Neuro Review
CCRN Review
Cheryl Newton RN,MSN,CCRN,CNRN

Objectives
- Describe the essential aspects of neurological physiology and pathophysiology
- Identify the goals of neurological assessment
- Analyze the nursing care interventions for the patient with neurological diseases

Overview of the Nervous System
- Central Nervous System
  - Brain Stem
  - Spinal Cord
- Peripheral Nervous System
  - Cranial Nerves (12 pairs)
  - Spinal Nerves (31 pairs)
  - Somatic NS (sensory)
  - Autonomic NS (motor)

Anatomy of the Brain
- Cerebral Hemispheres
  - Cerebral dominance: right/left
  - Diencephalon
  - Major division of cerebrum
  - 4 regions
  - Brain Stem
  - Cerebellum
    - Balance
    - Coordination
- Cerebrum
  - Two cerebral hemispheres
  - Broca's speech area
  - Basal ganglia
    - Provides circuitry for conscious bodily movements, motor control, and fine body movements

General Overview of the Brain
The Brain

- 4 lobes of cerebral hemispheres
- Basal ganglia
- Limbic system
- Diencephalon
- Cerabellum
- Brain stem

Lobes of the Brain

- Frontal
  - Higher mental function, speech, Broca’s area (articulate speech)
- Parietal
  - Sensory area, body awareness
- Temporal
  - Sound, Wernicke’s area (spoken word and written language)
- Occipital
  - Visual, integrates visual stimulation

Diencephalon

- Thalamus
  - Sensory and motor relay
- Hypothalamus
  - Neuro-endocrine function
  - Visceral, autonomic, endocrine, and emotional functions
  - Temperature, appetite, ADH, sleep
- Pituitary gland
  - Secretes and stores hormones

Brain Stem

- Second major division of the brain
  - Midbrain
  - Pons
  - Medulla
- Contains respiratory and autonomic control centers
- Coordinates activity of the cerebellum and rest of the brain
- 10 cranial nerves originate from brainstem
  - CN III oculomotor, pupillary constriction
  - CN X Vagus: PNS motor
Brain Stem
- **Midbrain (III, IV)**
  - Motor and sensory pathways
  - Location of reticular activating system
  - Responsible arousal from sleep, wakefulness, focusing of attention
- **Pons (V, VI, VII)**
  - Connects cerebral cortex and cerebellum
  - Contains motor and sensory pathways
  - Contains respiratory center

Brain Stem
- **Medulla oblongata (VIII, IX, X, XI, XII)**
  - Connects motor and sensory tracts of spinal cord to medulla
  - Contains cardiac and respiratory centers
  - Continuous with spinal cord

Cerebellum
- **Cerebellum**
  - Coordinates muscle movement with sensory input
  - Controls balance and equilibrium
  - Influences muscle tone
  - Affects locomotion and posture
  - Controls non-stereotyped movements
  - Synchronizes muscle action

Spinal Cord
- **Spinal Cord--Level of Injury**
- **Spinal Cord**
  - Complex intricate system that communicates motor and sensory impulses from the brain to body; and body to brain
  - Tracts (groups of Neuron Fibers)
    - Spinothalamic tract
    - Corticospinal tract
Meningeal Layers

- 3 layers of connective tissue covering the brain
- **Dura Mater** (Outer layer)
  - Lines interior of skull
- **Arachnoid membrane** (middle)
  - Thin delicate layer that loosely encloses the brain
  - CSF flows in the subarachnoid space
- **Pia Mater** (inner)
  - Mesh-like vascular membrane that covers the entire surface of the brain

Cerebrospinal Fluid Characteristics

- Clear and colorless
- 500 mls/day
- Composed of water, small protein, O₂, and CO₂
- Composed of electrolytes: Na, K, Cl, and Glu
- Pathology: RBCs, WBCs, ↑Prot, ↓Glu

Cerebrospinal Fluid Functions

- Flows within ventricles, subarachnoid space and spinal cord
- Supports and cushions the brain and spinal cord
- Decreases the effective brain weight
- Acts as medium for the transfer of substances b/t the blood and nervous tissue

Normal CSF Profile Values

- **pH**: 7.35-7.45
- **Specific gravity**:
- **Appearance**: Clear and colorless
- **Cells**: 0 WBC's, 0 RBC's, 1-10 Lymphocytes
- **Glucose**: 50-75 mg/dL
- **Protein**: 5-25 mg/dL
- **Volume**: 135-150 mls
- **Pressure**: 3-15 mmHg
Internal Carotid Arteries
- Frontal Lobes
- Parietal Lobes
- “legs” areas

Middle Cerebral
- Anterior 2/3 of frontal, temporal, parietal lobes

Anterior Communicating
- Connects two Anterior Cerebral A.
- Primarily Frontal lobes

Vertebral Arteries
- Basilar Artery
  - Formed by 2 vertebral Arteries joining
  - Feeds Brain stem
- Posterior Cerebral arteries
  - Occipital
  - Part of temporal lobe
- Posterior Communicating
  - Thalamus
  - Hypothalamus
  - Optic tracts

Venous Circulation
- Venous Sinuses
  - Drain via the internal jugular system
- Ventricular system
  - R and L ventricles
  - 3rd and 4th ventricles
- Blood Brain Barrier
  - No barrier, pineal region, basal hypothalamus, floor of the 4th ventricle

Blood Brain Barrier
- Regulates and maintains the internal environment
- Protects cells via selective permeability
- Highly permeable to water, CO2, O2, and lipid soluble substances
- Restricts large molecules
  - Heavy metals, some antibiotics, toxins
- No barrier:
  - Pineal region
  - Basal hypothalamus
  - The floor of the fourth ventricle
Concepts of Neuro-Dynamics

- Cerebral Blood Flow (CBF)
- Intracranial Volume
- Pressure
- Compliance
- Elastance
- Autoregulation

Cerebral Blood Flow

- Components are blood and CSF
- Flow rate 50-60 mL/100g brain tissue
- 750 ml/min
- Regulated by baroreceptors and chemoreceptors

Intracranial Volume

- Volume of the cranial vault is:
  - Brain Mass: 80%
  - CSF: 10%
  - Blood Volume: 10%

Monro-Kellie Doctrine

- Within the fixed non-distensible skull of the adult, intracranial volume must remain constant to maintain a normal ICP

Monro-Kellie Doctrine

- An increase in volume of one compartment must be offset by a decrease in another compartment to keep ICP normal
- This is called compensation

Intracranial Pressure

- Normal ICP = 0-15 mmHg
- Intracranial HTN is considered an ICP >20 mm Hg
- Cerebral Perfusion Pressure
  - Pressure at which the brain cells are perfused
  - CPP = MAP - ICP
Cerebral Perfusion Pressure

- Normal CPP: 60 – 95 mmHg
- Ischemia: 50 – 60 mmHg
- Cell Death: < 30 mmHg
- CBF ceases: 0 mmHg

Autoregulation

- Brain’s ability to maintain a constant rate of CBF despite variations in systemic arterial pressure
  - CBF=blood +CSF
    - 50-60ml/100gms of brain tissue
    - 750ml/min
  - When autoregulation is intact: CBF and cerebral blood volume are independent of MAP, CO, and body activity

Compliance

- Refers to the brain’s ability to tolerate increases in volume without sustaining an increase in pressure
  - C = change in volume/change in pressure

Compliance

- Normal ICP: Compliance is high
- When C is high: a change in volume results in a small change in pressure
- When C is low: a small change in volume results in a large change in pressure

Causes of Increased ICP

- Increase in tissue volume
  - Tumors
  - Abscess
  - Edema
- Increase in blood volume
  - Hemorrhage
  - Decreased venous return
- Increase CSF volume
  - Obstruction
  - Overproduction
  - Decreased absorption

Compensatory Mechanisms

- Displacement of CSF to the Spinal SAS
- Increased absorption of CSF
- Decreased production of CSF
- Displacement of blood volume
  - Venous blood is shunted away from affected area into the distant venous sinuses
- Displacement of brain tissue
  - Shift of brain mass
Cerebrospinal Fluid (CSF)

- Functions
  - Cushions brain and spinal cord
  - Compensation for changes in ICP
- Properties
  - Clear, colorless, odorless
  - Xanthochromic = yellow = old RBC
  - Volume 120 – 150 ml in ventricular system and SAS
  - Specific gravity: 1.007
  - Glucose: 60% of serum glucose level
- Most CSF is contained in the spinal cord

Cerebrospinal Fluid

- CSF production and reabsorption
  - Formed by choroid plexus in ventricles
  - 95% is produced in the lateral ventricles
  - Absorbed via arachnoid villi

Ventricular System

- Communication system within brain
- Ventricles
  - Contain specialized epithelium called choroid plexus
  - Produce CSF
  - Lateral ventricles are the largest
  - The 3rd ventricle lies midline between the 2 lateral ventricles
  - 4th ventricle lies in posterior fossa

Categories of Assessment

- Glasgow Coma Scale (3-15)
  - Eye opening (1-4)
  - Verbal response (1-5)
  - Best motor response (1-6)
- Muscle strength
  - Grade 1, 2, 3, 4, 5
- Reflexes (+ or -)
  - Babinski’s
  - DTRs

Homonculus-sensory

Homonculus-motor
Cranial Nerves

I. Olfactory  Smell
II. Optic  Visual
III. Oculomotor  Eye movement
IV. Trochlear  Extra Eye Movement
V. Trigeminal  Mastication
VI. Abducens  Extra Eye Movement
VII. Facial  Facial Muscle
VIII. Acoustic  Hearing
IX. Glossopharyngeal  Swallowing
X. Vagus  Phonation
XI. Spinal Accessory  Shoulders
XII. Hypoglossal  Tongue movement

Neuro Assessment

Level of Consciousness
- Alert
- Confused
- Delirious
- Lethargic- increased drowsiness
- Obtunded- indifference to stimuli
- Stuporous- requires vigorous stimuli
- Comatose

Increased ICP: Clinical Presentation

>15 mmHg
Change in LOC
- Early – subtle and transient
  - restlessness, confusion, HA, drowsiness
- Late – diminishing LOC, posturing
  - Bradycardia, abnormal respiratory patterns, vomiting

Clinical Presentation

Cranial Nerve changes
- Oculomotor (III)
  - Early –
    - Ipsilateral pupil changes
    - Change in size, shape (oval)
    - Sluggish reaction to light
    - Conjugate eye deviation
  - Late
    - Ipsilateral pupil changes
    - Dilated, non-reactive
    - Ptosis
    - Disconjugate eye movement with brainstem lesions

Optic (II)
- Visual changes
  - Diplopia
  - Blurring
  - Decreased visual acuity
  - Visual field deficit

Trigeminal (V)
- Impaired corneal reflex

Glossopharyngeal (IX) and vagus (X)
- Impaired gag and swallow reflexes
Clinical Presentation

- **Motor changes**
  - **Contralateral**
    - Due to compression or pressure on the corticospinal tracts
      - Early
        - Paresis, plegia
      - Late
        - Posturing

- **Vomiting**
  - May occur
  - Pressure on the vomiting center in the brainstem causes projectile vomiting without nausea

- **Headache**
  - Increasing severity but inconsistent symptom

- **Seizure**
  - May occur

Clinical Presentation

- **Vomiting**
  - May occur
  - Pressure on the vomiting center in the brainstem causes projectile vomiting without nausea

- **Headache**
  - Increasing severity but inconsistent symptom

- **Seizure**
  - May occur

Clinical Presentation

- **Vital sign changes**
  - Cushing’s triad
    - Due to pressure on or ischemia of vasomotor center in brainstem
      - **Components**
        - Increased SBP
        - Widening pulse pressure (DBP normal or decreased)
        - Bradycardia
      - Respiratory pattern
        - Dependent on location of injury
      - Temperature
        - Central hyperthermia may occur late r/t pressure on thermoregulatory center in hypothalamus

Increased ICP - Monitoring

- **Early recognition**
  - With ICP monitoring, intracranial problems can be identified and treatment initiated before clinical signs and symptoms develop

- **Treatment can be evaluated**
  - Allows monitoring of post-op cerebral edema, infections, or following intracerebral hemorrhage

- **ICP can be measured**
  - Intraventricular cannula - Ventriventriculoscopy
  - Intraparenchymal catheter or bolt - Camino

Management of Increased ICP

- **Goal is to prevent ischemia and herniation**

- **Maintain Fluid**
  - Improving MAP to normalize CPP
  - Fluids may be limited to keep serum osmolality 290-320
  - Hypotonic solutions avoided: No D5W
  - 3% NS (hypertonic saline)
  - Fluids not restricted if vasospasm an issue

- **Osmotic diuretic therapy**
  - Mannitol and loop diuretics may help decrease the ICP
  - Draw fluid out of swollen brain
  - CSF drain via ventriculoscopy

Management of Increased ICP

- **Keep CPP > 60**

- **Promote hemodynamic stability**

- **Control pain and agitation**

- **Prevent seizure**

- **Barbiturate coma**
  - May be tried if motor responses such as hemiparesis or decorticate or decerebrate positioning occur as a result of cortical and midbrain compression of motor tracts.
Nursing Interventions

- Good neuro assessment for early identification of increased ICP
- Transient increases in ICP may be seen with
  - Confusion, difficulty in arousal, sluggishness, slight pupillary dilatation, monoplegia or hemiparesis, H/A, aphasia, Cheyne-Stokes respirations or change in VS.

Nursing Interventions

- Most nursing procedures have an affect on the ICP
  - Turning the pt may increase ICP, but can be lessened if the pt is log-rolled with the head in alignment.
  - If 2 nursing actions both increase ICP, space the actions to allow the ICP to diminish
  - Suctioning
    - Increase in ICP may be decreased by pre-oxygenating, limiting suctioning to 10 sec, 1 or 2 passes, and use of less than 120 mm Hg pressure.

Nursing Interventions

- Careful monitoring/corrections of electrolytes and osmolality
- Monitor for infections and treat fever
  - Fever increases cerebral metabolism and may increase the ICP
  - Use of hypothermia blankets
- Maintaining HOB elevation of 30 degrees to promote venous return from the head
- Maintain head in neutral position

Nursing Interventions

- Maintain a quiet environment
- Provide adequate pain medications
- Dexamethasone
  - Decreases cerebral edema
- Propranolol
  - Decreases metabolic rate/demand
- Barbiturate Coma
  - Decreases metabolic demand

Traumatic Brain Injury (TBI)

- Concussion, contusion, epidural hematoma, SDH, SAH, IVH, seizure
- Etiology: falls and MVCs
- DX: CT and MRI
- Injury Severity Score (GCS)

TBI

- Injury Severity Score (GCS)
  - 13-15 mild injury
  - 9-12 moderate
  - <=8 severe
- Primary Injury
  - Happens at the time of the trauma
- Secondary Injury
  - Inflammatory response
  - Electrolyte imbalances
  - Hypotension, hypoxia
Closed Head Injuries (CHI)
- Damage occurs to the cranium without causing a break in the skull
- Commonly result from MVC, falls, and assaults
- Alcohol involved

CHI
- Concussion
  - Temporary neural dysfunction
  - HA, dizziness, irritability, nausea
  - Transient LOC, post-traumatic amnesia
  - Contusion

CHI
- Contusion
  - More severe
  - Bruising of the brain surface, causing neuronal dysfunction
  - HA, vomiting, restlessness, decreased LOC, aphasia, hemiplegia, sensory deficits

CHI
- Laceration
  - Traumatic tear of the cortical surface of the brain
- Diffuse Axonal Injury (DAI)
  - Extensive damage to the axons in the white matter
  - Occurs from high velocity acceleration-deceleration accidents
- Hypoxic Injury
  - Oxygen deficit and injury (metabolic or cardiac)

Management of TBI/CHI
- Perfuse the brain
  - CPP>65, SBP>90, HOB>30 degrees
- Prevent secondary injury
  - Neuro checks
  - Osmotic diuretics (Mannitol), electrolyte replacement
  - Decrease brain swelling
  - Steroids
  - Minimize cerebral edema
- Prevent complications
  - SCDs for DVT prophylaxis
  - Antacids and histamine blockers
  - Stool softeners
  - Prevent Valsalva

Skull Fractures
- Disruption in the integrity of the cranial vault
- Blunt or penetrating trauma
- Symptoms include
  - HA, pain at the site, altered LOC, sensory deficits
- 3 types
  - Basilar
  - Depressed
  - Linear
Skull Fractures

- Basilar
  - Base of the skull
  - Paranasal sinuses involvement
    - Rhinorrhea, periorbital ecchymosis (raccoon eyes)
    - Otorrhea, vertigo, nystagmus, and ecchymosis over mastoid bone (Battle’s sign)
- Depressed
  - Inward displacement of the outer skull
  - Can occur with open or closed injuries
- Linear
  - Most common type of skull fracture
  - No displacement of bone

Clinical Manifestations

- Based on location and type of fracture
  - Rhinorrhea
  - Otorrhea
  - Vertigo
  - HA
  - Nystagmus
  - Pain at fracture site
  - Change in consciousness

Management

- Relieving pressure
- Relieving ischemia
- Preventing complications
  - Internal carotid artery injury and CSF leaks are associated with basilar fractures
  - Infections are associated with depressed fractures
  - Hemiation from increased ICP
  - Hemorrhages

Nursing Interventions

- Neuro assessment
- Monitor for signs of infection
- Prevent secondary injury

Intracranial Hematomas

- Intracranial hematomas are hemorrhages that produce bleeding into the epidural, subdural, subarachnoid space, or the ventricles
- May occur spontaneously or as result of trauma
- Up to 15% of all head injuries are SDH

Intracranial Hematomas

- Etiology: Usually trauma
- Types
  - Subdural (SDH)
    - May be spontaneous
  - Epidural (EDH)
  - Associated with linear skull fx
  - Intracerebral (ICH)
    - Result of GSW or stab, laceration of brain from depressed skull fx, severe acceleration / deceleration injury
    - Bleeding from aneurysm, AV malformation, tumor, rupture of vessel r/t HTN
**Subdural Hematoma (SDH)**
- Usually venous, forms slowly, cerebral contusion
- Accumulates below dura mater
- **Classification**
  - **Acute**
    - Clinical indication occur within 48 hours after injury
  - **Subacute**
    - Clinical indication occur within 2 weeks after injury
  - **Chronic**
    - Clinical indication occur weeks to months after injury

**Epidural Hematoma**
- Usually arterial, may be associated with tearing of arteries from skull fractures
  - Middle meningeal artery
- May be due to venous bleeding, associated with fractures that cross major vascular channels (superior sagittal or transverse sinus)
- Accumulates above dura mater

**Intracerebral (ICH)**
- SAH or IVH
- Hematoma into brain parenchyma
- May be due to bleeding caused by missile injury or severe acceleration / deceleration force

**Subarachnoid Hemorrhage**
- SAH
- Bleeding into the subarachnoid space
- Common in severe head injuries

**Arteriovenous Malformations**
- Abnormal vascular network
- AVMs are a tangle mass of dilated vessels
- AVMs are congenital lesions that develop in the 4th to 8th week of embryonic life
- Most AVMs do not become symptomatic until the 3rd decade of life

**Arteriovenous Malformations: Signs and Symptoms**
- Hemorrhage is the most common presenting symptom
- Seizures
- Headache, nausea, vomiting
- Hydrocephalus (uncommon)
Arteriovenous Malformations
- Steal phenomenon: steal from the adjacent brain tissue
- Blood is diverted from one area of brain to another due to lower vascularization
- Occurs when AVM is large
- Symptoms: seizure, HA

Arteriovenous Malformations: Diagnostic Studies
- Cerebral angiography: most definitive study reveals the feeding vessels and the draining vessels, size and location
- CT scan
- MRI

Treatment
- Surgery, embolization, Gamma knife
- Supportive Care
- Prevent secondary injury
  - Nutrition
  - Skin care
  - Ulcer prophylaxis
  - DVT prophylaxis

Cerebral Aneurysms
- Dilatation of a cerebral artery resulting from weakness in the arterial wall
- 95% of all aneurysms occur close to the Circle of Willis at the bifurcations of the internal carotid, middle cerebral, and basilar arteries, and in relation to the anterior and posterior communicating arteries.

Aneurysms
- Most occur in the vessels of the anterior portion of the circle of Willis
- About 20% will have multiple aneurysms
- Rupture of an aneurysm cause SAH in more than 95% of patients
- Increased ICP, hydrocephalus and vasospasm can occur especially with worsening clinical grade

Types of aneurysms
- Berry-most common
  - Looks like berries
- Saccular
  - Out pouching from one wall
- Fusiform
- Diffuse enlargement
- Charcot-Bouchard
  - Brain stem, related to HTN
- Mycotic-rare
  - Septic emboli/endocarditis
Etiology
- Hemodynamic factors, arteriosclerosis, and breakdown of the internal elastic membrane contribute to the formation and growth of aneurysms
- HTN not a major factor, genetics is
  - Familial, Marfan's, PCKD, neurofibromatosis
- Asymptomatic until aneurysm ruptures
- Associated with AVMs

Diagnostic Testing
- CT scan
- MRI
- MRA
- Angiography
- Transcranial doppler

Classification of Aneurysms
- Determines the severity of the rupture
- Hunt and Hess
  - Index of surgical risk
- Fisher Classification
  - Index of spasm risk
- GCS

Hunt and Hess
- Grade 0: Unruptured aneurysm
- Grade I: Asymptomatic or slightly symptomatic
- Grade II: Moderate to severe HA, nuchal rigidity, third nerve palsy
- Grade III: Drowsiness, confusion or mild focal deficits
- Grade IV: Stupor, moderate to severe hemiparesis and posturing
- Grade V: Deep coma, decerebrate posturing

Aneurysm Treatment
- Ventriculostomy placement
- Clipping (OR for surgery)
- Endovascular Coiling
- Monitor for complications
  - Infections
  - Vasospasm
  - Hyponatremia
- Monitor and treat electrolytes

Vasospasm
- Commonly occurs between 3 and 14 days after rupture (up to 21 days)
- Peak incidence between day 5 and 9
- The incidence and degree of vasospasm is related to the amount of blood in the subarachnoid space
- DX clinically, TCD, angiography
Treatment of Vasospasm
Calcium channel blockers such as oral Nimodipine. Usual dose: 60 mg q 4 hours.
- Adjust dose for BP
- Watch for hypotension
- Hypervolemia, hemodilution, and hypertension (Triple H therapy)

Triple H Therapy
- Hemodilution and hypervolemia are usually accomplished with a combination of colloid (5% albumin) and crystalloid solutions.
- Objectives:
  - Decrease hematocrit by 30%
  - Increase cardiac output to 6.5-8 L/min
  - CVP 8-10 mmHg
  - PAWP 12-16 mmHg

Vasospasm
- Can also use Vasopressors if triple H therapy does not increase blood pressure and CPP to desired range
  - Dopamine
  - Phenylephrine
  - Norepinephrine

Cerebrovascular Disease
- Third leading cause of morbidity & mortality
- AHA efforts to educate community and health care providers.....TIME IS TISSUE...“brain attack”
- Interventions within 3 hours of onset of signs and symptoms may reverse the cerebral ischemia

Cerebral Vascular Disease
- Sudden or rapid onset of focal Neuro deficits which may be temporary or permanent
- Acute impairment of blood flow → Neuro deficits
- TIA---- RINDS---- CVA
- CVA
  - Ischemic (thrombotic, embolic)—85%
  - Hemorrhagic (intracerebral, subarachnoid)—15%
**Transischemic Attack (TIA)**

- A temporary disruption in blood flow due to spasm or microemboli
- Brief neurological deficit lasting minutes to hours
- Symptoms depend on vessel affected
  - R cerebral hemisphere
    - L hemianopia, hemiparesis, hemiplegia
  - L cerebral hemisphere
    - R hemianopia, hemiparesis, hemiplegia, and aphasia
  - Expressive, receptive, or global

**Cerebrovascular Accident**

- Neurological deficit that is the result of damage or disease in the cerebral blood vessels
- An area of the brain becomes hypoxic due to edema or an obstructed vessel
- Deficits depend on the area affected
- Thrombosis, embolism, ICH from ruptured blood vessel (middle cerebral artery)

**Cerebrovascular Accident (CVA) or Stroke**

- Thrombotic
  - Most common
  - HTN is most significant factor
  - DM, CAD, increasing age, men>women
- Embolic
  - Cardiac in origin
  - Endocarditis, a-fib, valve abnormalities
- Hemorrhagic (ICH)
  - Chronic HTN, vascular malformations, coagulopathies, alcohol use, and tumors
  - Sympathomimetic drugs
    - Cocaine and amphetamines

**Symptoms**

- Numbness or weakness
- Confusion
- Trouble speaking or understanding
- Visual disturbances
- Sudden severe HA
- Difficulty walking, loss of balance, dizziness

**Stroke Management**

- Time is brain tissue
- Blood pressure control
  - Thrombotic therapy (rt-PA)
    - SBP<185, DBP<110
      - Decrease risk of bleeding
  - Non-thrombotic
    - SBP up to 210, DBP up to 110
      - Adequate perfusion
- Carotid stenosis >70% should be treated
- Supportive therapy for ICH

**Hemorrhagic Stroke**

- Hemorrhage
  - 25% of strokes
  - Bleeding into parenchyma, causing irritation of and pressure on cerebral tissues and nerves, leading to loss of fx and death of neurons.
  - HTN intracranial hemorrhage usually occurs in the basal ganglia, cerebellum or brain stem but may affect more superficial areas of the cerebrum.
Hemorrhagic
- Intracerebral
  - Trauma
  - Hypertensive rupture of vessel
  - Vascular intracerebral tumor, thrombolytics, anticoagulants, bleeding disorders
- Subarachnoid
  - Cerebral aneurysm
    - Accounts for majority of SAH
    - Most are small
  - Weakened bulging area on intracranial blood vessel
  - AV malformation
    - Tangle of abnormal arteries and veins, artery feeds into vein directly without capillary bed
    - Always congenital
    - Causes ischemia of surrounding tissue

Risk Factors for SAH
- Smoking
- Hypertension
- Moderate to heavy alcohol use

Presentation of SAH
- Abrupt HA, localized
- Usually occurs at night
- Worst HA of my life
- Associated with nausea, vomiting, sz

Management
- SAH
  - ID vasospasm by worsening of neuro status
  - Vasospasm occurs anytime from the 3rd day post bleed to 2-3 weeks after initial bleed
  - Administer calcium channel blockers
  - Maintain triple-H therapy (HTN, hypervolemia, hemodilution)
    - HTN – goal BP
    - Vasodilators
    - Vasopressors
    - Hypervolemia and hemodilution
      - Colloids and crystalloids
      - Goal PAOP
      - Goal Hematocrit

Brain Tumors
- Act as a space occupying lesion
- Tumors are classified by histological features and grade of malignancy
- Prognosis is based on histological type, grade of tumor, location, size, the patient’s age, clinical status prior to surgery

Minimize potential for rebleed and promote stabilization of patient
- Rebleed occurs most often within 24 hours or a 7-10 days after bleed
Types of Brain Tumors

- **Gliomas**
  - Astrocytoma: usually grades I to II
  - Anaplastic astrocytoma
  - Cerebellar astrocytoma
  - Optic nerve glioma
  - Oligodendroglioma: rare slow growing

- **Glioblastoma Multiforme**: 50% of primary brain tumors
- **Brain Stem Glioma**: poor prognosis
- **Ependymoma**: arise from cells lining ventricles
- **Medulloblastoma**: highly malignant
- **Pineal region tumor**: rare

Extra-Axial Tumors

- **Meningioma**
  - Make up 15-20% off all primary BT
  - Arise from meningeal tissue
  - Encapsulated
  - Prognosis: usually good
- **Acoustic Neuroma**
  - 10% of all primary brain tumors
  - Arises from CN VIII
  - Slow growing and benign
  - Affects CN V, IX, X

Pituitary Tumors

- **Make up 10-15% of all primary BTs**
- **Arise from pituitary gland**
- **Technically an endocrine tumor**
- **Secreting vs non-secretting**
  - Non-secretting:
    - 90% of all pituitary tumors
    - Compression of optic chiasm
    - Visual changes
  - Secreting:
    - Prolactin secreting
    - Growth hormone
    - ACTH secreting

Signs and Symptoms

- **Depends on location of tumor and how rapidly the tumor grows**
  - Headache
  - Seizures
  - Visual changes
  - Mental status changes
  - Vomiting

Signs and Symptoms: Frontal Lobe Tumors

- Inappropriate behaviors
- Inattentiveness
- Inability to concentrate
- Loss of self restraint
- Impaired recent memory
- Flat affect, apathy
- Expressive aphasia
- Motor weakness (usually contralateral hemiparesis)
Signs and Symptoms: Parietal Lobe Tumors
- Hyperesthesia
- Paresthesia: tingling, burning, crawling
- Loss of two point discrimination
- Unilateral neglect

Signs and Symptoms: Temporal Lobe Tumors
- Psychomotor seizures
- Homonymous hemianopsia
- Receptive aphasia
- Alterations in hearing

Signs and Symptoms: Occipital Lobe Tumors
- Contralateral homonymous hemianopsia
- Visual hallucinations
- Seizures with an aura

Signs and Symptoms: Pituitary Tumors
- Visual defects
- Hypopituitarism
- Headache
- Cushing syndrome
- Acromegaly
- Endocrine dysfunction
- Menstrual dysfunction

Signs and Symptoms: Ventricular Tumors
- Hydrocephalus
- Headache
- Change in LOC

Signs and Symptoms: Cerebellar Tumors
- Ataxia
- Dizziness
- Nystagmus
Signs and Symptoms: Brain Stem Tumors
- Cranial nerve deficits
- Cerebellar dysfunction
- Vomiting
- Obstructive hydrocephalus

Diagnostic Studies
- CT scan
- Skull films
- Cerebral angiography
- MRI

Treatment
- Craniotomy
- Radiation Therapy
- Chemotherapy

Seizures
- Abnormal electrical discharge of cerebral neurons
- Sensory, motor, or behavioral changes, change in LOC
- Ictal period- lethargic and disoriented

Seizures
- Generalized
  - Absence—sudden lapse of consciousness—staring spell
  - Myoclonic—sudden, brief muscle jerking on one or more muscle groups. Commonly associated with metabolic, degenerative, and hypoxic causes.
  - Atonic—sudden loss of muscle tone.
  - Clonic—rhythmic muscle jerking.
  - Tonic—sustained muscle contraction.
  - Tonic-clonic—muscle activity varies between sustained contraction and jerking.

Seizures
- Partial
  - Occurs when there is a focal discharge in one area of the cerebral cortex.
  - Simple and complex
- Epilepsy = recurrent seizures
- Status epilepticus = continuous or repetitive seizures > 30 min
Seizure Management
- Controlling the seizure as quickly as possible, preventing recurrence, maintaining patient safety, and identifying the underlying cause!
- Position patient on side to decrease aspiration
- Provide supplemental oxygen
- Nothing should be placed in the patient’s mouth during a seizure.
- EKG monitoring, pulse oximetry, and blood pressure monitoring are required in patients with prolonged seizures.
- Hypoglycemia can induce seizure activity, so a glucose level is checked immediately and treated as appropriate.

Lorazepam 0.1 mg/Kg
Phenytoin SLOW IV
  - no more than 50 mg over 1 min
  - May cause cardiac dysrhythmias
  - Must check blood level
  - Free DPH level is more accurate
Phenytoin PO
  - TF must be stopped 1 hr before and 2 hrs after the dose

Encephalitis
- Inflammation of the brain parenchyma
- Most commonly due to Herpes Simplex Virus
- Symptoms
  - Fever, neurologic changes, HA, SZ, stiff neck

Meningitis
- Acute inflammation of the meninges of the brain and spinal cord
- Can be caused by bacteria, viruses, fungi, or parasites
- Risk factors: immunocompromise, trauma or surgery, crowded living conditions

Meningitis Symptoms
- Fever
- Headache
- Neck stiffness
- Irritability
- Vomiting
- Photophobia
- Change in LOC
- Seizures
- Weakness
- Cranial nerve signs

Meningitis Diagnosis
- LP for pressure and CSF analysis
- Blood Cultures
- Sputum/ nasopharyngeal cultures
- EEG
- Head CT

Complications of Meningitis:
- Hydrocephalus
- Cerebral Edema
- Vasculitis
Kernig’s Sign
- Patient can not extend leg at the knee when the thigh is flexed because of stiffness of the hamstring muscle

Brudzinski’s sign
- Have patient attempt to touch their chin to their chest
- Chin to chest causes patient’s hips to flex when the neck is flexed

Meningitis Diagnosis

Spinal Cord Injury
- Results from:
  - Compression
  - Contusion
  - Trans-section of spinal cord
- Etiology
  - Trauma
  - Disease processes
    - Tumors
    - Ruptured spinal AV malformation
    - Infectious process
    - Abscess
    - Hematoma

Mechanism of Injury: Trauma
- Hyperflexion
  - Chin forced to chest
  - Occurs most often in cervical region
  - Results from
    - Sudden deceleration
    - Ligament tears, stretching of spinal cord
    - Dislocated or subluxation of intervertebral disks

Mechanism of Injury: Trauma
- Hyperextension – whiplash
  - Head thrown back
  - Occurs most often in cervical region
  - Forces of acceleration-deceleration
  - Results in backward and downward movement, stretching spinal cord
- Rotation injury
  - Spinal cord is rotated
  - Can involve all parts of vertebral column
  - Tearing spinal ligaments
  - Displacement of intervertebral disks, compression of spinal nerve roots
Mechanism of Injury: Trauma

- Vertical compression
  - Vertebral column is compressed
  - Occurs primarily in area of T12 – L2
  - Force applied downward from head
  - Burst vertebra and intervertebral disks

Mechanism of Injury: Trauma

- Penetrating trauma
  - Can occur at any level
  - Results from a penetrating object
  - Can be complete or incomplete trans-section of the spinal cord

Clinical Presentation

- Depends on type and extent of lesion
- Subjective
  - Hx of precipitating event
- Objective
  - Level of lesion
  - Complete versus incomplete
    - Complete – loss of sensory and motor fx below level of lesion, irreversible
    - Incomplete –
  - Spinal shock
    - Results from loss of inhibition of descending tracts

Incomplete SCI

- Central Cord Syndrome
  - Weakness in both upper extremities and lower extremities
  - Weakness is greater in upper extremities

Incomplete SCI

- Anterior Cord Syndrome
  - Anterior paralysis
  - Decreased sensation
  - Decreased pain
  - Position, touch, vibration remain intact

Incomplete SCI

- Brown-Sequard
  - ½ spinal cord is affected
  - Same side→loss of motor, position, and vibration
  - Opposite side→loss of pain and temperature sensation
Incomplete SCI

- Cauda Equina Syndrome
  - Injury to nerve roots below L1
  - Lower extremity motor loss
  - Loss of bowel and bladder function
  - Areflexia

Spinal Shock

- May last days to months
- Loss of all motor, sensory and reflex responses
- Bradycardia and hypotension
- Loss of autonomic control
- Transient reflex depression below level of injury
- Flaccid paralysis below level of injury
- Paralytic ileus
- Urinary and fecal retention
- Impaired temperature regulation
- Priapism may occur

Diagnostic

- ABGs – evaluation ventilatory and oxygenation status
- Spinal x-rays
- EMG
- CT
  - Visualize fractures
- MRI
  - ID extent of damage, degree of cord contusion, presence of blood, edema necrosis, disk herniation, tumor

Management

- Immediate immobilization
  - Cervical traction with Gardner-Wells or Crutchfield tongs inserted in to skull’s outer table with traction applied
  - Halo traction
  - Surgery
    - Decompression laminectomy
    - Closed or open fracture reduction
    - Cervical spinal fusion
    - Harrington rods – thoracic injury
    - Log roll

Management

- Prevent further spinal cord edema
  - High dose steroids
    - Protects the neuro-membrane from further destruction and improves blood flow
  - Contraindications
    - Injury more than 8 hours old
    - Injury below L2
    - Injury to cauda equina
  - SUP
  - Osmotic diuretics
  - Therapeutic hypothermia

Management

- Maintain airway, ventilation and oxygenation
- Treat pain and discomfort
- Monitor for and treat spinal shock
- Administer colloids, avoid hypotonic solution
- Administer atropine as prescribed for bradycardia