Chapter 15: Alterations of Neurologic Function – Part 1
Trauma and Cerebrovascular Disorders

I. Trauma

A. Brain Trauma

- **Major head trauma** – A traumatic insult to the brain possibly producing physical, intellectual, emotional, social, and vocational changes.

- **Causes** – most common are motor vehicle crashes, falls, sports-related events, and violence.

- The most common types of traumatic brain injury (75% to 90%) are mild concussion and classical cerebral concussion

1. Classifications
   a. **Closed trauma** (blunt, nonmissile)
      - Head strikes hard surface, or a rapidly moving object strikes the head.
      - The dura mater remains intact and brain tissues are not exposed to the environment.
      - Causes focal (local) or diffuse (general) brain injuries.

   b. **Open trauma** (penetrating, missile)
      - Injury involves a skull fracture that breaks the dura and exposes the cranial contents to the environment.
      - Causes primarily focal injuries.

   c. **Focal brain injury**
      - Specific, observable brain lesions.

   d. **Diffuse brain injury** (diffuse axonal injury)
      - Injury to neuronal axons in many areas of the brain caused by stretching and shearing forces received during brain injury.

2. Focal Brain Injury

- Force of impact typically produces contusions (in which part of the brain is bruised).

- Contusions can cause:
  - Extradural (epidural) hemorrhages or hematomas
  - Subdural hematomas
  - Intracerebral hematomas

- **Coup injury** - injury directly below the point of impact.

- **Contrecoup** - injury on the pole opposite the site of impact.

- Contusion results in brain edema and increased intracranial pressure.
  - Infarction, necrosis, and multiple hemorrhages may occur.
3. **Extradural hematomas** (epidural hematomas or hemorrhages)
   - Accumulation of blood above the dura mater next to the cranium.
   - Arise in 1% to 2% of persons with traumatic brain injury.
   - Bleeding is usually from an artery (85%) and less often from injury to the meningeal vein or dural sinus (15%).
   - Most common site - temporal fossa. The temporal lobe shifts medially, causing hippocampal and uncal herniation through the tentorial notch, compressing the brain stem (usually fatal).
   - Individuals with classic temporal extradural hematomas lose consciousness at injury, and then one third become lucid for a few minutes to a few days (if a vein is bleeding).

4. **Subdural hematomas**
   - Accumulation of blood between the dura mater and arachnoid
   - Arise in 10% to 20% of persons with traumatic brain injury.
   - Bleeding is usually from bridging veins that tear, causing either rapidly or subacutely developing subdural hematomas.
     - Act like expanding masses that increase intracranial pressure.
   - **Acute subdural hematomas** - develop rapidly (hours); usually located at the top of the skull.
   - **Subacute subdural hematomas** - develop more slowly (48 hours to 2 weeks).
   - **Chronic subdural hematomas** - develop over weeks to months; commonly found in elderly persons and persons who abuse alcohol (brain atrophy causes increase in extradural space).
     - Subdural space gradually fills with blood.

5. **Intracerebral hematomas**
   - Bleeding into the brain.
   - Arise in 2% to 3% of persons with traumatic brain injury.
   - May be single or multiple, and are associated with contusions.
   - Most commonly located in frontal and temporal lobes, but may occur in deep white matter.
   - Penetrating injury or shearing forces damage small blood vessels.
     - Intracerebral hematoma then acts as an expanding mass, increasing intracranial pressure, compressing brain tissues, and causing edema.
   - Delayed intracerebral hematomas may appear 3 to 10 days after the head injury.
   - Intracerebral hematomas cause a progressively decreasing level of consciousness.

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<thead>
<tr>
<th>ACTIVITY 1: Match the injury with its characteristic.</th>
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<tbody>
<tr>
<td>_____ 1. Bleeding into the brain tissue.</td>
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<td>_____ 2. Bruising of part of the brain.</td>
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<td>_____ 3. Usually due to arterial bleeding.</td>
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<td>_____ 4. Usually due to venous bleeding.</td>
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</table>
6. **Diffuse brain injury** (diffuse axonal injury [DAI])

- Rotational acceleration (twisting movement) is the primary mechanism of injury, producing shearing forces within the brain and tearing or stretching of nerve fibers.
- The most severe axonal injuries are located more peripheral to the brain stem, causing extensive cognitive and affective impairments.
- DAI is not associated with intracranial hypertension immediately after injury so initially they often do not suffer headache (unlike most other brain traumas).
- Over time brain edema often occurs causing increased intracranial pressure and coma.
- Symptoms include loss of consciousness, changes in respiration, and altered pupil reflexes.

**Categories of diffuse brain injury**

- **Mild concussion**
  - Temporary axonal disturbance causing attention and memory deficits but no loss of consciousness
  - Grade I: confusion, disorientation, and momentary amnesia
  - Grade II: momentary confusion and retrograde amnesia after 5-10 minutes
  - Grade III: confusion with immediate retrograde and anterograde amnesia

- **Classical concussion**
  - Grade IV (involves loss of consciousness)
  - Disconnection of cerebral systems from the brain stem and reticular activating system
  - Physiologic and neurologic dysfunction without substantial anatomic disruption
  - Loss of consciousness (<6 hours)
  - Anterograde and retrograde amnesia

- **Diffuse axonal injuries (DAI)**
  - Prolonged traumatic coma (longer than 6 hours)

- **Mild DAI**
  - Posttraumatic coma lasts 6–24 hours
  - Death uncommon
  - Persistent residual cognitive, psychologic, and sensorimotor deficits
  - Rare—only 8% of severe head injuries.

- **Moderate DAI**
  - Widespread physiologic impairment throughout the cerebral cortex and diencephalon
  - Actual tearing of axons in both hemispheres
  - Prolonged coma (longer than 24 hours)
  - Incomplete recovery among survivors
  - Common—20% of severe head injuries.
Severe DAI
- Formerly called primary brain stem injury or brain stem contusion
- Severe mechanical disruption of axons in both hemispheres, diencephalon, and brain stem
- Many exhibit reduced consciousness for a prolonged period of time.
- 16% of severe head injuries

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<tr>
<th>ACTIVITY 2: Match the injury with its characteristics.</th>
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<tr>
<td>a. Classic concussion (Grade IV)  b. Mild DAI  c. Moderate DAI  d. Severe DAI</td>
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<tr>
<td>_____ 1. Disruption of many axons in both hemispheres, diencephalon and brain stem which causes prolonged unconsciousness.</td>
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<td>_____ 2. Involves posttraumatic coma of 6-24 hours, with residual cognitive, psychologic, and sensorimotor deficits.</td>
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<td>_____ 3. Damage to axons causes loss of consciousness lasting less than 6 hours with both retrograde and anterograde amnesia.</td>
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<tr>
<td>_____ 4. Involves posttraumatic coma of more than 24 hours, with axonal damage to the cerebral hemispheres and diencephalon.</td>
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B. Spinal Cord Trauma

1. Overview
   - Most commonly occurs due to vertebral injuries.
   - Elderly are most at risk because of preexisting degenerative conditions of vertebrae.
   - Traumatic injury of vertebral and neural tissues occurs as a result of compressing, pulling, or shearing forces.
   - Most common locations: cervical (1, 2, 4-7), and T10-L2 lumbar vertebrae.
     - Locations reflect most mobile portions of vertebral column and the locations where the spinal cord occupies most of the vertebral canal.
     - Injury to the cervical spinal region is life-threatening because it interferes with the phrenic nerve which controls the diaphragm.

2. Spinal shock
   - Normal activity of the spinal cord ceases at and below the level of injury. Sites lack continuous nervous discharges from the brain.
   - Damage to the sympathetic nervous system interferes with normal thermal control causing body temperature to drop.
   - Spinal shock generally lasts 7 to 20 days, with a range of a few days to 3 months.
   - Edema of spinal tissue contributes to the loss of function.
   - As edema resolves, the spinal shock terminates with the reappearance of reflex activity, hyperreflexia, spasticity, and reflex emptying of the bladder.

3. Autonomic hyperreflexia (dysreflexia)
   - May occur after spinal shock resolves.
   - Most likely with lesions at the T6 level or above.
• Caused by massive, uncompensated cardiovascular response to stimulation of the sympathetic nervous system, including hypertension, sweating, and flushing.
• It can be stimulated by pain, touch, or often a full bladder or bowel.
• Relieved by parasympathetic stimulation such as bowel- or bladder-emptying.
• Symptoms include headache, blurred vision, hypertension, sweating, and flushing.
• Life threatening condition.

4. Spinal cord transection
• Complete loss of reflex function (skeletal, bladder, bowel, sexual function, thermal control, and autonomic control) occurs below any transected (severed) area.
• Loss of motor and sensory function depends on the level of injury.
  o Paraplegia - paralysis of the lower half of the body with both legs involved; occurs with injuries of the thoracic spinal cord.
  o Quadriplegia - paralysis involving all four extremities; occurs with injuries of the cervical spinal cord.

II. Degenerative Disorders of the Spine

A. Degenerative disk disease - an alteration in intervertebral disk tissue and can be related to normal aging.
B. Spondylolysis - a structural defect of the spine with displacement of the vertebra.
C. Spondylolisthesis - involves forward slippage of the vertebra and can involve a crack or fracture of the lamina between the superior and inferior articular facets, usually at the L5-S1 vertebra.
D. Spinal stenosis - narrowing of the spinal canal that causes pressure on the spinal nerves or cord.
E. Low back pain - pain between the lower rib cage and gluteal muscles and often radiates into the thigh.
  • Most causes of low back pain are unknown; however, some secondary causes are disk prolapse, tumors, bursitis, synovitis, degenerative joint disease, osteoporosis, fracture, inflammation, and sprain.
F. Herniation of an intervertebral disk - a protrusion of part of the nucleus pulposus.
  • Herniation most commonly affects the lumbosacral disks (L4-5 and L5-S1).
  • The extruded pulposus compresses the nerve root, causing pain that radiates along the sciatic nerve course, a condition called sciatica.

III. Cerebrovascular Disorders

A. Cerebrovascular disease - any abnormality of the blood vessels of the brain.
  • Most frequently occurring neurologic disorder.
  • Associated with two types of brain abnormalities:
    1. ischemia with or without infarction
    2. hemorrhage
B. Cerebrovascular accidents (CVA) (stroke syndromes)

- A localized brain infarction that may result in facial, arm, or leg numbness and weakness, confusion, difficulty speaking or understanding, visual disturbances, dizziness, loss of balance, difficulty walking, and headache.
- Leading cause of disability and third leading cause of death in United States.
- Classifications:
  - Global hypoperfusion (as in shock)
  - Ischemic (thrombotic, embolic)
  - Hemorrhagic
- Risk factors (very similar to those for myocardial infarction):
  - Arterial hypertension (both elevated systolic and diastolic blood pressures)
  - Smoking - increases the risk of stroke by 50%
  - Diabetes - increases the risk of ischemic stroke between 2½ and 3½ times
  - Insulin resistance - increases risk for ischemic stroke
  - Polycythemia and thrombocythemia - place the person at risk for ischemic stroke
  - Impaired cardiac function - increases risk for ischemic stroke
  - Nonrheumatic atrial fibrillation - associated with a 5-fold increase in the incidence of ischemic stroke
  - *Chlamydia pneumoniae* - can increase the risk of stroke by infiltrating and inflaming the vascular endothelium
  - Obstructive sleep apnea - increases risk for stroke independent of other risk factors
  - Increased levels of homocysteine; lipoprotein-a; lipoprotein-associated phospholipase A2 (Lp-PLa2) and C-reactive protein (indicators of inflammation) - risk factors for ischemic stroke.
- CVAs occur most frequently in those over 65, in males, and in the black population
- *Cerebral infarction* - results when an area of the brain loses its blood supply because of vascular occlusion.
- *Cerebral hemorrhage* - bleeding into brain tissue, usually due to hypertension.

1. Thrombotic stroke
   - Arterial occlusions caused by thrombi formed in arteries supplying the brain or in the intracranial vessels.
   - Cerebral thrombi often develop due to atherosclerosis, inflammation (arteritis), or increased coagulation disorders.
   - Risk is increased by conditions causing inadequate cerebral perfusion (e.g., dehydration, hypotension, prolonged vasoconstriction from malignant hypertension).
- Transient ischemic attacks (TIAs)
  - Temporary decreases in brain blood flow resulting in brief changes in brain function.
Symptoms - changes in vision, speech, motor function, or symptoms of dizziness or loss of consciousness.
Most often occur when small thrombi cause a temporary blockage of circulation.
All neurologic deficits clear completely within 24 hours.
No residual dysfunction and no permanent brain injury.
Without treatment, 80% of persons have a recurrence of symptoms by 1 year.

2. **Embolic stroke**
- Caused by fragments that break from a thrombus formed outside the brain.
- These frequently are from the heart, aorta, or common carotid artery.
- Embolus usually obstructs small brain vessels (often branches of the middle cerebral artery), causing ischemia.
- Risk factors - atrial fibrillation, myocardial infarction, endocarditis, rheumatic heart disease, valvular prostheses, atrial-septal defects, and disorders of the aorta, carotids, or vertebral-basilar circulation.
- Often a second stroke follows because the source of emboli continues to exist.

3. **Hemorrhagic stroke**
- Occurs when cerebral vessels rupture, causing bleeding into the brain tissue.
- Caused by hypertension, ruptured aneurysms or vascular malformation, bleeding into a tumor, hemorrhage associated with bleeding disorders, anticoagulation, head trauma, and illicit drug use.
  - Hypertensive hemorrhage - associated with significantly increased systolic and diastolic pressure over several years.
- A mass of blood is formed and grows, displacing and compressing adjacent brain tissue.
- Rupture or seepage into the ventricular system occurs in many cases.

4. **Lacunar stroke**
- Infarcts smaller than 1cm in diameter.
- Involve the small perforating arteries, predominantly in the basal ganglia, internal capsules, and pons.
- Associated with smoking, hypertension, and diabetes mellitus.
- Because of the subcortical location and small area of infarction, these strokes may have pure motor or pure sensory deficits.

**ACTIVITY 3:** Match the type of stroke with its description.

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<tbody>
<tr>
<td>1</td>
<td>Due to rupture of blood vessels and bleeding into brain.</td>
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<tr>
<td>2</td>
<td>Due to narrowing of cerebral or carotid vessels.</td>
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<tr>
<td>3</td>
<td>Due to blockage of small perforating arteries.</td>
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<td>4</td>
<td>Due to blockage of a cerebral vessel by fragments from elsewhere.</td>
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<tbody>
<tr>
<td></td>
<td>a. Thrombotic</td>
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<td>b. Embolic</td>
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<td>c. Hemorrhagic</td>
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<td>d. Lacunar</td>
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C. Intracranial Aneurysm
- Result from defects in the vascular wall.
- Most aneurysms are located at bifurcations in or near the circle of Willis, in the vertebral or basilar arteries, or within the carotid system.
- Classified on the basis of form and shape.
  - Saccular (berry) aneurysms - result from congenital abnormalities and degenerative changes.
  - Fusiform (giant) aneurysms - result from diffuse arteriosclerotic changes.
- They are often asymptomatic, but signs vary depending on the location and size of the aneurysm.
- Rupture results in cerebral hemorrhage, hemorrhagic stroke, and/or subarachnoid hemorrhage.

D. Arteriovenous Malformation (AVM)
- A tangled mass of dilated blood vessels.
- Although sometimes present at birth, AVM exhibits a delayed age of onset.
- With moderate to large AVMs, sufficient blood is shunted into the malformation to deprive surrounding tissue of adequate blood perfusion, causing headache.
- AVMs have abnormally thin walls which may rupture, causing intracerebral, subarachnoid, or subdural hemorrhage.
- Bleeding from an AVM into the subarachnoid space causes symptoms identical to those associated with a ruptured aneurysm.

E. Subarachnoid Hemorrhage
- Occurs when blood escapes from defective or injured vasculature into the subarachnoid space.
- Caused by head injuries, intracranial aneurysm, intracranial AVM, or hypertension.
- Blood in the cerebrospinal fluid causes meningeal irritation and inflammation.
- Acute manifestations:
  - Ruptured vessel causes a sudden, throbbing, “explosive” headache, accompanied by nausea and vomiting, visual disturbances, motor deficits, and loss of consciousness related to a dramatic rise in intracranial pressure.
  - Meningeal irritation causes neck stiffness (nuchal rigidity), photophobia, blurred vision, irritability, restlessness, and low-grade fever.
  - Kernig sign (straightening the knee with the hip and knee in a flexed position produces pain in the back and neck regions).
  - Brudzinski sign (passive flexion of the neck produces neck pain and increased rigidity).
- As hemorrhage progresses, vasospasms often occur in adjacent vessels.
  - Vasospasms cause cerebral ischemia, edema, infarcts, and often seizures.
- Rebleeding often occurs within the first month.
- Mortality in subarachnoid hemorrhage is 50% at 1 month.
**Subarachnoid Hemorrhage Classification Scale**

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
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<tbody>
<tr>
<td>Grade I</td>
<td>Neurologic status intact; mild headache, slight nuchal rigidity</td>
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<tr>
<td>Grade II</td>
<td>Neurologic deficit evidenced by cranial nerve involvement; moderate to severe headache with more pronounced meningeal signs (e.g., photophobia, nuchal rigidity)</td>
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<tr>
<td>Grade III</td>
<td>Drowsiness and confusion with or without focal neurologic deficits; pronounced meningeal signs</td>
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<tr>
<td>Grade IV</td>
<td>Stuporous with pronounced neurologic deficits (e.g., hemiparesis, dysphasia); nuchal rigidity</td>
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<td>Grade V</td>
<td>Deep coma state with decerebrate posturing and other brain stem functioning</td>
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