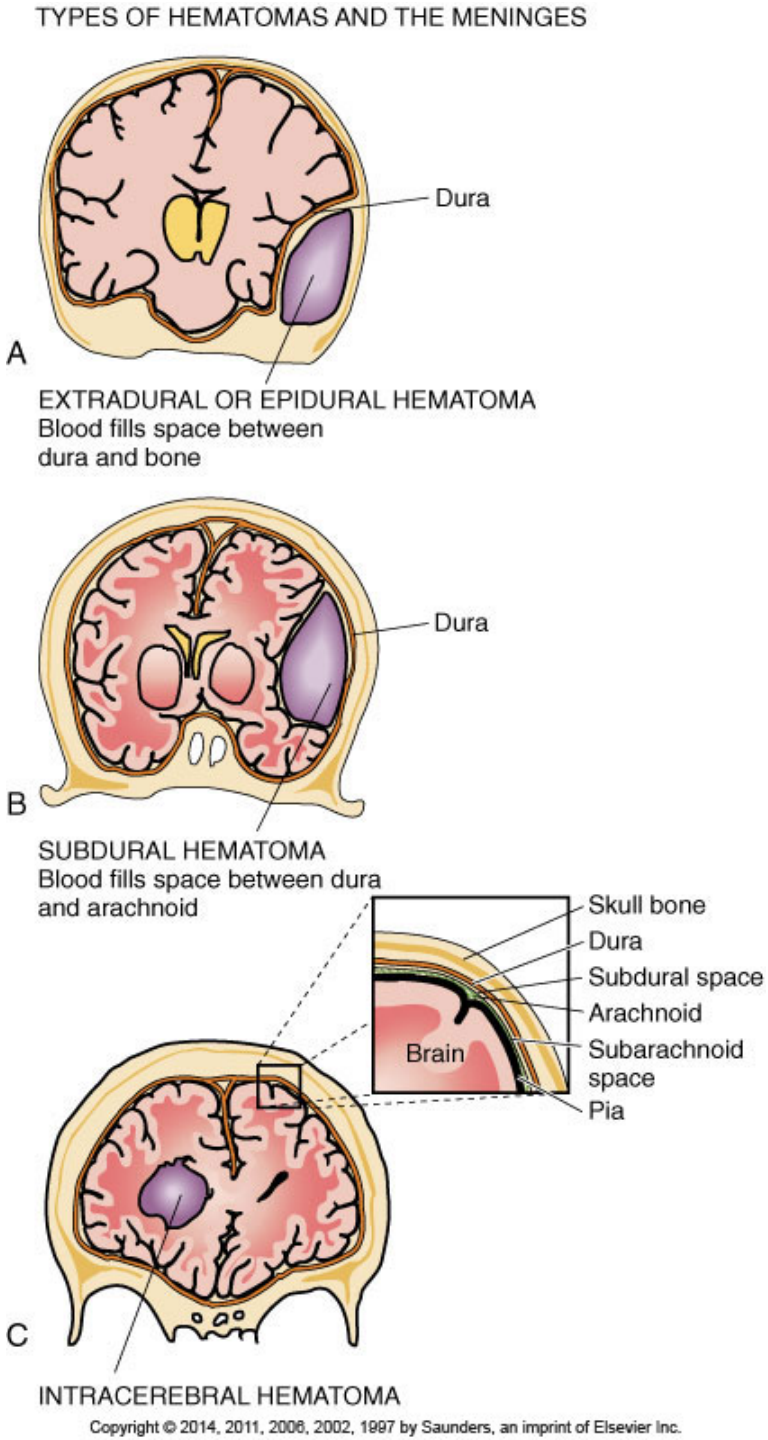
Possible Effects of Head Injury



Head Injuries

- Trauma to brain tissue
  - Causes loss of function in part of body controlled by that area of the brain
  - Cell damage and bleeding lead to inflammation and vasospasm around injury site
    - Increased ICP, general ischemia, dysfunction
    - Some recovery may occur—scar tissue formation
- Hematoma // Classified by location in relation to the meninges

Types of Hematomas



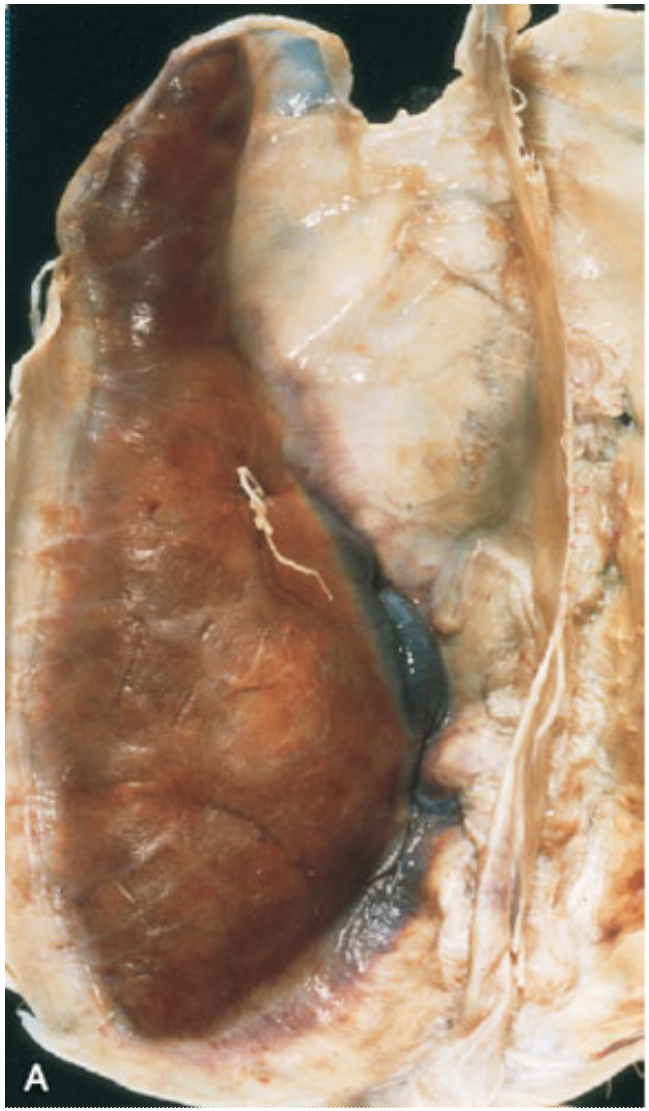
Types of Hematomas

- Epidural hematoma
  - Results from bleeding between dura and skull
  - Signs usually arise within few hours of injury
- Subdural hematoma
  - Develops between dura and arachnoid
  - Hematoma may be acute or subacute
  - Tear in arachnoid may allow CSF to leak into subdural space
    - Creates additional pressure
    - Hematoma disintegrates about 7 days postinjury.
    - Hemolysis increases osmotic pressure → ICP

Types of Hematomas

- Subarachnoid hemorrhage
  - Occurs in space between arachnoid and pia
    - Associated with traumatic bleeding from the blood vessels at the base of the brain
    - Blood mixes with CSF—no localized hematoma formation
- Intracerebral hematoma
  - Results from contusions or shearing injuries
  - May develop several days after injury

Subdural Hematoma



Head Injuries

- All types of hematomas lead to local pressure on adjacent tissue.
  - General increase in Intra Cranial Pressure
- Causes
  - In young adults - sports injury, automobile or motorcycle accident
  - Falls are common causes in any age group.

Signs and Symptoms of Head Injuries

- Focal signs and general signs of increased ICP
- Seizures // Often focal but may be generalized
- Cranial nerve impairment may occur
- Otorrhea or rhinorrhea // Leaking of CSF from ear or nose
- Fever // May be sign of hypothalamic impairment or cranial or systemic infection

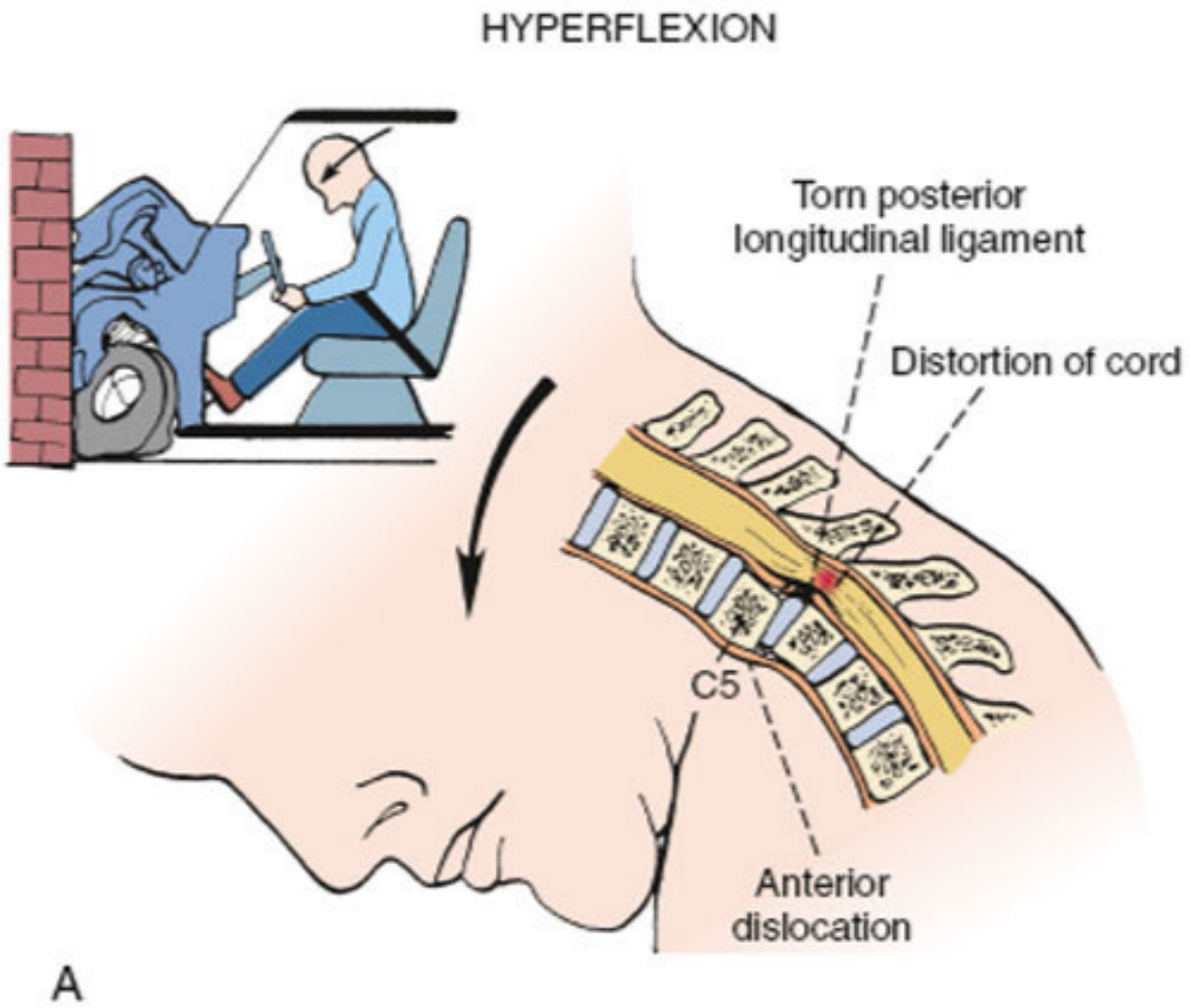
Head Injuries

- CT and MRI // Useful to determine extent of brain injury
- Treatment
  - Glucocorticoid agents // Decrease edema
  - Antibiotics // Reduce risk of infection
  - Surgery may be necessary. // Reduction in ICP
  - Blood products and oxygen // Used to protect remaining brain tissue

Spinal Cord Injury

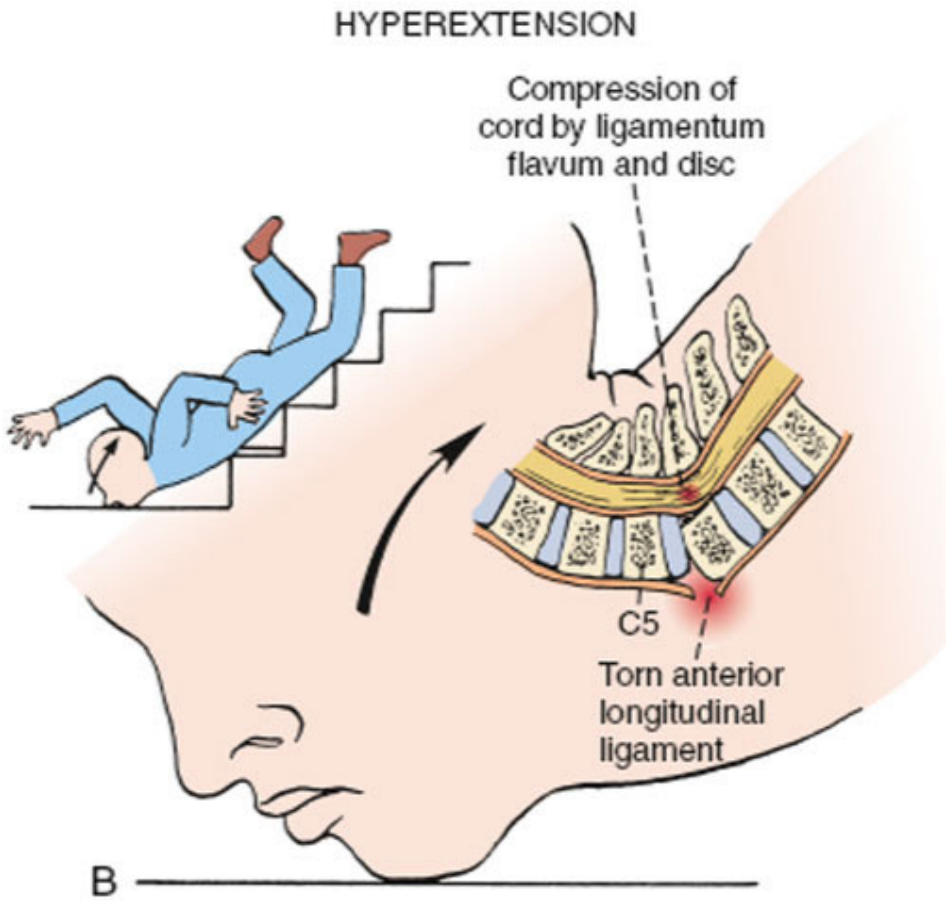
- Results from fracture, dislocation of vertebrae // Compresses, stretches, or tears spinal cord
- Cervical spine injuries // May result from hyperextension or hyperflexion of neck with possible fracture
- Dislocation of vertebra // May crush or compress spinal cord
- Compression // Causes injury to spinal cord when great force is applied to top of the skull or to the feet

Types of Spinal Cord Injuries



From Copstead LC: *Perspectives on Pathophysiology*, Philadelphia, 1995, Saunders.

Types of Spinal Cord Injuries



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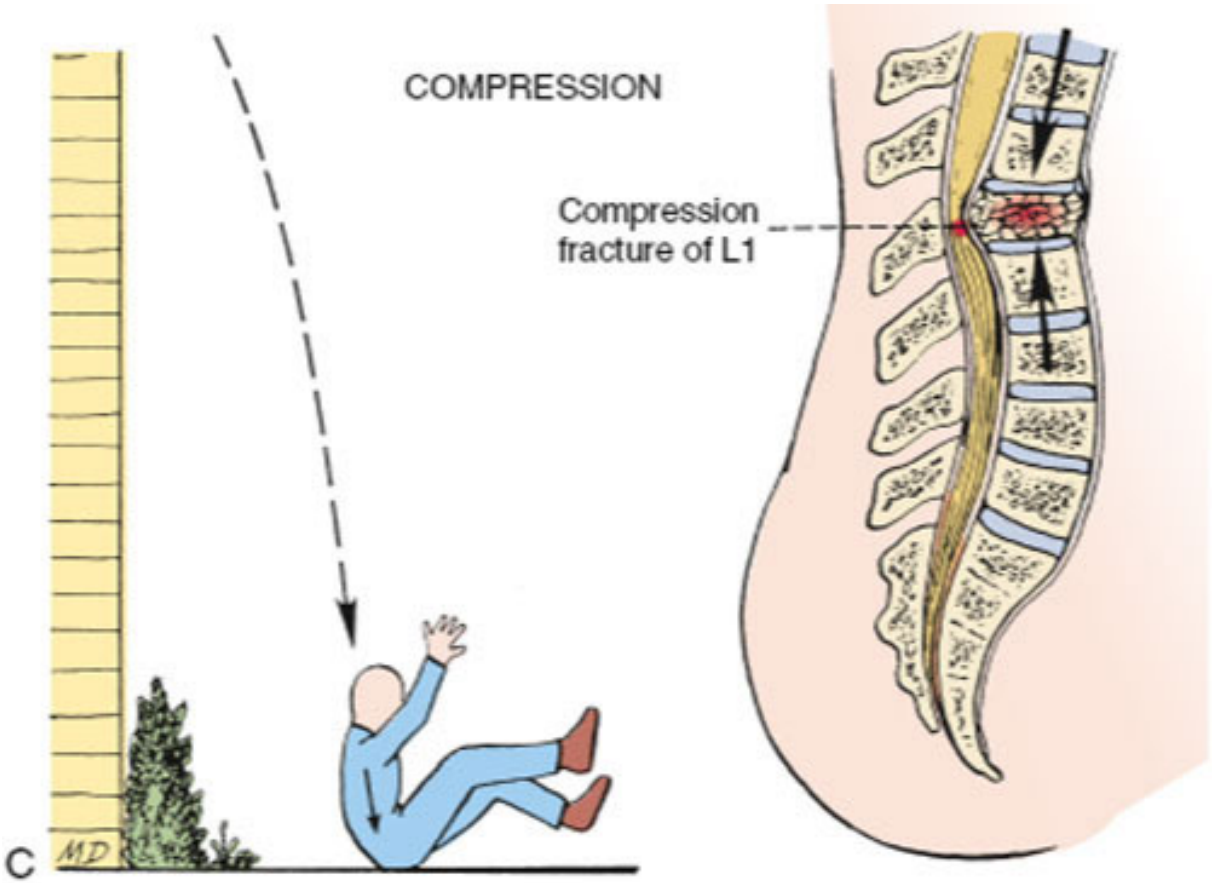
Types of Spinal Cord Injuries



From Thibodeau GA: *Anatomy and Physiology*, St. Louis, 1987, Mosby.

Spinal Cord Injury

- Classification of vertebral fractures
  - Simple // Single line break
  - Compression // Crushed or shattered bone in multiple fragments
  - Wedge // Displaced angular section of bone
  - Dislocation // Vertebra forced out of its normal position

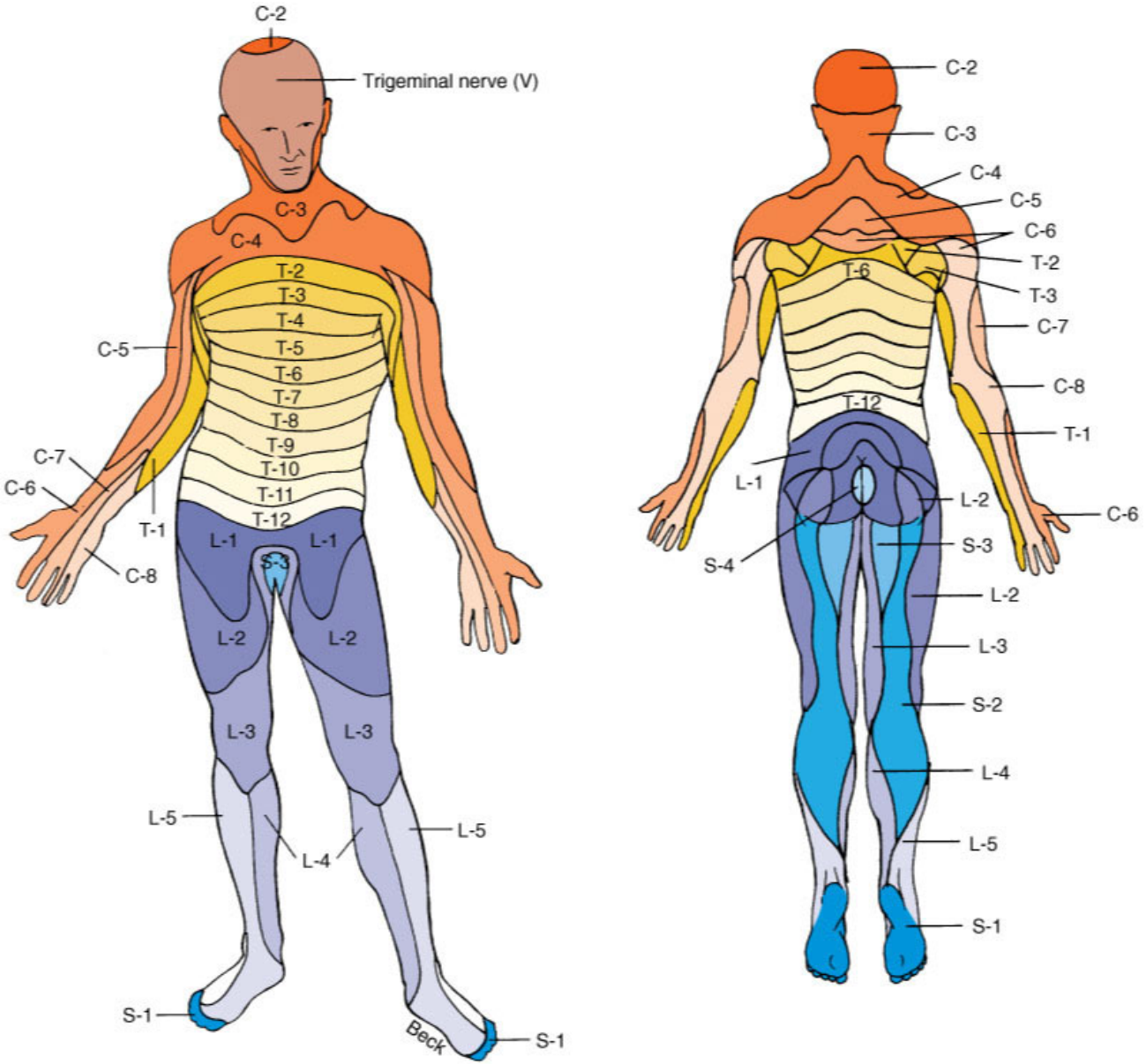
Spinal Cord Injury

- Damage may be temporary or permanent. // Axonal regrowth may occur.
- Laceration of nerve tissue by bone fragments // Usually permanent loss of conduction in affected tracts
- Complete transsection or crushing of cord // Irreversible loss of all sensory and motor function at and below the level of injury
- Partial transection or crushing // May allow recovery of some function

Spinal Cord Injury

- Bruising // Reversible damage
- Prolonged ischemia and necrosis // Lead to permanent damage
- Release of norepinephrine, serotonin, histamine // Released by damaged tissue - vasoconstriction
- Assessment using dermatome map // Assessment of movement and sensory responses // Can determine the degree of damage or recovery

Dermatomes

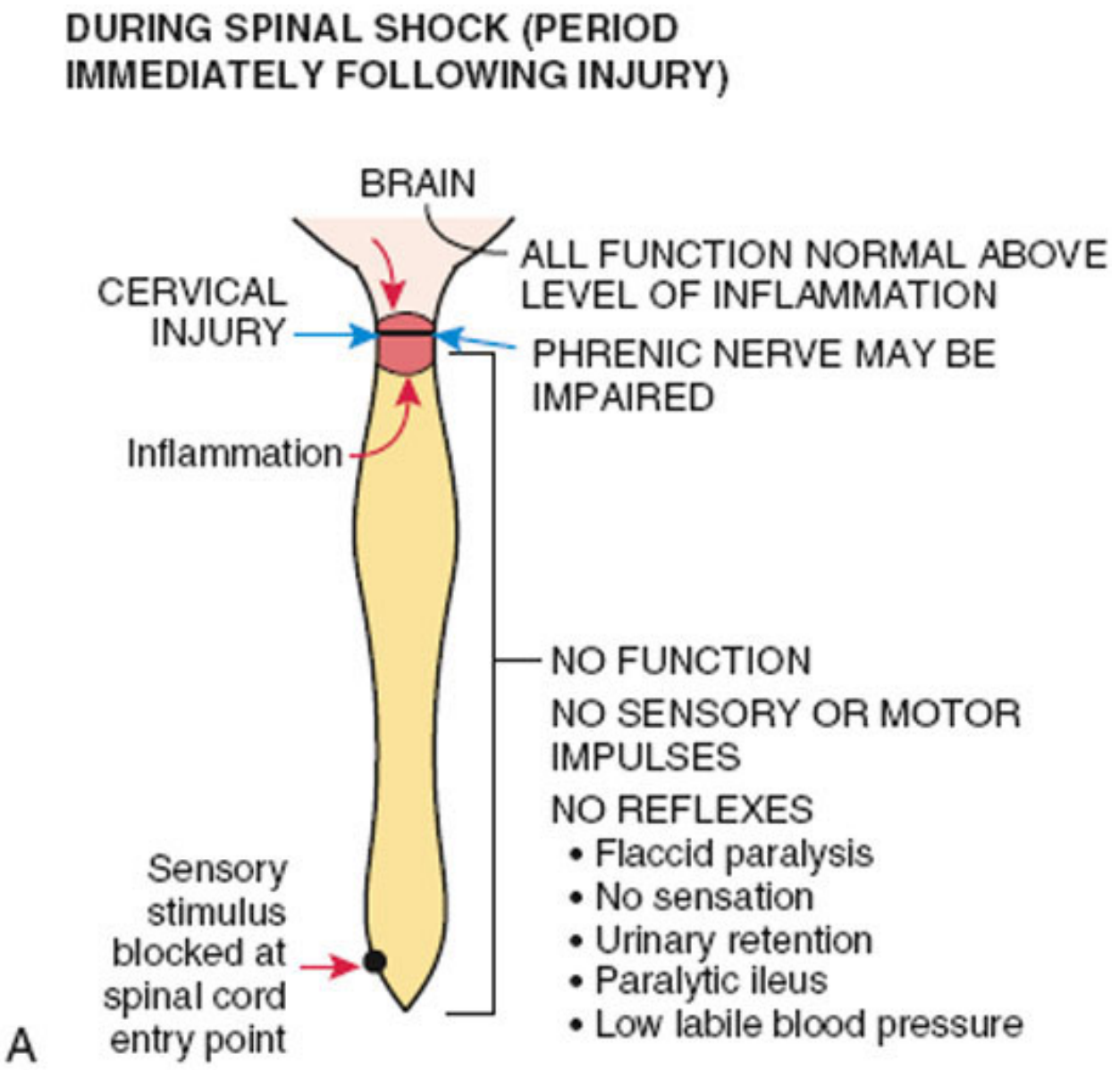


From Thibodeau GA: Anatomy and Physiology, St. Louis, 1987, Mosby.

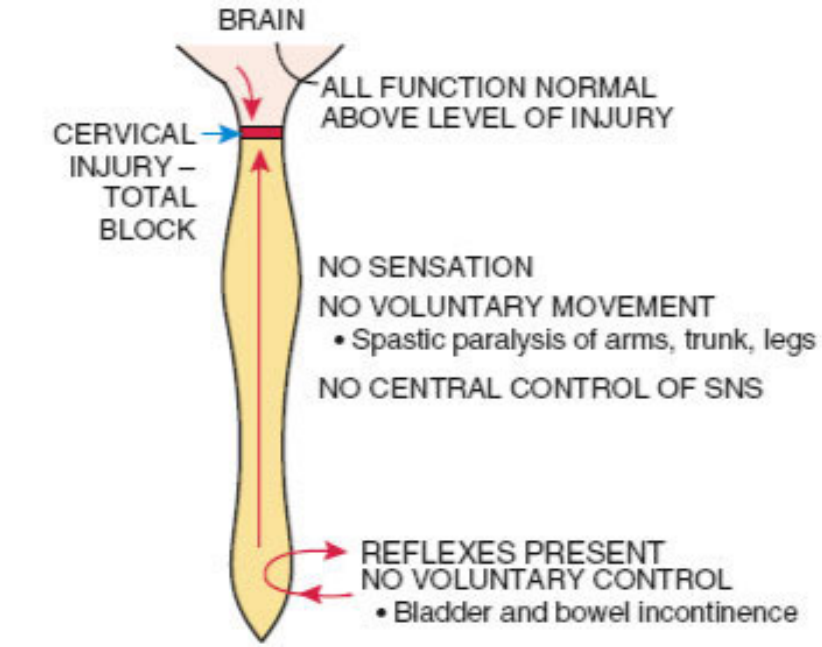
Spinal Cord Injury // Spinal Shock

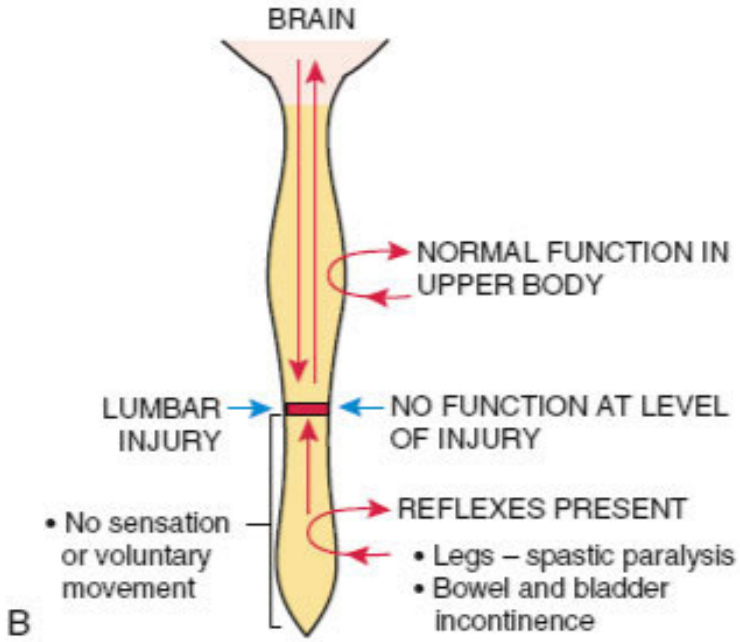
- Initial period after injury - ANS reflexes absent
- Conduction of impulses ceases // Recovery dependent on amount of bleeding and surgical intervention
- Inflammation gradually subsides.
  - Damaged tissue removed by phagocytes
  - Scar tissue formation
- Reflex activity resumes below level of injury.
- No communication with higher levels of brain
- Control of reflexes below the level of damage is lost.

Effects of Spinal Shock



Effects of Spinal Cord Damage After Initial Paralysis As Reflexes Return (Hyperreflexia)





Spinal Cord Injury // Spinal Shock

- Two stages in post-traumatic period
  - Spinal shock // Recovery and recognition of extent of functional loss
- Spinal shock
  - Initially, all neurological activity ceases below and slightly above the level of injury.
  - No reflexes present
  - Condition may persist for days or weeks
    - Flaccid paralysis
    - Sensory loss at and below injured area
    - Absence of all reflexes
    - Loss of central control of autonomic function

Spinal Cord Injury // Spinal Shock

- Recovery
  - Gradual return of reflex activity below level of injury
  - No impulses through specific area of damage
  - Hyperreflexia may develop.
  - Gradually, extent of damage will be revealed.
  - Voluntary motor activity and sensory impulses are blocked at and below the level of damage.
  - Many injuries are incomplete, and permanent damage varies among individuals.

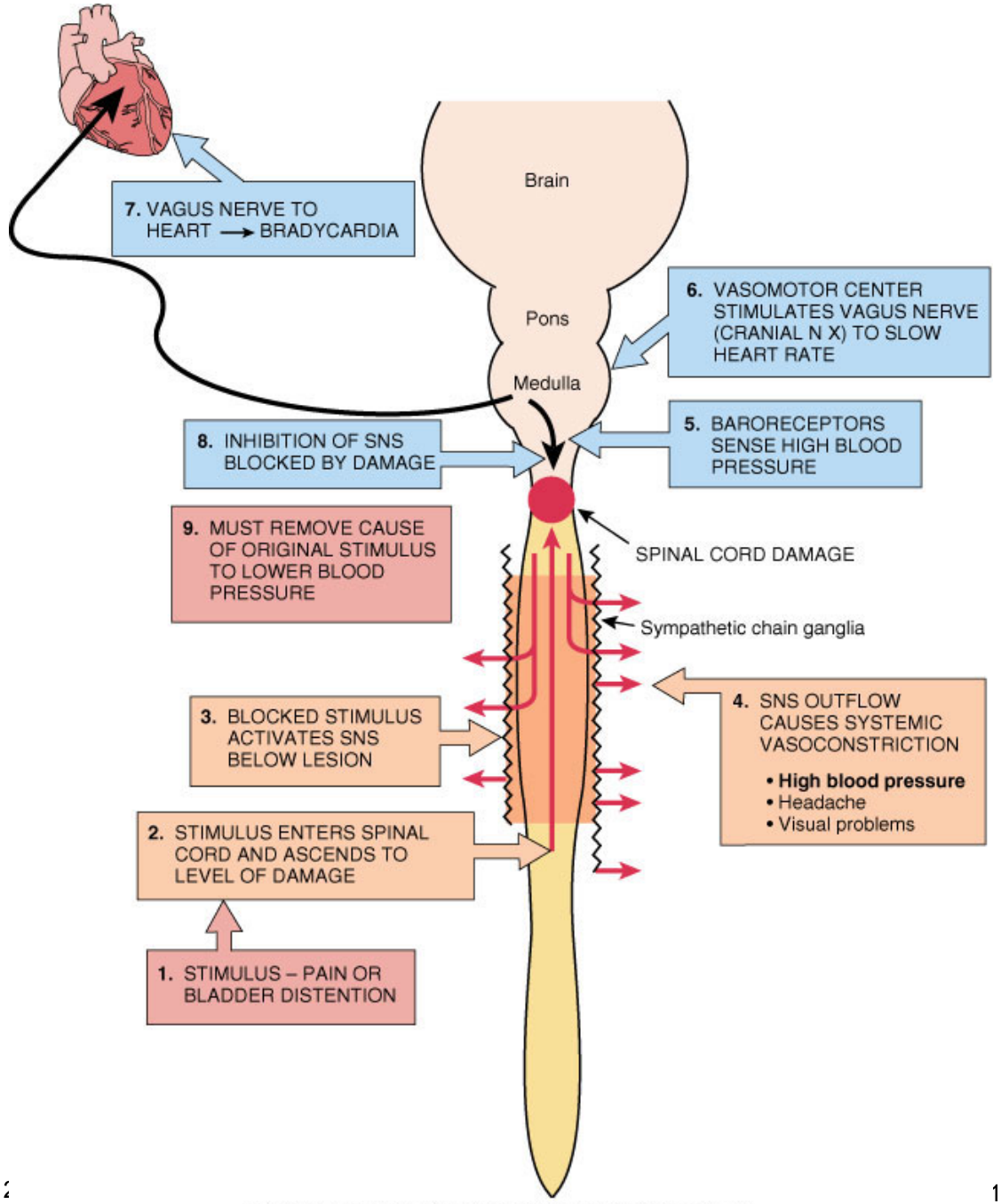
Spinal Cord Injury // Spinal Shock

- Tetraplegia (quadriplegia) // Paralysis of all four extremities
- Paraplegia // Paralysis of the lower part of the trunk and legs
- Ipsilateral paralysis and contralateral loss of pain and temperature sensation // Depends on the point of decussation and location

Autonomic Dysreflexia

- Massive sympathetic reflex response that cannot be controlled from the brain
- Often initiated by infection, genital stimulation, or other stimuli
- Leads to:
  - Increased blood pressure
  - Vasoconstriction below the injury
  - Vasodilation above the injury
  - Tachycardia

Autonomic Dysreflexia Following Spinal Cord Damage



Complications of Spinal Cord Injury

- Urinary tract infections
- Pneumonia
- Skin breakdown
- Spasm and pain
- Depression

Treatment of Spinal Cord Injury

- Treatment and rehabilitation begin at the time of injury.
- Immobilize spine.
- Maintain breathing and prevent shock.
- Hospital traction or surgery // Relieve pressure and repair tissues
- Glucocorticoids // Reduce edema and stabilize vascular system
- Ongoing care to prevent complications related to immobility

Seizure Disorders

- Uncontrolled, excessive discharge of neurons in the brain
- May be localized or generalized
- Two basic categories, generalized and partial
- Many possible causes
- Classified by their location in the brain and clinical features

Congenital Neurological Disorders

- Hydrocephalus // Excess CSF accumulates within the skull
- Spina bifida // Group of neural tube defects of varying severity
- Cerebral palsy // Group of disorders with some degree of *motor impairment*

Hydrocephalus

- Excess CSF accumulates in the skull.
  - Compresses brain tissue and blood vessels
  - Generally, CSF accumulates because more is produced than reabsorbed.
- If cranial sutures have not closed: // Infant's head enlarges beyond normal size
- Two types of hydrocephalus
  - Noncommunicating or obstructive hydrocephalus
  - Communicating hydrocephalus

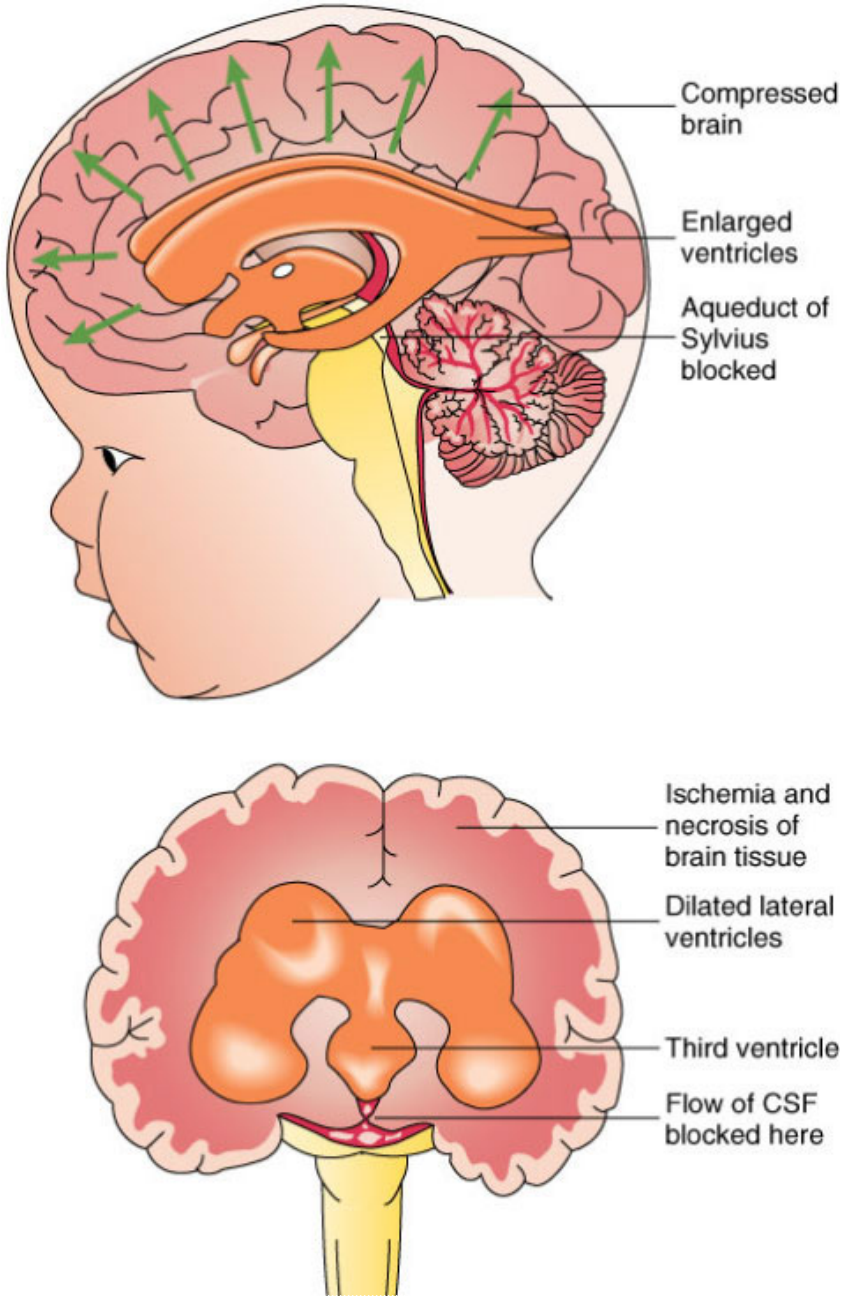
Non-communicating Hydrocephalus / Obstructive

- Flow of CSF through the ventricular system is blocked
- Usually results from a fetal developmental abnormality
  - Stenosis or neural tube defect // May be associated with myelomeningocele // Or Arnold-Chiari malformation is present in many neonates
- Obstruction leads to backpressure of fluid in the brain ventricles.
  - Enlarges ventricles and compresses blood vessels and brain tissue

Communicating Hydrocephalus

- Absorption of CSF through subarachnoid villi is impaired
- Neonates // Skull can expand to a certain degree to relieve pressure.
  - If not treated, brain tissue is permanently damaged.
- Older children and adults
  - ICP increases more rapidly than in neonates.
  - Amount of brain damage depends on the rate of pressure increase and time of relief.

Hydrocephalus



Hydrocephalus

- Obstruction from tumors, infection, scar tissue
- Meningitis may cause obstructive hydrocephalus.
  - Fibrosis in meninges
  - Impaired absorption

Signs and Symptoms Hydrocephalus

- Signs of increasing CSF depends on age of patient
- Pupil response light to sluggish
- Scalp veins appear dilated.
- Infant
  - Lethargic, irritable, difficult to feed
  - Eyes show “sunset sign”—white sclera visible above colored pupil
  - High-pitched cry when moved or picked up
- Must be diagnosed and treated as soon as possible to minimize brain damage

Hydrocephalus

- Diagnostic tests
  - CT, MRI
    - Helps locate the obstruction or abnormal flow
    - Determines the size of the ventricles
- Treatment
  - Surgery // To remove obstruction
    - Provides a shunt for CSF from ventricle into the peritoneal cavity or other extracranial site // Shunt will have to be replaced as child grows

Spina Bifida

- Group of neural tube defects
- Common developmental abnormality
- Failure of the posterior spinous processes on the vertebrae to fuse
  - Permit meninges and spinal cord to herniate
  - Result in neurological impairment
- Any number of vertebrae can be involved. // Most common location - lumbar area

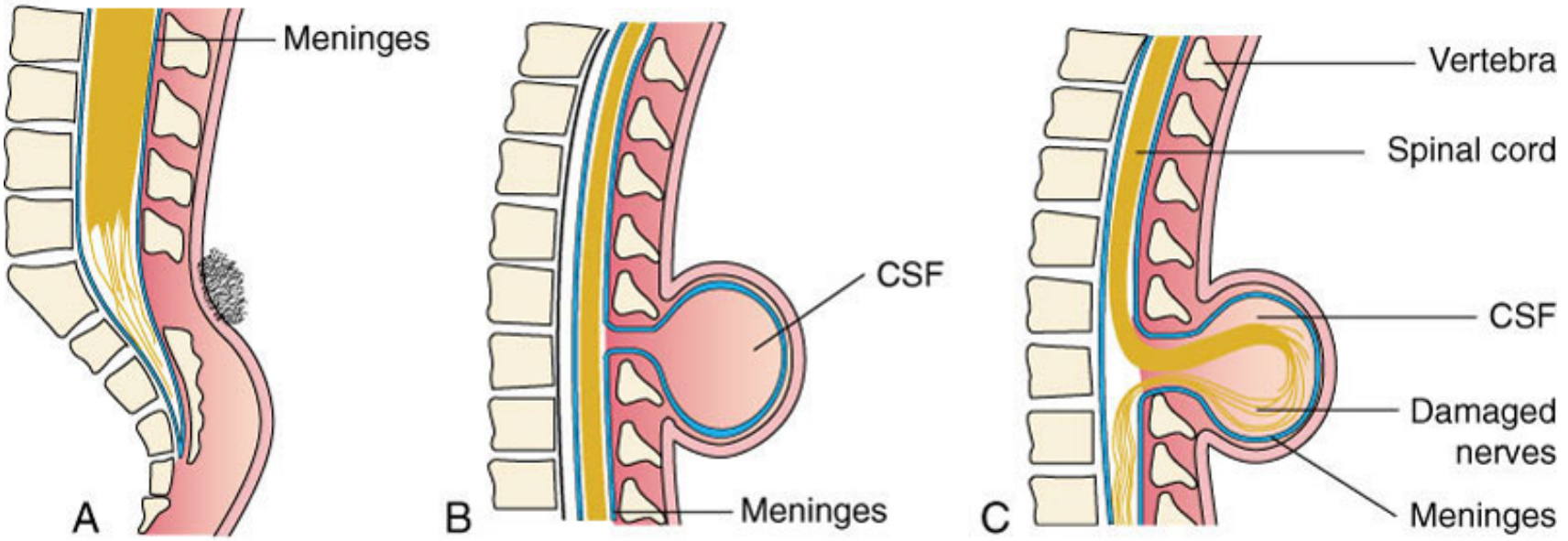
Types of Spina Bifida

- Spina bifida occulta
  - Spinous processes do not fuse.
  - Herniation of spinal cord and meninges does not occur.
  - Defect may not be visible. // Dimple or a tuft of hair may be present on the skin over the site.
  - Diagnosed by routine radiograph or on the basis of mild neurological signs

Types of Spina Bifida

- Meningocele // Herniation of the meninges occurs through defect
  - Meninges and CSF form a sac on the surface
  - Transillumination confirms absence of nerve tissue in sac
- Myelomeningocele // Most serious form
  - Herniation of spinal cord and nerves, along with meninges and CSF
  - Considerable neurological impairment

Spina Bifida



Cause of Spina Bifida

- Appears to have multifactorial basis
  - Combination of genetic and environmental factors // Radiation, gestational diabetes, deficits of folic acid
  - High familial incidence
- Diagnostic tests
  - Alpha-fetoprotein (AFP) level elevated in maternal blood
    - Leaked from the defect
    - Specimen obtained at 16 to 18 weeks of gestation (amniocentesis)
  - Diagnosis prenatally by ultrasound and/or amniotic fluid analysis

Signs and Symptoms Spina Bifida

- Meningocele or myelomeningocele visible as a protruding sac over the spine
  - Neurological deficit depends on level of defect and on status of nerve tissue.
  - Sensory and motor function below level of herniation is impaired.
- Treatment
  - Surgical repair
  - Ongoing assistance and occupational and physical therapy after repair

Cerebral Palsy

- Group of disorders marked by motor impairment
- Damage may occur before, during, or shortly after birth
- Caused by:
  - Genetic mutations
  - abnormal fetal formation of functional brain areas
  - brain damage in the perinatal period
  - Brain tissue altered by malformation
  - mechanical trauma
  - Hypoxia
  - Hemorrhage
  - Hypoglycemia
  - Hyperbilirubinemia
  - infection → necrosis

Cerebral Palsy

- Single or multiple factors
- Hypoxia or ischemia cause of high incident of major brain damage
  - Can be caused by placental complications, difficult delivery, vascular occlusion, hemorrhage, aspiration, respiratory impairment in premature infant, high bilirubin levels
- Infection or metabolic abnormalities
- Hypoglycemia in mother or child

Cerebral Palsy Associated Conditions

- Spastic paralysis // Results from damage to the pyramidal tracts, motor cortex, general cortical damage // Characterized by hyper-reflexia

- Dyskinetic disease // Damage to the extrapyramidal tract, basal nuclei, cranial nerves // Manifested by athetoid or choreiform involuntary movements

- Ataxic cerebral palsy // Damage to the cerebellum // Loss of balance and coordination

Cerebral Palsy

- Other common areas of dysfunction
  - Intellectual function // Varies from normal intelligence to severely cognitively disabled in a small percentage of cases
  - Communication and speech // Difficult because of motor disability
  - Seizures
  - Visual problems // Visual or hearing deficits

Treatment of Cerebral Palsy

- Individualized and immediate therapy
- Stimulation programs
- Assessment
- Speech therapy
- Physical therapy and regular exercise therapy
- Devices that can improve mobility and coordination
- Occupational therapy
- Monitoring of hearing and vision
- Use of alternate modes of communication

Chronic Degenerative Disorders

Multiple Sclerosis (MS)

- Progressive demyelination of neurons in the brain, spinal cord, and cranial nerves
- Different types of MS // Variation in effects, severity, and progression
- Loss of myelin interferes with conduction of impulses in affected fibers
  - May affect motor, sensory, and autonomic fibers
  - Occurs in diffuse patches in the nervous system

Multiple Sclerosis (MS)

- Earliest lesions // Inflammatory response
  - Loss of myelin in white matter of brain or spinal cord
- Plaques
  - Larger areas of inflammation and demyelination
  - Develop later, become visible // Frequently next to the lateral ventricles, brainstem, optic nerve
  - Plaques vary in size. // Several may coalesce into a single patch.

Multiple Sclerosis (MS)

- Recurrence
  - Initial inflammation may subside
  - Neural function may return to normal for short period of time.
  - In time, neural degeneration becomes irreversible // Function is lost permanently.
  - *Each recurrence causes additional areas of the CNS to become affected*.
  - MS varies in severity.

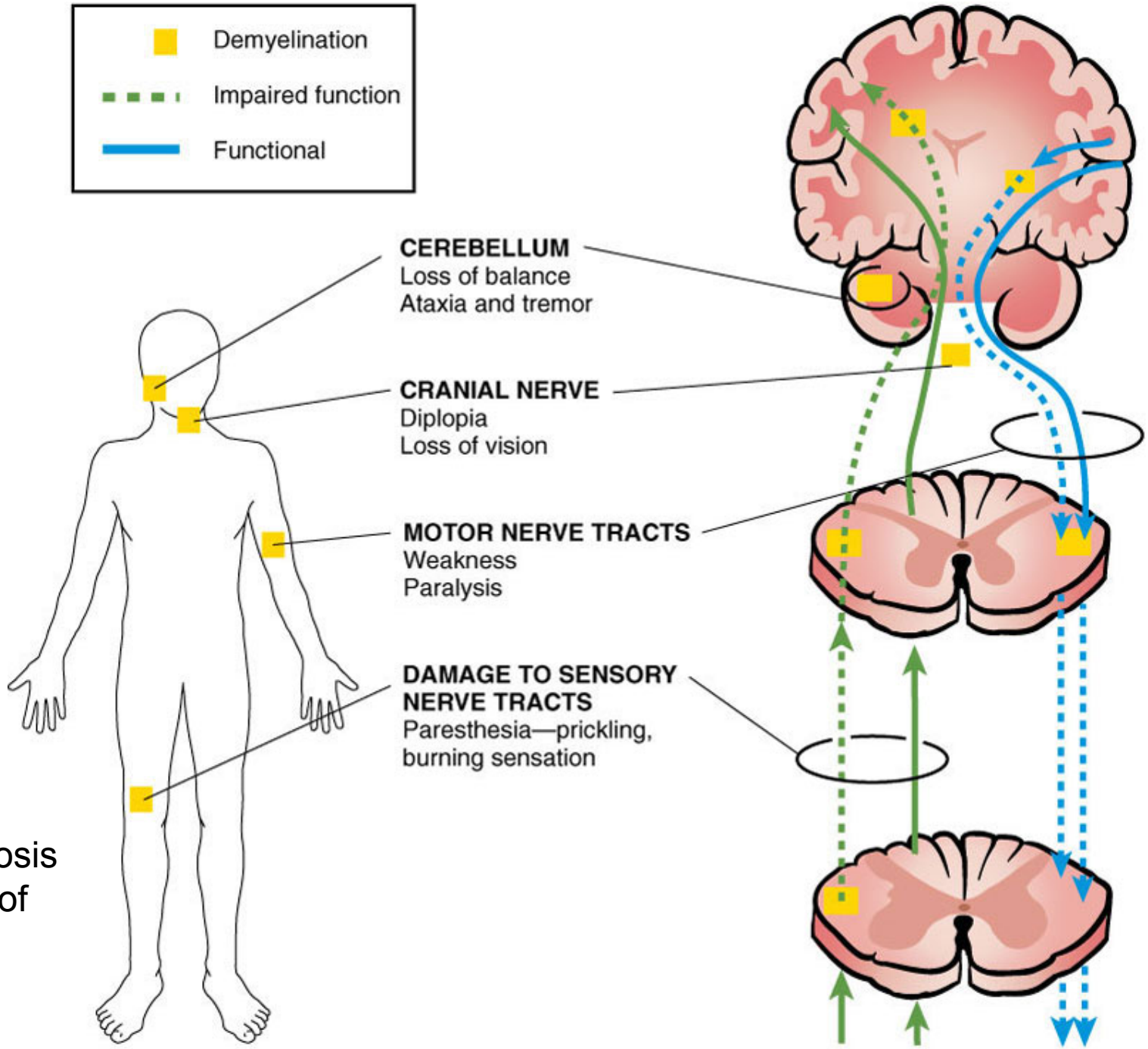
Multiple Sclerosis (MS)

- Onset usually occurs between 20 to 40 years of age.
- Cause unknown
  - May be an autoimmune disease
  - May be nutritional deficit
  - May be change in blood flow to neurons // astrocytes
- May have genetic, immunological, and environmental components
- Increased risk for close relatives of affected individuals

Signs and Symptoms of Multiple Sclerosis
(Some May Apply)

- Manifestations determined by areas of demyelination
- Blurred vision, weakness in legs
- Diplopia (double vision), scotoma (spot in visual field)
- Dysarthria
- Paresthesia, areas of numbness, burning, tingling
- Progressive weakness and paralysis extending to the upper limbs
- Loss of coordination, bladder, bowel and sexual dysfunction, chronic fatigue

Multiple Sclerosis
Distribution of
Lesions



Multiple Sclerosis (MS)

- Diagnostic tests // No definitive test
  - MRI for diagnosis and monitoring best option
- Treatment
  - No definitive treatment approved at this time
  - Several research trials in progress
  - Therapy includes physical therapy, occupational therapy
  - Manifestations require individual attention.

Parkinson's Disease

- Progressive degenerative disorder
- Dysfunction of the extrapyramidal motor system
  - Progressive degeneration in basal nuclei
  - Imbalance between excitation and inhibition in basal nuclei
  - Excess stimulation affects movement and posture.
    - Resting tremors
    - Muscular rigidity
    - Difficulty initiating movement
    - Postural instability

Parkinson's Disease

- Primary or idiopathic Parkinson's disease // Usually develops after age 60
- Secondary parkinsonism caused by:
  - Encephalitis
  - Trauma (e.g., sports injury)
  - Vascular disease
  - Drug-induced (e.g., phenothiazine tranquilizers)

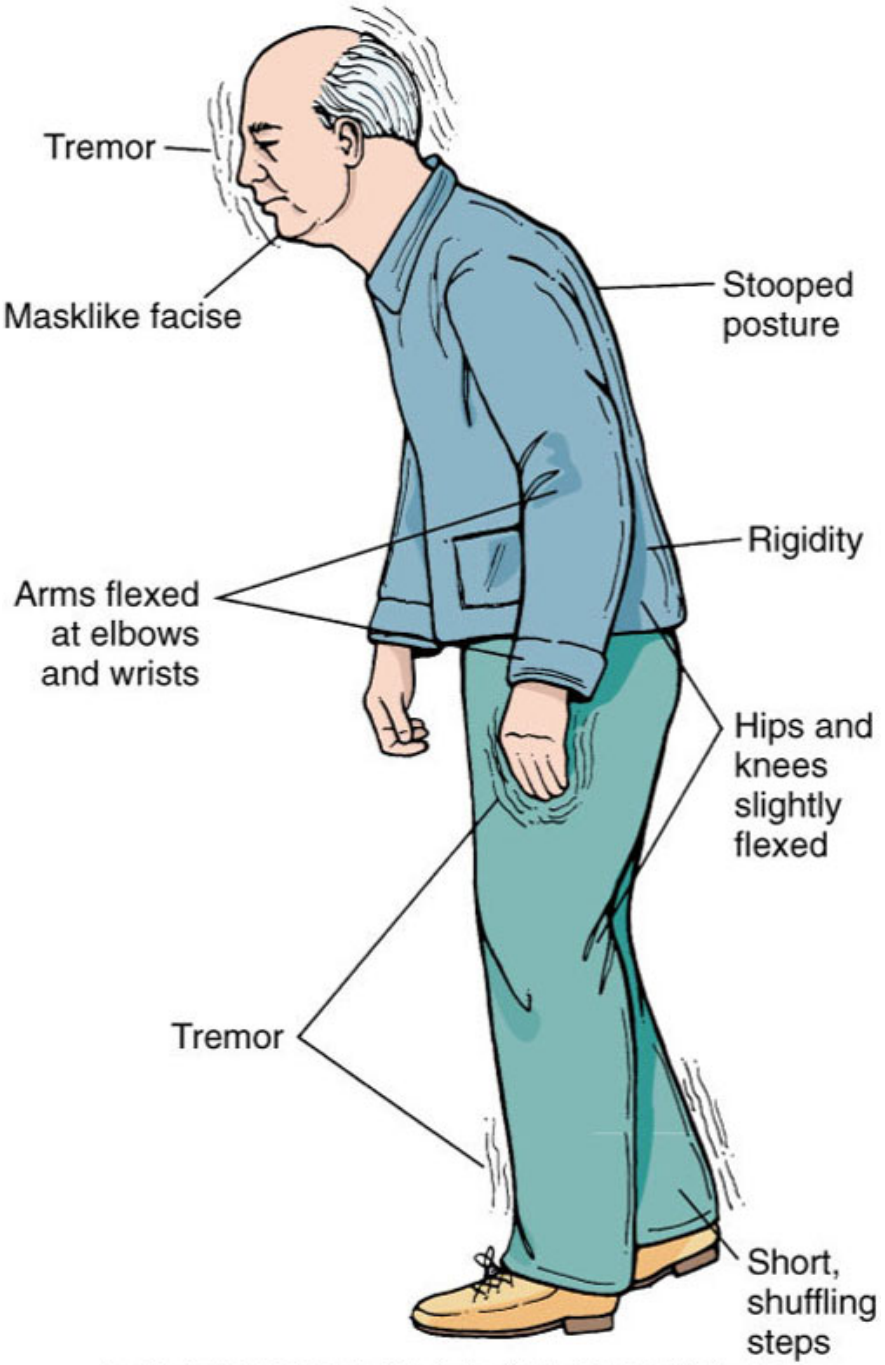
Parkinson's Disease

- Early signs and symptoms
  - Fatigue
  - Muscle weakness, muscle aching,
  - Decreased flexibility
  - Less spontaneous changes in facial expression
  - Tremors in the hands at rest, repetitive pill-rolling motions of hands

Parkinson's Disease

- Later signs and symptoms
  - Tremors affect hands, feet, face, tongue, lips
  - Increased muscle rigidity
  - Difficulty initiating movements
  - Slow movements
  - Lack of associated involuntary movements
  - Characteristic standing posture is stooped, leaning forward
  - Propulsive gait
  - Complex activities become slow and difficult.

Parkinson's Disease



From Monahan FD, Drake T, Neighbors M: Nursing Care of Adults, Philadelphia, 1994, Saunders.

Parkinson's Disease

- Other functions affected
  - Low voice, devoid of inflection
  - Dysarthria
  - Chewing and swallowing become difficult. // Prolonging eating time // Recurrent drooling
  - Face might resemble a mask
    - Blinking of eyelids reduced
    - Blank, staring face
    - Impairs communication

Parkinson's Disease

- Other functions affected
  - Autonomic dysfunction
    - Urinary retention // Constipation
  - Orthostatic hypotension // Threat of falls increases
  - Urinary tract and respiratory tract infections are common complications.
  - Dementia develops late in course of disease

Treatment of Parkinson's Disease

- Removal of cause, if known
- Dopamine replacement therapy // Levodopa - dopamine precursor (L-Dopa)
- Anticholinergic drugs
- Speech and language pathologist
- Physical therapy
- Occupational therapy // Improves balance, coordination
- Monitoring and treatment of respiratory and urinary tract infections

Amyotrophic Lateral Sclerosis

- Also referred to as Lou Gehrig's disease
- No identified cause // Genes on various chromosomes have been linked to the disease.
- Progressive degenerative disease affecting upper motor neurons in the cerebral cortex and lower motor neurons in brainstem and spinal cord
- No indication of inflammation around the nerves
- Cognition unimpaired

Amyotrophic Lateral Sclerosis

- Loss of upper motor neurons in cerebral cortex // Spastic paralysis with hyperreflexia
- Damage to lower motor neurons
  - Flaccid paralysis
  - Decreased muscle tone and reflexes
- Progressive muscle weakness and loss of fine motor coordination
  - Stumbling and falls are common.
- *Death occurs because of respiratory failure*.

Treatment for Amyotrophic Lateral Sclerosis

- No specific treatment to slow degeneration
- Stem cell therapy under investigation
- Pharmaceutical treatment (e.g., with Riluzole [Rilutek]) to slow further damage to neurons
- Moderate exercise and rest
- Respiratory therapy, appropriate nutrition, speech pathology, occupational therapy, physical therapy, psychological treatment

Myasthenia Gravis

- Autoimmune disorder
- Autoantibodies to acetylcholine (ACh) receptors form. // Destruction of receptor site // Prevention of further muscle stimulation
  - Skeletal muscle weakness
  - Facial and ocular muscles usually affected first
  - **NOTE:** Dysphagia and aspiration are significant problems!

Myasthenia Gravis

- Diagnostic tests
  - Electromyography
  - Serum antibody test
- Signs and symptoms
  - Muscle weakness in face and eyes
  - Impaired vision, monotone speech
  - Difficult chewing and swallowing
  - Head droops, arms become weaker
  - Upper respiratory infections common

Myasthenia Gravis

- Treatment
  - Anticholinesterase agents // Temporary improvement of neuromuscular transmission
  - Glucocorticoids // Suppression of immune system
  - Plasmapheresis // Removal of antibodies from the blood
  - Thymectomy

Huntington's Disease

- Inherited disease // Autosomal dominant gene - Carried on chromosome 4
- Does not usually manifest until individual is older than 40 years
- Progressive atrophy of brain // Particularly in basal ganglia (nuclei) and frontal cortex
- Depletion of gamma-aminobutyric acid (GABA) in the basal nuclei
- Levels of ACh in brain appear to be reduced.

- Signs and Symptoms of Huntington's Disease
  - Mood swings, personality changes
  - Restlessness, choreiform movements in arms and face
- Diagnostic tests // DNA analysis
- Treatment // Currently no therapy to slow progression of disease // Symptomatic therapy only

Dementia

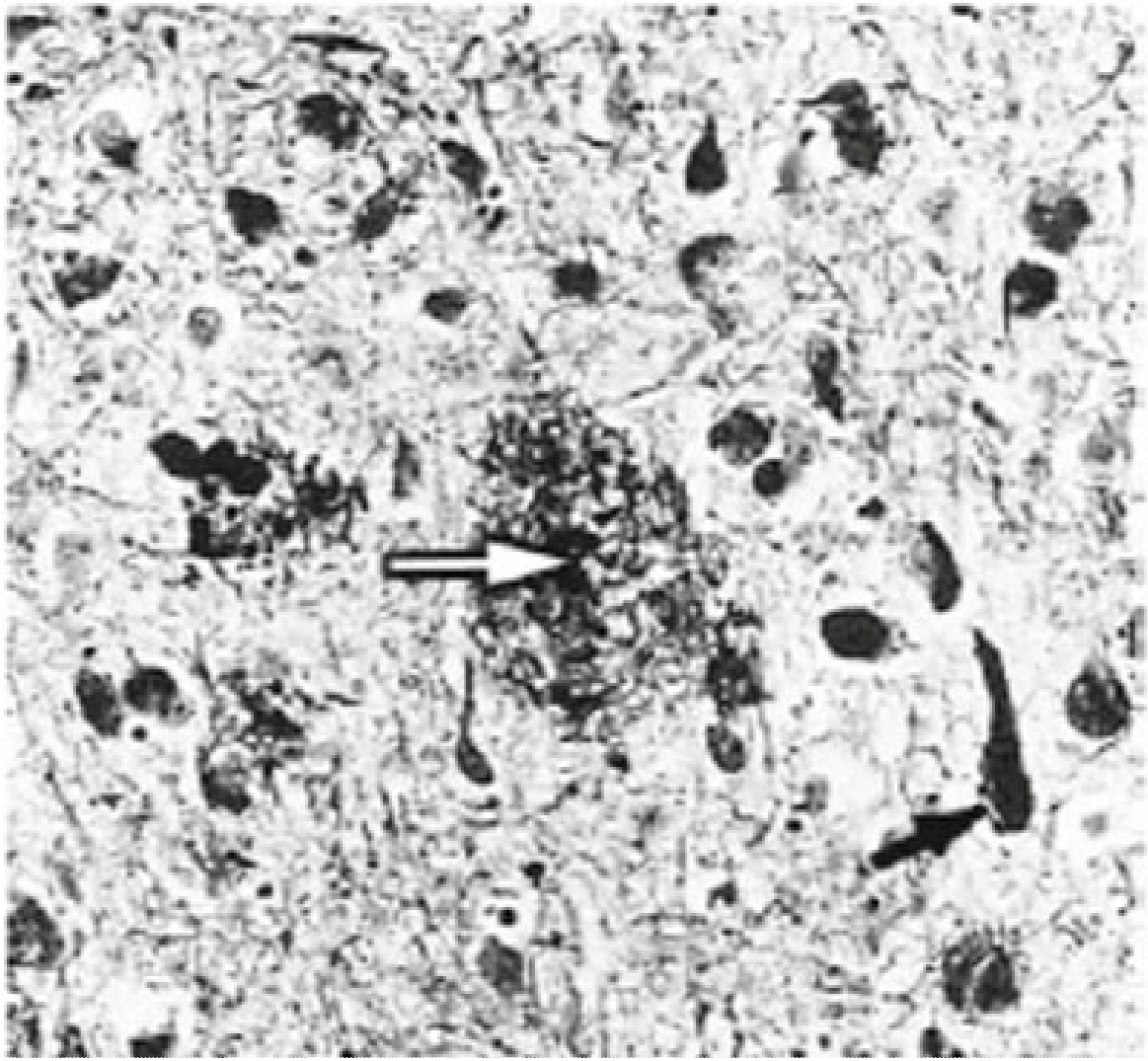
- Progressive chronic disease
  - Cortical function is decreased.
    - Impaired cognitive skills
    - Impaired thinking, judgment, and learning
    - Memory loss
    - Confusion
    - Behavioral and personality changes
- Many causes of dementia
  - Vascular disease
  - Infections
  - Genetic disorders

Alzheimer's Disease

- Progressive cortical atrophy
  - Neurofibrillary tangles and plagues
  - ACh deficit caused by loss of neurons
- No definite diagnostic tests available
  - Exclusion of other disorders
  - Careful medical and psychological history
- Specific cause unknown // Repetitive DNA sequences on different chromosomes have been associated with AD.

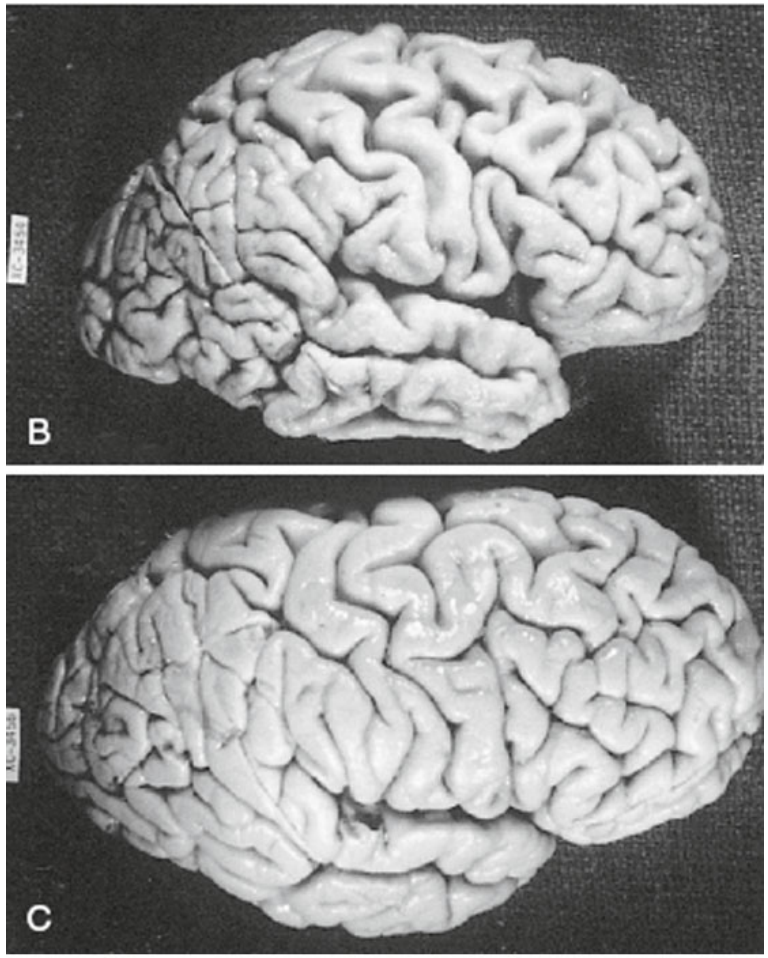
Pathological Changes with Alzheimer's Disease

- mature plaque with central amyloid core (*white arrow*) and neurofibrillary tangle (*black arrow*).



Pathological Changes with Alzheimer's Disease

- Alzheimer's disease // Brain is smaller, with narrower gyri and wider sulci.
- Normal brain.



From Damjanov I, Linder J: *Anderson's Pathology*, ed 10, St. Louis, 1996, Mosby.

Signs and Symptoms of Alzheimer's Disease

- Extend over 10 to 20 years
- Behavioral changes // Irritability, hostility, mood swings
- Gradual loss of memory and lack of concentration
- Impaired learning, poor judgment
- Decline of cognitive function, memory, language
- Change in food intake
- Inability to recognize family, lack of environmental awareness, incontinence, inability to function

Alzheimer's Disease

- Treatment
  - No specific treatment
  - Anticholinesterase drugs // Some temporary improvement
  - Occupational therapy, psychologists, speech therapy
  - Team approach needed to support client and caregivers

Other Forms of Dementia

- Vascular dementia
  - Caused by cerebrovascular disease
  - Often a result of multiple small brain infarctions
  - Common in persons older than 70 years // Especially in those with hypertension
  - Onset insidious // Memory loss, apathy, inability to manage daily routines
  - Progression may be in stages.
  - Other neurologic impairment is common.

Other Forms of Dementia

- Creutzfeldt-Jacob disease (CJD)
  - Rare, rapidly progressive
  - Caused by prion ingested or transmitted through contaminated blood
  - May be iatrogenic // Invasive procedures, surgery (e.g., corneal transplantation) transfer prions
  - Most sporadic
  - Long incubation period
  - Memory loss, behavioral changes, motor dysfunction, progressive dementia

AIDS Dementia

- Common in later stages of AIDS
- Virus invades brain tissue
- May be exacerbated by other infections and tumors
- Gradual loss of memory and cognitive ability
- Impaired motor function
- Children with congenital HIV infection
  - Brain frequently affected
  - Severe developmental delay

Mental Disorders

- Classified in the *Diagnostic and Statistical Manual of Mental Disorders,* 5th edition (DSM-5)
- Dysfunctions in the areas of behavior or personality
- Biochemical and/or structural abnormalities may be identified.
- Genetic component possible
- Psychotic illnesses include: // Schizophrenia, delusional disorders, mood disorders, anxiety, panic disorders

Schizophrenia

- Variety of syndromes // Different in each individual
- Common changes include:
  - Reduced gray matter in temporal lobes
  - Enlarged third and lateral ventricles
  - Abnormal cells in the hippocampus
  - Excessive dopamine secretion
  - Decreased blood flow to frontal lobes
- Theories include: // Genetic predisposition or Brain damage in fetus

Schizophrenia

- Grouped symptoms
  - Positive // Delusions, bizarre behavior
  - Negative // Flat emotions, decreased speech
- General symptoms include
  - Disorganized thought process
  - Impaired communication, with inadequate language skills
  - Delusions or false beliefs and ideas

Treatment / Schizophrenia

- Antipsychotic drugs
  - Frequently cause side effects related to excessive extrapyramidal activity
  - Involuntary muscle spasms in face, neck, arms, or legs
  - Chewing or grimacing
  - Repetitive jerky or writhing movements
  - Some side effects may be reduced by antiparkinson agents.
- Psychotherapy and psychosocial rehabilitation

Depression

- Depression is a common problem and often occurs in clients with chronic illness.
- Classified as mood disorder with several subgroups
  - Unipolar // Endogenous, diagnosis based on biological factors or personal characteristics
  - Bipolar // Alternating periods of depression and mania // Response to life event or secondary to a systemic disorder
- Factors include genetic, developmental, and psychosocial stressors.

Depression

- Results from decreased activity by excitatory neurotransmitters // Norepinephrine and serotonin
- Exact mechanism unknown // Suggested genetic component
- Indicated by prolonged period of unfounded sadness and by:
  - Lack of energy
  - Loss of self-esteem and motivation
  - Irritability and agitation
  - Insomnia or excessive sleep
  - Loss of appetite and libido

Treatment // Depression

- Recommended that counseling be combined with:
  - Antidepressant drugs that increase norepinephrine activity
  - Selective serotonin reuptake inhibitors
  - Serotonin-norepinephrine reuptake inhibitors
  - Tricyclic antidepressants
  - Monoamine oxidase inhibitors
- Electroconvulsive therapy may be used for severe depression.

Panic Disorders

- Panic attack // Sudden brief episode of discomfort and anxiety
- Panic disorder // Develops when panic attacks are frequent and prolonged
- Genetic factor implicated
  - Increased discharge of neurons in temporal lobes
  - Neurotransmitter abnormalities include norepinephrine, serotonin, and GABA.

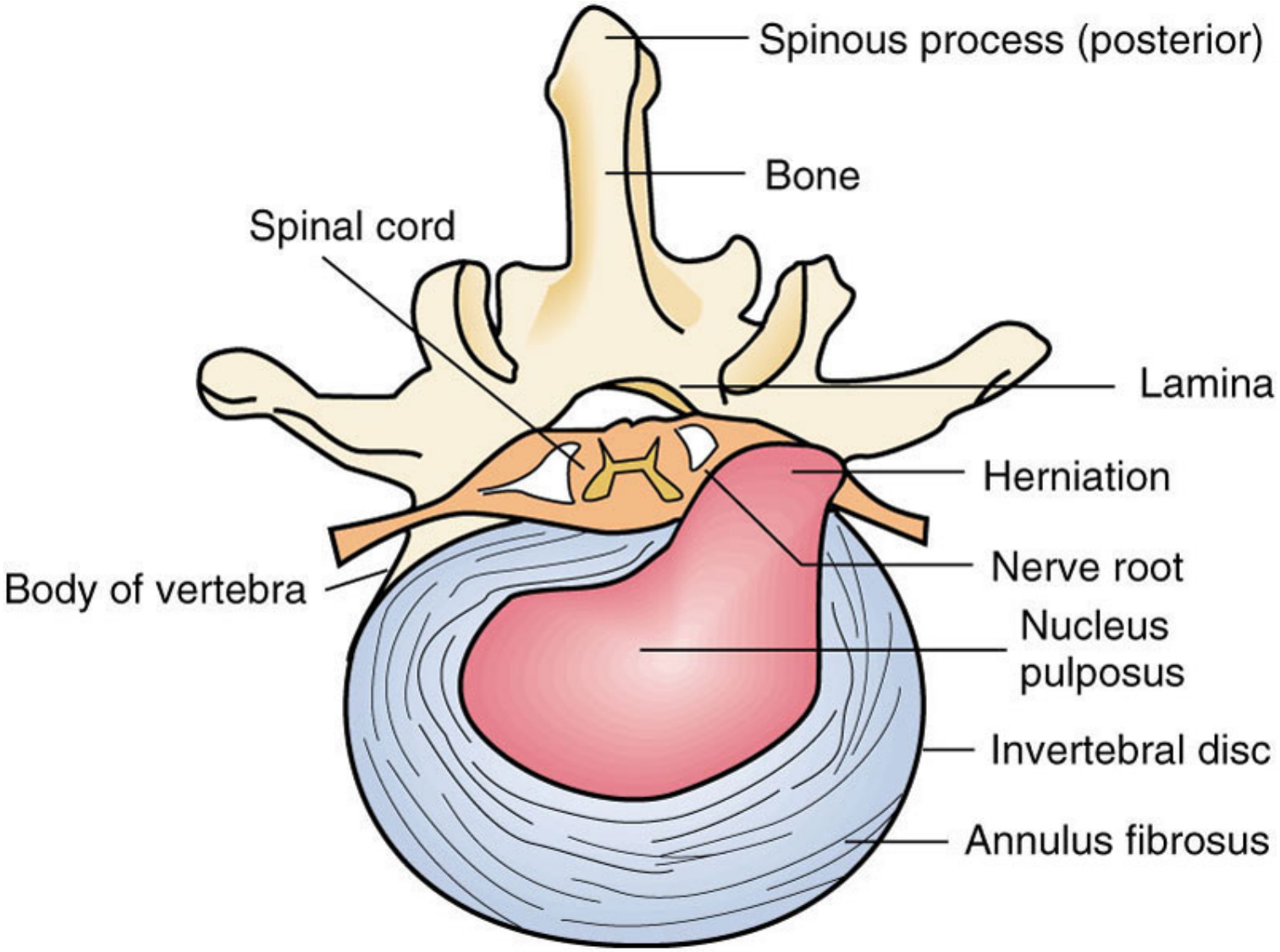
Panic Disorders

- Signs and symptoms
  - Episodes of intense fear without provocation
  - May last minutes or hours // Palpitations or tachycardia, hyperventilation, sweating, sensations of choking or smothering, nausea
- Treatment
  - Psychotherapy
  - Drug therapy // Antianxiety agents // Antidepressants may be prescribed for some.

Spinal Cord Problem

- Herniated intervertebral disk
  - Involves protrusion of the nucleus pulposus
  - Tear in capsule may occur suddenly or develop gradually with aging or obesity.
  - Sensory, motor, or autonomic function may be impaired.
  - Most common location—lumbosacral disks
  - Some herniations involve cervical disks.
  - If pressure is prolonged, severe permanent damage may occur.

Herniated Intervertebral Disk



Herniated Intervertebral Disk

- Predisposition to herniated disks
  - Degenerative changes in the intervertebral disk
    - Age
    - Metabolic changes
    - Obesity
  - Herniation usually caused by trauma or poor body mechanics
- Signs depend on location and extent of protrusion.
  - Most effects are unilateral.
  - Large protrusions may cause bilateral effects.

Herniated Intervertebral Disk

- Diagnostic tests
  - Myelography with contrast dye
  - CT, MRI
- Treatment
  - Reduced weight-bearing, rest as needed
  - Application of heat, ice, traction
  - Drugs to relieve muscle spasm and pain
  - Physiotherapy and occupational therapy
  - Surgery in severe cases // Laminectomy or diskectomy, spinal fusion