CCRN / PCCN REVIEW: Neurology / Musculoskeletal / Psychosocial

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Neuro Anatomy

- Cerebral hemispheres
- Diencephalon
  - Thalamus
    - Central command for incoming sensory impulses
  - Hypothalamus
    - Regulation of temperature, appetite, sleep
  - Limbic system
    - Self preservation
- Brainstem (origin of cranial nerves except I and II)
  - Midbrain
  - Pons (respiratory centers)
  - Medulla (cardiac and respiratory centers)
    - Gag
- Cerebellum (Balance)

Cerebral Hemisphere Key Functions

- Left
  - Analysis
  - Problem solving
  - Language
  - Mathematics
  - Abstract reasoning
- Right
  - Spatial relationships
  - Non verbal communication
  - Music
  - Artistic ability

Corpus Callosum
Cerebral Lobes: Key Functions

- Frontal: Voluntary motor function, intellectual function, personality
- Temporal: Memory function and emotion
- Parietal: Sensory function, object recognition and position sense, body awareness and image
- Occipital: Visual reception
Intracranial Pressure

- **Monro – Kellie Hypothesis (18th century)**
  - Any increase in one component necessitates a decrease in another
  - Cerebral blood flow (CBF) (10%)
  - CSF (5%)
  - Brain Tissue (85%)

- Methods of compensation
  - Displacement of CSF
  - Increase of CSF absorption
  - Compression of the low pressure venous system

- **Normal ICP**
  - 0-15 (7 to 15) mmHg
  - > 15 = intracranial hypertension
  - > 20 = pathological

Intracranial Space

Increased ICP associated with 20% mortality.
Causes of Intracranial Hypertension (Increase ICP)

- Conditions that increase brain parenchyma volume
  - Hematomas
  - Cerebral edema
  - Tumors
- Conditions that increase cerebral blood flow
  - Hypercapnea
  - Hypoxia
- Conditions that increase CSF volume
  - Increased production of CSF
  - Decreased absorption of CSF
  - Hydrocephalus from blockage of CSF circulation and ventricular dilatation

ICP Monitoring

- Indications
  - A Glasgow coma score of ≤ 8
  - Intradural hematomas or contusions
  - Demonstrated cerebral edema on CT scan
  - Midline shift
  - If normal CT & have 2 or more of the following
    - Age older than 40
    - Motor posturing
    - SBP <90 mm Hg
ICP Monitoring Locations

- **Intra ventricular**
  - Ventriculostomy
  - Placed in anterior horn of lateral ventricular
  - Prefer to place in non dominant ventricle
  - Removal of CSF possible
  - Very accurate

- **Subarachnoid**
  - Easy to insert
  - Screw or bolt
  - Unreliable at high ICP

- **Epidural Space**
  - Easy to insert
  - Least invasive
  - Head position has no effect on reading

- **Intra parenchymal**
  - Easy to insert
  - Very accurate
  - 1cm into brain tissue
  - Head position has no effect on reading
ICP Monitoring Locations

ICP Monitoring Wave

- **P1: Percussion wave**
  - Arterial pulsation

- **P2: Tidal Wave**
  - Represents intracranial compliance
  - Ends on dicrotic notch
  - **Most clinically significant**: As ICP increase so does P2
  - If P2>P1 ⇒ decreased brain compliance

- **P3: Dicrotic wave**
  - Immediately after dicrotic notch
  - Represents venous pulsation
ICP Monitoring Waves:
A Waves

- Most critically significant of the 3 waves
- Already elevated baseline with sharp increases in ICP
- 50 to 100 mm Hg which plateau for 5 to 20 minutes
- Often preceded by B waves
- A waves have been associated with brain herniation
- May be caused by an impairment in CSF absorption
ICP Monitoring: B and C Waves

- **B Waves**
  - Sharp, rhythmic oscillations – saw tooth appearance
  - Entire wave duration is ½ to 2 minutes
  - Can raise ICP from 20 to 50 mm Hg
  - B waves should not be lightly dismissed, may precede A waves
  - May also be due to respiratory variations and alterations in cerebral blood flow
  - B waves precede changes in blood pressure

- **C Waves**
  - Occur 4-8 fluctuations / minute
  - Can be seen with normal ICP

Ventriculostomy Care

- **Zero**
  - Q shift or q 12 or when break in interface with monitor
  - Level of the Foramen of Monro
    - Tragus of ear or outer canthus of eye
    - Use same landmark consistently

- **Measurement**
  - When the ICP is higher than the prescribed pressure level, CSF will drain

- **Drainage**
  - CSF absorption capacity is normally approximately 2-4 times the rate of production.
    - CSF diversion / reabsorption occurs in response to increased ICP
    - CSF exists in the craniospinal space
      - Can also drain from lumbar space

- **Safety Issues**
  - Risk of bacterial infection
  - Do not prime with IV bag or pressure bag
Complications of Ventriculostomy

- Infection
  - Common complication (1% to 12%)
- Intracerebral hemorrhage / hematoma
  - Also common complication
  - Monitor for changes in color of CSF
- CSF Leak
  - Maintain closed system
- CSF Over drainage
  - Do not drain below a pressure of 15 mm Hg

Cerebral Spinal Fluid Pressure Monitoring and Drainage after Aortic Surgery

- Cross clamping of aorta proximal to descending thoracic aorta:
  - Acute elevation in cerebrospinal fluid pressure
- Spinal cord perfusion pressure is the difference between the spinal arterial pressure and the cerebrospinal fluid pressure
- Monitoring of cerebrospinal fluid pressure and potential drainage is a strategy used to maintain adequate spinal cord perfusion pressure

- Consequences of over drainage
  - Subdural hematoma
  - Over drainage results in a collapse of the ventricles with increased negative pressure leading to tearing of the dural veins
- Complications
  - Post dural puncture headache, with some patients requiring an epidural blood patch to seal meningeal leak
  - Others: spinal or epidural hematoma, meningitis, introduction of blood into subarachnoid space causing vasospasm, decreased spinal blood flow; and persistent leak of cerebrospinal fluid
# Signs and Symptoms of Increased ICP

**Early signs:**
- Decreased level of consciousness
- Deterioration in motor function
- Decreased language articulation / comprehension
- Headache, nausea, vomiting
- Visual disturbances, changes

**Late signs:**
- Pupillary abnormalities (asymmetry of > 2 mm)
  - Anisocoria
- Cushing’s triad
- Changes in arterial blood gases

## Cushing’s Triad
- Widened pulse pressure
  - Increased systolic
  - Decreased diastolic
- Bradycardia
- Irregular respirations

- Occurs when medulla is compressed
Optic Disc Edema

Cerebral Blood Flow

- Normal: 750 ml / minute (arterial blood)
- Brain takes up 2% of total body weight
- Brain requires 15% to 20% of resting cardiac output
- Brain requires 15% of body’s oxygen demand
Cerebral Blood Flow

• Auto regulation
  – Capacity to maintain constant CBF despite systemic BP
  – In place when cerebral perfusion pressure is in range to 60 mmHg to 160 mmHg

Auto-regulation is altered in an injured brain. There is oligemia (reduced blood flow) the first 24 hours after injury.

Cerebral Blood Flow

• When auto regulation fails cerebral blood flow alters with changes in systemic pressure
  – Hypertension = increased ICP
  – Hypotension = hypoperfusion and ischemia
Cerebral Blood Flow

- Increase in cerebral blood flow
  - Acidosis ($\uparrow$PaCO$_2$)
  - Increased metabolic rate

- Decrease in cerebral blood flow
  - Alkalosis ($\downarrow$PaCO$_2$)
  - Decreased metabolic rate

Cerebral Perfusion Pressure (CPP)

- Pressure at which the brain is perfused
- **Cerebral blood flow is impacted by CPP**
- CPP = MAP − ICP
- Normal: 80–100 mmHg
- Acceptable: 60-150 mmHg
- Injured brain needs a CPP > 70 mmHg
Brain Tissue Oxygenation

- Jugular Venous Saturation
  - Normal 55-70%
  - Global measurement
  - SjO2 < 50-55%
    - Cerebral Oligemia
      - Low Flow
      - Demand > Supply
  - SjO2 > 70-75%
    - Cerebral Hyperemia
      - Luxury Perfusion
      - No extraction (brain death)

½ of patients with a normal ICP will have low oxygen states with head injury

Brain Tissue Oxygenation

- PbtO2
- Measured by LICOX catheter (trade name)
- Normal 20 – 40 mmHg
- Risk for death increases
  - < 15 for 30 minutes
  - < 10 for 10 minutes
- 90% mortality when < 5
- Neuronal death when < 2
Patient Management Strategies: ICP, CPP and Brain Oxygenation

- Optimize the CPP using the ICP and PbtO2
  - ICP > 20 mmHg should be treated

- Need to find the right blood pressure for the patient
  - Raising the blood pressure may actually benefit patient
  - Goal is usually high normal range

Patient Care Management: ICP, CPP and Brain Oxygenation

❖ Osmotherapy - Direction of flow is from hypoconcentrated to hyperconcentrated

❖ Osmotic diuretics
  - Mannitol
  - Require intact blood brain barrier
  - Large molecule that stays in the extra cellular space
  - Careful attention to electrolytes (potassium and sodium)
  - Careful attention to volume status (body weight, CVP)
  - Maintain serum osmolality 305 to 315 mOsm/L (normal 280 to 300mOsm/L)

❖ Hypertonic saline
Patient Care Management: ICP, CPP and Brain Oxygenation

❖ Nonosmotic Diuretics
   • Loop diuretics (furosemide)
   • Pulls sodium and water from edematous area
   • May even decrease CSF production
   • Electrolyte imbalances not as severe

❖ Volume Maintenance
   • Prefer isotonic solutions
   • Avoid hypotonic fluids (D5W)

Patient Management Strategies: ICP, CPP and Brain Oxygenation

• Standard of Care PaCO2 35-45 mmHg
  – Brief periods of hyperventilation (< 2 to 3 hours) can decrease ICP; prolonged periods results in cerebral ischemia (arterial vasoconstriction)
  – Number one cause of cerebral oligemia is hyperventilation
  – PaCO2 Goal: Approximately 35

• Oxygenation
  – Initially use 100% FIO2
  – Do not bag
  – Hyperoxygenate for 5 minutes prior to suctioning and limit to two passes (< 10 sec)
  – Driving force of oxygen is gradient between PaO2 and PbtO2
  – Prevent hypoxemia and hyperoxemia
Patient Care Management: ICP, CPP and Brain Oxygenation

• Avoid PEEP > 20 cm H2O, coughing, suctioning, tight trach ties
• Lidocaine
  – Helps decrease sensory stimulation
  – Administration through endotracheal tube before suctioning
  – Administration IV push before nasotracheal suction

Patient Care Management: ICP, CPP and Brain Oxygenation

• Adequate sedation and analgesia
  – Propofol: Decreases ICP but can also decrease PbtO2
• Keep neck midline – don’t impede venous return
• Keep head of bed elevated 30 degrees
  – Individualize head elevation to maximize CPP
• Quiet room, low lighting, gentle touch
• Temperature 36 to 37 degrees
  – Cerebral metabolic rate increase 5% to 7% per degree C of increase in body temperature
• Keep blood glucose 80-110
Patient Care Management: ICP, CPP and Brain Oxygenation

• **Seizure Control**
  – Seizures occur in 5% to 20% of head injured population
  – Seizures increase metabolic demands
  – Increase demands can result in increased blood flow to the brain or ischemia due to lack of supply
  – Phenytoin is drug of choice
  – Lorazepam may be used until phenytoin is therapeutic

Management of Increased ICP

• **Control of Metabolic Demand**
  – Barbiturate Coma (pentobarbitol)
    • Decreases ICP by decreasing cerebral blood flow and metabolism
    • May shunt blood from healthy brain to ischemic area
    • Used when other therapies fail to control ICP
    • ICP > 40 mm Hg for 15 minutes or more
    • Goal: Decrease ICP to 15 –20mm Hg Sustain MAP 70-80mmHg
    • Expect decrease of 10mmHg within 10 minutes
    • Maintain until ICP has been controlled for 24 hour
    • Wean off over several days
STROKE

Anterior Cerebral Circulation

Two internal common carotid arteries

- Arise from the common carotids
- Provide the major blood supply to the brain
- **Supply** optic nerves, retina, and the majority of the cerebral hemispheres
- Divides into: *Anterior cerebral artery and middle cerebral artery*
**Posterior Cerebral Circulation**

**Two vertebral arteries**
- Arise from right and left subclavian arteries.
- Merge to form basilar artery; basilar artery *divides into two posterior cerebral arteries.*
- *Supplies* the cervical cord, brainstem, medulla, cerebellum, caudal part of diencephalons, medial and posterior temporal lobes, and the occipital lobes.

**Cerebral Circulation**

- **Circle of Willis**: an anatomical ring of vessels joining the carotid artery system and the vertebrobasilar system.
  - Posterior communicating artery.
  - Posterior cerebral artery.
  - Anterior communicating artery.
  - Anterior cerebral artery.
Anatomy Application

Middle Cerebral Artery
Most common intracerebral vessel affected by stroke

<table>
<thead>
<tr>
<th>Symptoms of TIA</th>
<th>Carotid</th>
<th>Vertebrobasilar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of vision</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Weakness</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Numbness or tingling</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Slurred speech</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Language difficulty</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Vertigo</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Ataxia, imbalance</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Double vision</td>
<td>✓</td>
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Ischemic Stroke

• Sudden, severe disruption of the cerebral circulation with a subsequent loss of neurologic function caused by thrombus or embolism

• Ischemic strokes account for 87% of all strokes (Go et al., 2013).
  – 10% Intracranial hemorrhage
  – 3% subarachnoid hemorrhage
Causes of Ischemic Stroke

- Large artery atherosclerotic disease involving either thrombosis or embolic plaque: often affects the cortex of the brain.
- Thrombotic stroke
  - Intracranial vessels or extra cranial vessels
  - Thrombotic strokes are more common in older persons with extensive atherosclerotic disease.
  - Thrombosis is often associated with a nighttime stroke due to more sluggish blood flow at night.
  - Can be progressive, exhibiting worsening symptoms over minutes, hours, or days.
Stroke Classifications

- **Cardiogenic embolic strokes** are from cardiac sources such as atrial fibrillation, ventricular aneurysm, or bacterial endocarditis.
  - The middle cerebral artery is a common location for these strokes, and they have a sudden onset with maximum deficit occurring immediately.
  - More severe and result in greater residual deficits

- **Lacunar stroke**: Small or penetrating vessel occlusive disease
  - Affect the deeper non-cortical areas of the brain.
  - Infarcted brain tissue leaves small cavities referred to as lacunes.
  - Multiple lacunes can impair intellectual capacity, and dementia can be a complication.
  - Typical lacunar symptoms include pure motor or pure sensory hemiplegia, clumsy hand syndrome, and dysarthria (a pure motor speech disorder)
  - CT scan is not sensitive in diagnosis

- **Watershed or border zone infarct**
  - Focal ischemia from decreased perfusion pressure
  - Bilateral watershed infarcts are usually caused by systemic hypotension.

- **Other**: carotid dissections, hypercoagulable states, sickle cell disease, fibromuscular dysplasia, cystic medial necrosis, arteritis, etc.

- **Cryptogenic strokes** are of unknown cause.
  - It is estimated that up to 40% of cryptogenic strokes come from a cardioembolic source (Goldstein et al., 2011).
ISCHEMIC CASCADE

- **Primary cell death**
  - Within 4-5 minutes

- **Secondary cell death**
  - Compromised cells
  - Penumbra
  - Potential for recovery within 3 hours

- **Inflammation and immune response**
  - Impair ability to dissolve clot
  - Results in cerebral edema
  - Increased risk for intracerebral hemorrhage

<table>
<thead>
<tr>
<th>Signs and Symptoms of Stroke by Etiology</th>
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<tbody>
<tr>
<td><strong>Right Internal Carotid or Middle Cerebral Artery</strong></td>
</tr>
<tr>
<td>- Left sided weakness</td>
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<tr>
<td>- Left sided paresthesia</td>
</tr>
<tr>
<td>- Left sided sensory loss</td>
</tr>
<tr>
<td>- Left sided neglect</td>
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<tr>
<td>- Right eye monocular blindness</td>
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<tr>
<td>- Loss of right visual field in both eyes (homonymous hemianopsia)</td>
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<tr>
<td>- Visual-spatial deficits</td>
</tr>
<tr>
<td>- Aphasia in left handed or ambidextrous persons</td>
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Source: Brott et al., 2011.
Signs and Symptoms:

**Brainstem**
- Nausea and/or vomiting
- Diplopia, dysconjugate gaze, gaze palsy
- Dysarthria, dysphagia
- Vertigo, tinnitus
- Hemi (quadri) paresis or hemi (quadri) plegia
- Sensory loss in hemibody or all 4 limbs
- Decreased consciousness
- Hiccups, abnormal respirations

**Cerebellum**
- Truncal/gait ataxia
- Limb ataxia, neck stiffness

Signs and Symptoms: Hemorrhage
- Focal neurological deficits as in ischemic stroke
- Headache (especially in SAH)
- Neck pain
- Light intolerance
- Nausea, vomiting
- Decreased level of consciousness
Initial Diagnostic Testing

• CT
  – Most widely used
  – Identifies presence or absence of a mass or hemorrhage
  – Identifies location and characteristic of stroke
    • Insensitive to recent ischemic strokes and to brainstem and small infarcts
  – May show distortion or shift of ventricle if large stroke
    – Hypodensity = ischemia
      • When seen on initial CT = very large stroke

• MRI
  – Sensitive and accurate for ischemic stroke
  – May replace CT as initial diagnostic tool
  – More difficult to perform in critically ill patients
Treating Ischemic Stroke

- Admit to special care unit
- Maintain SpO2 > 94%; no routine oxygen in mild to moderate stroke without hypoxemia
- Cardiac monitoring for at least 24 hours
- Monitor for and treat hypoglycemia / avoid hyperglycemia with goal of 140 to 180 mg/dL
- Treat hyperthermia with acetaminophen
- **Supine position in patients not hypoxemic; elevate HOB 15 to 30 degrees if suspected increased intracranial pressure or if high risk for aspiration**
Treating Ischemic Stroke: Blood Pressure

- Ideal blood pressure is not known
- Most patients present with hypertension – spontaneous decrease 90 minutes post onset
- Hypotension can extend stroke
- If no rtPA the blood pressure is not lowered in the first 24 hours unless it is > 220/120 mmHg
- Lower blood pressure goal in those receiving rtPA in order to avoid cerebral bleed
- Home BP meds resumed 24 hours after presentation

BP with rtPA Administration

- BP should be < 185/110 mmHg before administration, and should be maintained at < 180/105 mmHg to reduce the risk of cerebral hemorrhage post administration
- Most common agents: intravenous labetalol or nicardipine. Sodium nitroprusside should be considered if unable to control
- VS q 15 minutes times 2 hours, q 30 minutes times 6 hours, and q 1 hour times 16 hours. More frequent if treating for HTN
Fibrinolytics

- Recommended in select patients > 18 years of age, who present within 3 hours of symptom onset or last time known to be normal
  - Baseline imaging study must exclude hemorrhage.
- Intravenous rtPA at a dose of 0.9 mg/kg (maximum dose of 90 mg) is used. The rtPA is given over 60 minutes with the first 10% given as a bolus over one minute.

Fibrinolytics

- Same dose may be considered in select patients who present between 3 and 4.5 hours of symptom onset.

- Not eligible to receive rtPA within this extended time frame include patients:
  - > 80 years old
  - Taking oral anticoagulants regardless of INR
  - Baseline NIH Stroke Score of > 25
  - Imaging evidence of ischemia involving greater than one third of the middle cerebral artery territory
  - History of both stroke and diabetes
Fibrinolytics

• rtPA may also be considered in patients with major surgery within the past 3 months or recent myocardial infarction.
  – These were previously contraindications.
  – The potential benefits need to be weighed against the increased risk of bleeding.

• rtPA is currently not recommended for patients taking direct thrombin inhibitors or factor Xa inhibitors unless the appropriate agent specific laboratory tests are normal or the patient with normal renal function has not taken a dose for > 2 days.

Fibrinolytics

• rtPA is not routinely used in patients with a NIH Stroke Score of < 4
  – Up to one third of these patients have a poor final outcome
  – Future research is needed for use of rtPA in this population.
  – Current guidelines: rtPA may be considered with the anticipated benefits weighed against the risks

• Recommended door to needle time of 60 minutes be achieved for at least 80% of patients
Exclusion Criteria for Intravenous rtPA in Patients Presenting within 3 Hours

- **Significant head trauma or previous stroke within last 3 months**
- **Arterial puncture at non compressible site within last 7 days**
- **Symptoms suggestive of subarachnoid hemorrhage**
- **Intracranial neoplasm, AV malformation, or aneurysm**
- **History of intracranial hemorrhage**
- **Active internal bleeding**
- **Recent intracranial/spinal surgery**
- **CT demonstrates multilobar infarction**
- **Blood glucose < 50 mg/dL**
- **BP mmHg > 185 systolic or > 110 diastolic**
- **Acute bleeding diathesis - including but not limited to:**
  - Platelet count < 100,000 / mm$^3$
  - Recent anticoagulant with elevated aPTT, PT > 15 seconds, INR > 1.7, or other agents specific laboratory tests
- **Relative but not absolute exclusion criteria include:**
  - Pregnancy
  - Major surgery or trauma within the last 14 days
  - GI or GU tract hemorrhage within the last 21 days
  - Acute MI within last 3 months
  - Seizure at onset with postictal residual neurological deficits
  - Minor or rapidly improving stroke symptoms.

Source: Jauch et al., 2013.

Nursing Care and Fibrinolytics

- Routine neurological checks and vital signs are every 15 minutes during rtPA and for 2 hours post administration, then every 30 minutes for 6 hours, and then every hour until it has been 24 hours post administration
- Blood pressure assessment should be more frequent if BP is > 180 mmHg systolic or > 105 mmHg diastolic
- Assess for oralingual angioedema
Nursing Care and Fibrinolytics

• Stop rtPA if the patient develops a severe headache, nausea or vomiting, acute hypertension, or a new deficit in the neurological exam
  – An order should be obtained for an emergency CT scan

• Avoid indwelling urinary catheters, nasogastric or oral gastric tube, and arterial lines whenever possible to reduce potential bleeding sites

• Do not start anticoagulants or antiplatelet agents until a follow up CT or MRI has been done 24 hours after the rtPA administration

Endovascular Interventions

• Mechanical clot retrieval or aspiration
  – Stent retrievers over MERCI device

• Acute angioplasty or stenting
  – May be considered at time of thrombectomy but usefulness is unknown

• Intra-arterial fibrinolysis
  – Considered in large strokes caused by MCA
  – May be considered in patients with contraindication to systemic fibrinolysis
2015 Guideline Updates

• Patients eligible for intravenous r-tPA should receive intravenous r-tPA even if endovascular treatments are being considered (not new)
• Patients should receive endovascular therapy with a stent retriever if they meet all the criteria (new)
• Endovascular treatment should be done within 6 hours
• There is no need to wait for failed rTPA

Endovascular Stent Retriever Indications

• Prestroke mRS (modified Rankin score) score 0 to 1
• Acute ischemic stroke receiving intravenous r-tPA within 4.5 hours of onset according to guidelines from professional medical societies
• Causative occlusion of the internal carotid artery or proximal MCA (M1),
• Age ≥18 years,
• NIHSS score of ≥6,
• ASPECTS of ≥6, and
• Treatment can be initiated (groin puncture) within 6 hours of symptom onset
Anticoagulants and Antiplatelets

- No evidence that early administration of anticoagulation improves outcomes or reduces the risk of early recurrent stroke.
  - Anticoagulant therapy should not be administered within 24 hours of intravenous rtPA (Jauch et al., 2013).

- Evidence that aspirin administered within 48 hours of acute ischemic stroke reduces the risk of early recurrent stroke.
  - Aspirin (initial dose of 325 mg) should be started within 24 to 48 hours of acute stroke in most patients.
  - Aspirin should not be administered within 24 hours of fibrinolytic therapy. (Jauch et al., 2013).

Acute Neurological Complications of Stroke

- Brain edema
  - Treatment for any seizures but not prophylactic

- Hemorrhagic conversion

- Herniation
Sub Acute Care

- Pneumonia prevention (responsible for 15-25% of stroke deaths in acute period)
  - Fever
- Early bowel and bladder care should be instituted to prevent complications such as constipation and urinary retention or infection
- Use of indwelling catheters should be avoided if possible because of the risk of UTI

- DVT Prophylaxis for all stroke patients who cannot ambulate at 2 days and who are at risk (Class I, Level of Evidence A).
  - Early mobility should always be attempted if safe (Class I, Level of Evidence B).

Sub Acute Care

- Falls – no ambulation without assistance (Class I, Level B)
- Skin Prevention – Braden (Class I, Level A)
- A swallow screen should be performed in the first 24 hours after stroke, preferably by the speech language pathologist (Class I).
  - Nurses should be familiar with bedside swallow assessment if a formal evaluation cannot be done within the specified period. Stroke patients should be kept NPO until the screen has been performed (Class I, Level of Evidence B)
  - Patients who cannot swallow should have a nasogastric tube placed, or if severity warrants, a percutaneous endoscopic gastrostomy tube should be placed (Class I).
  - Assessment of proper hydration is included in this recommendation.
Subarachnoid Hemorrhage

- Traumatic injury most common cause
  - Minor trauma if on anticoagulants
- Ruptured aneurysm most common cause in non trauma patients
- **Risk Factors for SAH**
  - Age > 60 years, female gender
  - Smoking
  - Aneurysms in posterior circulation, symptomatic aneurysm, and size > 5 mm

![Subarachnoid Hemorrhage CT Scan](image.png)
Subarachnoid Hemorrhage (aSAH)

- High mortality and morbidity
  - 10 to 15% die before arrival to hospital
  - 25% die within 24 hours
  - Of patients presenting to hospital 40% mortality at one month and 50% at six months
  - 1/3 of survivors with major neurological deficit
  - Some cognitive impairment in patients with good outcomes

- Severity of presentation is strongest predictor of outcomes

- Improved outcomes due to early repair and aggressive management of complications

Subarachnoid Hemorrhage (aSAH)

- **Presentation**
  - Sudden and severe headache
  - Nausea and vomiting
  - Signs of meningeal irritation
  - Photophobia other visual disturbances
  - Focal neurological deficit
  - Seizure
  - Loss of consciousness
Subarachnoid Hemorrhage (aSAH): Complications

Rebleeding

• Highest risk 2 to 12 hours
• Early rebleeding has worst outcome
• Rebleeding has mortality of approximately 80%
• Control of HTN: < 160 mmHg systolic reasonable
  – Nicardipine
• Early treatment with coiling or clipping to eliminate risk post procedure

Subarachnoid Hemorrhage (aSAH): Complications

• Vasospasm resulting in delayed ischemia is leading cause of death and disability from rupture
  • Begins day 3 and reach maximum risk at days 5 to 7
  • Clinical signs:
    Deterioration in mental status or development of focal neurological defects (i.e. hemiparesis or dysphagia)
• TCDs performed routinely (days 3 to 12)
Treatment of Vasospasm

- **Treatment**
  - Euvolemia
  - HTN
  - Oral nimodipine

- **Endovascular options**
  - Transluminal balloon angioplasty
  - Intraarterial vasodilator – papaverine

Hydrocephalus from SAH

- **Blood blocks arachnoid** villi resulting in acute hydrocephalus
- Blood in ventricles also blocks foramen of Monroe
- Acute hydrocephalus can increase ICP
- A ventriculostomy catheter can be placed to divert CSF
- Ventriculoperitoneal shunt for persistent hydrocephalus
Rationale for Early Treatment of Aneurysms after SAH

- Successful surgery eliminates risk of rebleeding
- Facilitates treatment of vasospasm: may induced hypertension without risk of rupture
- Potential ability to use lavage during surgery to remove agents responsible for vasospasm

- Operative mortality is higher but overall patient mortality is lower
Treatment of Aneurysms

• Ruptured aneurysms should be treated early

• Unruptured aneurysms can be treated electively

Craniotomy and Clipping

• Neck of aneurysm is clipped to exclude aneurysm from circulation
  – Morbidity is higher when operating on a ruptured aneurysm
  – Longer ICU stay compared to coiling

Endovascular Coiling

• Access through cervical internal carotid or vertebral artery
  – Detachable platinum micro coils – induce thrombosis at site of deployment

• Initially used in non-surgical candidates – can now be used in most patients

• Complications
  – Procedural perforation
  – Thromboembolic complications
Closed Head Injuries

- Blunt or penetrating trauma causing injury to the brain
- Falls
- Trauma (MVA)
- Sport related accidents
- Violence / Direct assault to the head

Type of Brain Injury Related to Closed Head Injuries

- Primary
  - Focal
  - Diffuse

- Secondary

Severity of Traumatic Brain Injury:
- Mild: GCS 13 to 15
- Moderate: GCS 9 to 12
- Severe: GCS 3 to 8
More About Secondary Injury

• Intracranial Causes
  – Intracranial hypertension
  – Cerebral Edema
  – Vasospasm
  – Infection
  – Seizures

• Systemic Causes
  – Hypotension
  – Hypoxia
  – Anemia
  – Hyperthermia
  – Hyper / hypocapnia
  – Electrolyte imbalance
  – Acid Base imbalance
  – SIRS

Optimal outcome depends on minimizing secondary injury.

More About Focal Injury

• Contusion
  – From trauma the brain strikes the internal surface of the skull

• There is bruising and petechial hemorrhages
• Lacerations of the brain may occur
• Dysfunction of the central nervous system lasting < 24 hours
• Hemorrhage and edema may act as intracranial mass and cause ↑ ICP
• Injury can be at site of impact (coup) and / or opposite (countercoup)
Three Stages of Diffuse Injury

• Concussion

• Diffuse injury with loss of consciousness > 24 hours

• Diffuse axonal injury

More About Diffuse Injury

<table>
<thead>
<tr>
<th>Concussion</th>
<th>Diffuse Injury (with loss of consciousness &gt; 24 hours)</th>
<th>Diffuse Axonal Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>• Stretching of nerve fibers / failure of conduction</td>
<td>• Severe mechanical disruption of axons and neuronal pathways in:</td>
</tr>
<tr>
<td>Classic</td>
<td>• Partial or complete paralysis of cerebral functioning</td>
<td>• Both cerebral hemispheres</td>
</tr>
<tr>
<td></td>
<td>• Complete recovery within 12 hours</td>
<td>• Diencephalon</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(lymbic system, thalamus, hypothalamus)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Brainstem</td>
</tr>
</tbody>
</table>
## Clinical Presentation

<table>
<thead>
<tr>
<th>Contusion (Focal Injury)</th>
<th>Concussion (Diffuse Injury)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Signs and symptoms vary depending on severity of injury and location of injury</td>
<td>Deficits or altered LOC clear within 6-12 hours</td>
</tr>
<tr>
<td>Change in LOC, motor sensory dysfunction, cranial nerve dysfunction, hemiparesis or hemiplegia seizures, signs of ↑ ICP</td>
<td>Unconscious for 10-15 minutes, headache, visual changes, dizziness, nausea / vomiting, confusion, restlessness, irritability</td>
</tr>
<tr>
<td>Neurological deficits persist less than 24 hours</td>
<td>Memory Loss</td>
</tr>
<tr>
<td></td>
<td>Amnesia</td>
</tr>
<tr>
<td></td>
<td>Post traumatic amnesia (related to events of injury) – usually lasts less than 5 minutes</td>
</tr>
</tbody>
</table>

### Clinical Presentation

- **Diffuse Injury (loss of consciousness > 24 hours)**
  - Loss of consciousness may last days to weeks
  - Long periods of retrograde and post traumatic amnesia
  - Purposeful movements, withdrawal from pain, restlessness
  - Residual permanent defects (Memory, cognitive and intellectual, abilities, personality changes)

- **Diffuse Axonal Injury**
  - Immediate and prolonged period of unconsciousness
  - Decorticate and decerebrate posturing
  - High death rates
  - Many survivors persist in vegetative state
  - Profound permanent defects
Special Treatment Considerations

- Prevent or treat ↑ ICP or intracranial hypertension
- Correct hypotension and maintain CPP (Cerebral Perfusion Pressure) of at least 70 mm Hg

Complications of Closed Head Injury

- Vasogenic Cerebral Edema
  - Breakdown of blood brain barrier
- Post Concussion Syndrome (few days to several weeks)
- Seizures
- Residual Neurological Deficits
- Persistent Vegetative State / Persistent Coma
Complications of Closed Head Injury

- Neurogenic Pulmonary Edema
  - Pulmonary edema with normal wedge
  - Thought to be from massive stimulation of SNS
  - Treat with osmotic diuretics
- Diabetes Insipidus
- SIADH
- Stress Ulcers

Types of Skulls Fractures

- Linear
- Depressed
- Basal

Skull Fractures have the same causes as closed head injuries.
Linear Skull Fractures

- Accounts for 80% of all skull fractures
- There is no displacement of bone
- May interrupt major vascular channels
- Temporal parietal fractures – middle meningeal artery ► epidural hematoma
- Occipital fractures – occipital artery ► epidural hematoma

Depressed Skull Fracture

- Outer table of skull is left depressed
- Most common to parietal and frontal
- May cause brain laceration and intracranial hematoma
- 30% associated with cerebral hematoma or contusion
- Dural laceration likely
Basal Skull Fracture

- Fracture at the base of the skull
  - Temporal, occipital, sphenoid, ethmoid
- May injure the cranial nerves
- May cause tearing of the dura with a cerebral spinal fluid leak
Skull Fractures: Presentation

<table>
<thead>
<tr>
<th>Linear</th>
<th>Depressed</th>
<th>Basal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scalp tenderness, echymotic or swollen area on scalp</td>
<td>Altered level of consciousness</td>
<td>Anterior Fossa</td>
</tr>
<tr>
<td>May or may not have scalp laceration <em>(scalp is moveable so fracture may not be beneath laceration)</em></td>
<td>Seizures</td>
<td>Middle Fossa</td>
</tr>
<tr>
<td>May be seen on plain film X-rays</td>
<td>Hemi paresis, hemiplegia</td>
<td>Posterior Fossa</td>
</tr>
<tr>
<td></td>
<td>Specific signs related to cranial nerves</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Frontal: Cranial nerve I (olfactory)</td>
<td>Difficult to confirm on x-ray.</td>
</tr>
<tr>
<td></td>
<td>Temporal: Cranial nerve VII (facial) or VIII (acoustic)</td>
<td>CT or MRI may be helpful.</td>
</tr>
<tr>
<td></td>
<td>May be seen on plain film x-rays</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Open fracture: Scalp laceration</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Closed fracture: No scalp laceration</td>
<td></td>
</tr>
</tbody>
</table>

Basal Skull Fracture

<table>
<thead>
<tr>
<th>Anterior Fossa</th>
<th>Middle Fossa</th>
<th>Posterior Fossa</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Rhinorrhea</td>
<td>• Otorrhea or rhinorrhea</td>
<td>• May have epidural hematoma</td>
</tr>
<tr>
<td>• Raccoon's Eyes <em>(takes 3-4 hours)</em></td>
<td>• CSF or blood behind the tympanic membrane <em>(hearing deficit)</em></td>
<td>• Signs and symptoms of injury to cerebellum, brainstem or cranial nerves</td>
</tr>
<tr>
<td>• May injure cranial nerve I <em>(olfactory)</em> causing anosmia</td>
<td>• Battle’s Sign <em>(takes 4-6 hours)</em></td>
<td></td>
</tr>
<tr>
<td>• May have facial fractures</td>
<td>• Other cranial nerve injuries</td>
<td></td>
</tr>
</tbody>
</table>
### Skull Fractures: Treatment Considerations

<table>
<thead>
<tr>
<th>Linear</th>
<th>Depressed</th>
<th>Basal</th>
</tr>
</thead>
<tbody>
<tr>
<td>No special treatment unless accompanied by neurological deficits</td>
<td>Protect brain under depressed area (position away from area) Prevent infection from open fracture Watch for hemorrhage with removal of bone fragments Surgical intervention if depression is greater than thickness of skull (5-7cm) Craniotomy: Emergently is scalp laceration or brain laceration is present.</td>
<td>Prevent further tearing of the dura Avoid blowing nose, cough with mouth open, exhale when turning Prevent CNS Infection Do not obstruct otorrhea / rhinorrhea Elevate HOB 30 degrees</td>
</tr>
</tbody>
</table>

### Skull Fractures: Complications

<table>
<thead>
<tr>
<th>Linear</th>
<th>Depressed</th>
<th>Basal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidural hematoma</td>
<td>Laceration of brain tissue Contusion or intracerebral hemorrhage CNS infection</td>
<td>Intracerebral hemorrhage CNS Infection Cranial nerve injury <strong>Carotid cavernous fistula</strong> Rare but serious Blood escapes carotid artery into cavernous sinus Bruit and pulsation of orbit over affected eye with visual disturbances</td>
</tr>
</tbody>
</table>
Types of Intracranial Hematomas

- **Subdural**
- **Epidural**
  - Between Dura and Skull
- **Intracerebral**
  - Called hemorrhagic stroke when caused by aneurysm, AV malformation, vascular tumor, or rupture of a vessel due to hypertension

### Intracranial Hematomas: Etiology

<table>
<thead>
<tr>
<th>Subdural</th>
<th>Epidural</th>
<th>Intracerebral</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Trauma</td>
<td>• Often seen with linear skull fractures when a major vascular channel is lacerated</td>
<td>• Trauma</td>
</tr>
<tr>
<td>• May occur spontaneously</td>
<td>• May be bilateral</td>
<td></td>
</tr>
<tr>
<td>• Patients with coagulation disorders or on anticoagulants</td>
<td>• Gunshot wound</td>
<td></td>
</tr>
<tr>
<td>• Frequently seen in elderly with cerebral atrophy and in alcoholics</td>
<td>• Depressed skull fracture</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• May be bilateral</td>
<td>• Severe acceleration / deceleration injury</td>
</tr>
</tbody>
</table>
Subdural Hematoma

- Blood accumulates below dura mater
- Usually a venous bleed

Acute
- Signs and symptoms within 24 hours of injury

Sub Acute
- Signs and symptoms within 2 weeks of injury

Chronic
- Signs and symptoms can occur weeks to months after an injury

Epidural Hematoma

- Blood accumulates above the dura mater
- Usually arterial bleeding (tearing of arteries from skull fractures)
- May also be venous

Linear fracture
- Temporal and parietal bones ► middle meningeal artery
- Occipital bone ► occipital artery
Intracerebral Hematoma

- Hematoma in the brain tissue itself
- Missile injury

Force from acceleration / deceleration that causes bleeding deep into the tissue
Intracranial Hematomas: Presentation

<table>
<thead>
<tr>
<th>Subdural</th>
<th>Epidural</th>
<th>Intracerebral</th>
</tr>
</thead>
<tbody>
<tr>
<td>● Headache</td>
<td>● Headache</td>
<td>● Signs and symptoms vary depending on location of</td>
</tr>
<tr>
<td>● Irritability progressing to confusion and decreased LOC</td>
<td>● Ipsalateral oculomotor paralysis</td>
<td>hematoma and size of hematoma (rate of blood</td>
</tr>
<tr>
<td>● Ipsalateral oculomotor paralysis</td>
<td>● Contralateral hemiparesis, / hemiplegia</td>
<td>accumulation)</td>
</tr>
<tr>
<td>● Contralateral hemiparesis, / hemiplegia</td>
<td>● Importance of midline shift</td>
<td>● May or may not have increased ICP</td>
</tr>
<tr>
<td>● Importance of midline shift</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Special Treatment Considerations

- Prevent or treat for increased ICP
- Prevent further bleeding
  - No osmotic diuretics
  - Tamponade effect of hematoma helps stop bleeding
- Intracerebral hematoma requires surgery if large or if decreased neuro status
- Subdural and epidural hematomas require surgery unless very small
  - Burr hole and clot evacuation
  - Mortality increases significantly if surgery is delayed
Complications

- Post operative re-bleed
  - HOB elevated 20 to 30 degrees for acute and sub acute subdural and epidural hematomas
- Intracranial hypertension
- Hydrocephalus
- CNS Infection
- Diabetes Insipidus
- SIADH
- Seizures

Neurological Complications after Neurosurgery

- Temporary focal deficits from brain tissue manipulation
  - Cranial nerve palsies during posterior fossa operations
  - Motor weakness from tumors near motor cortex
- Cerebral edema (may cause transient defects)
  - Glioblastomas, prolonged operative time, repeat procedures
  - May be treated with corticosteroids (dexamethasone or methylprednisolone) or osmotherapy
- Intracranial hemorrhage
  - Most cases occur within 6 hours
  - Associated with emergency surgeries, post op HTN, an coagulopathy
  - > 50% are intraparenchymal or epidural
  - Surgical intervention often necessary
  - Venous infarction can also occur with craniotomy
- Stroke
  - Most common with carotid artery and endovascular intervention
Neurological Complications after Neurosurgery

- **Seizures**
  - Considered a result of hemorrhage until excluded
  - Cortical irritation from supratentorial operations
  - Associated with high grade astrocytomas, AVMs, metastatic tumors, abscesses, and midline meninigiomas
  - Antiepileptic medications may prevent early post operative seizures but are of no benefit for late seizures (> 1 to 2 weeks post op)
  - Fosphenytoin/phenytoin, valproic acid, phenobarbital, levetiracetam, lacosamide
  - Additional IV benzodiazepines for persistent or prolonged seizures

- **Pneumocephalus**
  - Cause HA
  - Frequent and often benign

- **Tension pneumocaphalus**
  - Infrequent
  - Can occur with open skull fracture
  - Bifrontal air on CT scan (Mt Fuji sign)
  - Requires immediate decompression with needle or burr hole

- **Cerebrospinal fluid leaks**
  - Common after transsphenoidal surgery
  - CSF rhinorrhea predisposes patient to meningitis and intracranial hypotension
  - Early indicator of meningitis may only be fever and elevated WBC
    - Early treatment for nosocomial bacteria methicillin resistant Staphylococcus and Pseudomonas

Neurosurgery Nursing Considerations

- **Careful, frequent neurological assessments**
  - Q 15 to 30 minutes for first 1 to 2 hrs; then q 1 hr for next six hrs
  - Extubation usually early, otherwise sedation holiday used for neurological evaluation

- **New deficit or worsening requires prompt reporting, more detailed neuro exam, and imaging**

- **Neuro ICU care:**
  - Supra or infra tentorial craniotomy for tumor or aneurysm
  - Craniofacial and transsphenoidal surgery
  - Major spine surgery
  - Carotid artery surgery
  - Endovascular procedures: embolization of AV malformations and aneurysms

- **Post craniotomy**
  - Physiologic stress results in fluctuations in BP, HR, blood glucose, and oxygen consumption (VO$_2$)
Neurosurgery Nursing Considerations

• Key elements
  – Early detection of complications (stroke, seizures, bleeding)
  – Emergence and recovery from anesthesia
  – Post operative nausea and vomiting
    • Particularly present in posterior fossa surgery
    • Causes increased risk of aspiration / HTN and increased ICP
    • Projectile vomiting can be indicator of hydrocephalus or intracranial hemorrhage
  – Restoring and maintaining normal body temperature
    • Shivering due to hypothermia occurs in 40% and increases VO$_2$ 200 to 400%
  – Pain management
    • Pain is often under controlled for fear of masking neurological assessment
    • Pain increase sympathetic tone and VO$_2$
  – DVT and GI prophylaxis

Post Operative Hypertension

• Labetalol – IV q 30 -60 minutes or infusion
• Nicardipine – infusion
• Hydralazie IV q 30 -60 minutes
• Enalaprilat q 6 hours PRN
Postoperative Issues with Carotid Endarterectomy or Stenting

- **Labile blood pressure**
  - Due to manipulation and edema of carotid baroreceptors
  - Baroreceptor stimulation → reflex stimulation of vagus nerve (bradycardia) and peripheral vasodilation (hypotension)
  - Hypertension can cause suture line disruption, neck hematoma, or hyperperfusion syndrome
  - Hypotension can cause inadequate cerebral perfusion - may require vasopressors (phenylephrine)

- **Bleeding**
  - Hematoma in neck can compromise airway and contribute to hypotension → careful monitoring of airway & breathing

- **Hyperperfusion syndrome**
  - Due to re-established blood flow to previously low flow cerebral circulation (post-op hypertension is major risk factor)
  - Symptoms: ipsilateral headache, focal seizures, intracranial hemorrhage
  - Treatment: blood pressure control (nitroprusside, labetalol, NTG), anti-seizure drugs (phenytoin)

- **Nerve injury**
  - Facial nerves and vagus nerve potentially affected
  - Neuro checks with emphasis on face, tongue, eye movement
  - Hoarseness, impaired swallowing, abnormal gag reflex are signs of nerve damage
Brain Tumors

• Primary: Arise from CNS structures
  – Typically don’t metastasize
• Metastatic lesions
• 95% of brain tumors
  – Gliomas
    • Astrocytoma (brain or spinal cord) – usually low grade
    • Glioblastoma Multiforme (IV)
      – Most common adult brain tumor (4th or 5th decade)
  – Meningiomas (benign)
  – Pituitary adenomas
  – Acoustic neuromas (Schwannomas)
    • Benign
    • Around 8th cranial nerve
  – Metastatic tumors

Brain Tumors: Pathophysiology

• Small tumors can damage neural pathways
• Invasion and infiltration of parenchymal tissue
• Angiogenesis disrupts normal blood brain barrier
  – promotes edema
• Tumors near 3rd and 4th ventricle can cause hydrocephalus
• Decreased cerebral perfusion
• End result may be increased ICP and possible herniation
Brain Tumors: Presentation

- Headache is often late complaint
  - New onset headaches in middle aged and older patients is of concern
- Seizures
  - New onset seizure in middle aged and older patients is a major concern
- Frontal lobe tumors often present in more subtle fashion
  - Decreased alertness, sleeping longer

Brain Tumors: Treatment

- Dexamethasone drug of choice to decrease edema
- Other treatment according to type
  - Surgery
  - Chemotherapy
  - Radiation
Encephalopathy

- Affects large part of brain and alters mental status
- Syndrome of global brain dysfunction
- Caused by change in structure or function of brain
  - Infectious diseases
  - Thiamine deficiency (Wernicke)
  - Metabolic dysfunction
  - Mitochondrial dysfunction
  - Exposure to toxins
  - Liver dysfunction
  - Hypertension
  - Hypoxia

Encephalopathy

- LOC
  - Arousal
  - Awareness
  - Most important clinical indicator
- Acute confusional state with altered LOC
  - Drowsiness, stupor, coma
- Changes in mental status:
  - Decreased oxygen delivery
  - Reduction in blood glucose
  - Reduction in cerebral perfusion pressure

* Infectious causes may also produce fever and HA as signs.
Coma

- Supratentorial mass
  - Can impinge on diencephalic structure
  - Compress ascending reticular activating system
- Subtentorial mass
  - Directly damage core of brainstem
- Metabolic disorders
  - More generalized

Deep sleep like state from which a person cannot be aroused.

Both cerebral hemispheres or brainstem are injured.
Herniation

- Shift of tissue or protrusion through abnormal opening
- Result of increased ICP
  - May be localized

- Supratentorial structures
  - Cerebral hemispheres
- Infratentorial structures
  - Cerebellum

Midbrain passes through tentorial opening (tentorial notch or incisura)

Herniation

- Supratentorial herniation
  - Cingulate
  - Central
  - Uncal
  - Transcalvarial

- Infratentorial herniation
  - Tonsillar (downward transtentorial herniation)
  - Upward transtentorial herniation
Herniation

• Most common uncal herniation
  – Temporal lobe displacement
  – Compression cranial nerve III (oculomotor), midbrain, and posterior cerebral artery
  – 3 Stages
    • Early 3rd cranial nerve stage
    • Late 3rd cranial nerve stage
    • Lower pons / upper medulla stage
Uncal Herniation

• Early signs
  – Altered LOC
  – Contralateral hemiparesis
  – Unequal pupillary response
  – Unilateral 3rd cranial nerve palsy

• Late signs
  – Changes in respirations
  – Ipsalateal nonreactive pupil
  – Disconjugate oculocephalic reflex
    • Normal: the eyes conjugately deviate in the direction opposite to the head’s movement
    • Loss of this reflex implies dysfunction of brainstem or oculomotor nerves

Brain Death

• The irreversible loss of all functions of the brain, including the brainstem
  – Coma
  – Absence of brain stem reflexes
  – Apnea

• Brain death is legal and clinical death

• Ancillary tests can be used as aides but do not replace a clinical exam and are not needed to declare brain death
  – EEG
  – Cerebral angiography
  – Nuclear scan
  – Transcranial Doppler

• One neurological exam sufficient in most states (some require two)
• No US Laws
• Most states legally allow all physicians (some states or hospital policies require specialized expertise)

• Wide variation in adherence to guideline recommendations
Practical Issues in Determining Brain Death in Adults: AAN Clinician Guideline Supplement 2010

• Assure an irreversible cause of coma
  – Exclude CNS depressant drugs (calculation of clearance by $5 \times \frac{1}{2}$ life if normal renal and hepatic function; drug screen, plasma levels below therapeutic range, consider hypothermia on drug clearance, legal limit of alcohol for driving, no recent neuromuscular blockade, no severe electrolyte or acid base disturbance)

• Achieve normal (near normal) core temperature

• Achieve normal systolic pressure (neurological exam usually reliable when systolic $\geq 100$ mmHg)

Brain Death: Clinical Exam

• Coma / lack of all responsiveness
  – No eye or motor response to noxious stimuli except for spinally mediated reflexes

• Absence of brainstem reflexes
  – Absence of pupillary response to light in both eyes
  – Pupils are usually 4 to 9 mm (mid sized dilated)
  – Constricted suggests possible drug effect

• Absence of ocular movements in response to oculocephalic testing or oculovestibular reflex testing
Brain Death: Clinical Exam

• Absence of corneal reflex
  – No eyelid movement with touching of cornea

• Absence of facial movement to noxious stimuli
  – Deep pressures on condyles at the level of temporomandibular joints and deep pressure at the supraorbital ridge

• Absence of pharyngeal (gag) and tracheal (cough response to tracheal suctioning) reflexes

Brain Death: Clinical Exam

• Apnea
  – \( \text{CO}_2 \) challenge
  – Prerequisites:
    • Normotension
    • Normothermia
    • Euvolemia
    • Eucapnia
    • Absence of hypoxemia
    • No history of \( \text{CO}_2 \) retention
### Apnea Procedure

- Preoxygenate 10 minutes / 100% FIO2 to PaO2 > 200 mmHg
- Decrease frequency to 10 breaths per minute (eucapnia)
- Reduce PEEP to 5 cm H2O
- If saturation remains > 95% obtain ABG
- Disconnect patient from ventilator
- Preserve oxygenation
- Observe for respiratory movements for 8 to 10 minutes (abdominal or chest excursions including a brief gasp)
- Abort if systolic BP drops below 90 mmHg
- Abort if O2 sat drops < 85% for > 30 seconds; retry with CPAP
- If no respiratory drive is observed – repeat blood gas after 8 minutes
- If PCO₂ is > 60 mmHg (or > 20 mmHg over a baseline normal PCO₂) in absence of respiratory movements the apnea test is positive and supports diagnosis of brain death
- If results inconclusive and patient is stable; preoxygenate again and repeat for a period of 10 to 15 minutes
- Time of death is the time the arterial PCO₂ reached the target

### Meningitis

- Acute infection of meninges
  - Pia matter
  - Arachnoid matter
  - Dura matter
- Communicating structure: therefore always cerebrospinal infection
Meningitis

- Infectious agent crosses blood brain barrier
- Inflammatory reaction in CSF and ventricles of brain
- No host defense in CSF: rapid duplication

Pathophysiology of Meningitis

- Infiltration of neutrophils in subarachnoid space
- **Exudate forms**
  - Polymorphonuclear neutrophils (PMNs) attack invading organism
  - Leukocytes / histiocytes try to **wall off exudate from pathogen**

- **Exudate forms two layers by end of 2\textsuperscript{nd} week**
  - Outer: under arachnoid membrane - (PMNs) and fibrin
  - Inner: next to pia-lymphocytes, plasma cells, macrophages

- Layers can resolve in response to drug therapy
- If infection lasts inner layer can remain and form permanent fibrous structure
Pathophysiology of Meningitis

- **Adhesions** between pia and arachnoid membranes
- **Congestion** of vessels and tissues
  - Increased ICP
  - Cerebral edema
- **Degeneration** of nerve cells
- **Progression** of vasculitis, cortical necrosis, petechial hemorrhage, hydrocephalus, cranial nerve damage

Etiology of Meningitis

- Fungal
- Viral (aseptic meningitis)
- Bacterial
  - 80% **Streptococcus pneumoniae**
  - Early and aggressive treatment
- **Access to subarachnoid space**
  - Head injury (basal skull fracture)
  - ICP monitoring
  - Cranial surgery / lumbar puncture
  - Paranasal sinus injury
  - Mastoiditis / otitis media
  - Sepsis / septic emboli
Clinical Presentation: Meningitis

- Fever
- Severe headache
- Nuchal rigidity
- Positive Kernig’s sign
- Positive Brudzinski’s sign
- Photophobia
- Decreased LOC
- Cranial nerve dysfunction II-VII

Diagnosis: Examination of cerebral spinal fluid.
Lumbar Puncture

• Obtains information about CSF
• **Should not delay initiation of life saving treatments** (i.e. antibiotics)

• Indications
  – Suspicion for meningitis
  – Suspicion for SAH
  – Suspicion for Guillain-Barre syndrome

Lumbar Puncture

• Absolute contraindications:
  – Infection over skin
  – Unequal pressures between supratentorial and infratentorial compartments

• Complications
  – Post-spinal headache
  – Infection
  – False positive bloody tap
Status Epilepticus

• Seizure (s) lasting > 30 minutes
  – Consciousness not regained between seizures
  – May be partial or generalized (both cerebral hemisphere)
• Mortality 20-30%

Status Epilepticus: Etiology

• Inadequate meds / sudden withdrawal
• Hyponatremia / hypocalcemia / hypoglycemia
• Fever / CNS Infection
• Cerebral vascular disease / anoxia / cerebral edema, hematomas, head trauma
• Drug abuse, alcohol abuse / sleep deprivation
Status Epilepticus: Pathophysiology

- Apnea during seizures – hypoxemia – cerebral anoxia
- Increased metabolic activity of brain
  - Hypoglycemia
  - Hyperthermia
- Results perpetuate more seizures
- Generalized tonic clonic seizures are most common in status epilepticus

Status Epilepticus: Treatment

- Airway – breathing
- Labwork to identify cause
- Thiamine 100 mg IV if alcoholism
- 50% glucose for hypoglycemia
- Benzodiazepines (diazepam, lorazepam) IV
  - Fast acting
- Phenytoin
  - Slow administration 50 mg / min
  - 15 to 20 minutes to peak
  - Bradycardia and hypotension in patients > 40
- Phenobarbital if seizures > 30 minutes
  - Slow admin 50 to 100 mg / min
  - Respiratory depression / hypotension
- Lidocaine 20% solution in NS
- General anesthesia / barbituate coma
- Neuromuscular blockade does not stop brain electrical activity
- Surgical excision
- Vagal nerve stimulation
Neuromuscular Disorders: Myasthenia Gravis

- Rare autoimmune disorder of peripheral nerves
- Antibodies (IgG) form against acetylcholine nicotinic post synaptic receptors at neuromuscular junction
- Ocular and generalized
- Progressive reduced muscle strength with use and increased strength with rest
  - Inefficient neuromuscular transmission
- Bulbar muscles (mouth and throat) affected most

Symptoms can include:
- Difficulty swallowing
- Double vision
- Unsteady walk
Myasthenia Gravis: Treatment

- Anticholinesterase medication
- Immunosuppressant therapy
- Plasmapheresis
- Thymectomy
  - Important if thyoma (benign thymus tumor) is present
    - Present in 15% of patients with myasthenia gravis
Myasthenia Gravis: Crisis

<table>
<thead>
<tr>
<th>Myasthenic</th>
<th>Cholinergic</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Severe weakness of respiratory muscles due to under treatment of disease</td>
<td>• Over stimulation at neuromuscular junction due to an excess of acetylcholine • Muscles stop responding and flaccid paralysis occurs</td>
</tr>
</tbody>
</table>

Neuromuscular Disorders: Guillain-Barre Syndrome

• Collection of clinical syndromes
• Post infectious immune mediated problem – Campylobactor jejuni infections
• Acute inflammatory demyelinating polyradiculoneuropathy
• Weakness and diminished reflexes resulting in acute flaccid paralysis
• Classic: Ascending paralysis
Neuromuscular Disorders: Guillain-Barre Syndrome

- Respiratory failure is potential complication
- Mortality increases with age
- Death usually occurs in ventilated patient
- In most common subset, symptoms resolve secondary to remyelination
- Most patients make full and functional recovery in 6 to 12 months
  - 15% to 20% have moderate residual deficits
Neuromuscular Disorders: Muscular Dystrophy

- Inherited non inflammatory progressive muscle disorder
- No central or peripheral nerve abnormality
- Weakness progresses proximal to distal
- Associated mental deficits
- Typically not diagnosed until child begins to walk

Progressive Mobility

- Progressive mobility is an important topic in critical care for several reasons.
- Mobility is closely associated with several other clinical issues including:
  - Skin integrity
  - Foley catheter use and associated infections
  - Falls
  - Hospital acquired pneumonia
  - DVTs
  - Delirium
Early Progressive Mobility

- Series of planned movements
- Protocol driven
- Positioning/mobility techniques
  - Elevation of HOB
  - Manual turning
  - Passive/active ROM
  - Continuous lateral rotation therapy
  - Prone positioning
  - Movement against gravity
  - Upright/leg-down position
  - Chair position (reverse Trendelenburg)
  - Dangling
  - Ambulation

Goal: Returning patient to baseline mobility status

Progressive Mobility Guidelines for Critically Ill Patients

Effects of Bedrest

- Muscle strength in a healthy person can decrease 1.3% to 3% for every day spent on bedrest. Topp R. Am J Crit Care. Clin Issues 2002
- Effects are more profound in older people and in those with critical illness. Yende S. Thorax. 2006.
- A new study suggests that 3% to 11% strength loss occurs for every day in bed in an ICU setting.
  - Age and days on bedrest are independent predictors of worsening function.
Benefits of Progressive Mobility

- Decrease ICU and hospital LOS
- Improve overall physical functioning
- Decrease duration of mechanical ventilation
- Decreased incidence of delirium
- Decreased incidence of other hospital acquired complications


Is Early Progressive Mobility Safe?

- Hopkins R. Crit Care Clinics. 2007;23:81-96

< 1% activity-related adverse events with 1,449 activity events in 103 patients

Early ICU Mobility Therapy Protocol

<table>
<thead>
<tr>
<th>Level 1</th>
<th>Level 2</th>
<th>Level 3</th>
<th>Level 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unconscious</td>
<td>Conscious</td>
<td>Conscious</td>
<td>Conscious</td>
</tr>
<tr>
<td>Passive ROM 3x/day</td>
<td>Passive ROM 3x/day</td>
<td>Passive ROM 3x/day</td>
<td>Passive ROM 3x/day</td>
</tr>
<tr>
<td>q2Hr Turning</td>
<td>q2Hr Turning</td>
<td>q2Hr Turning</td>
<td>q2Hr Turning</td>
</tr>
<tr>
<td>Active Resistance</td>
<td>Active Resistance</td>
<td>Active Resistance</td>
<td>Active Resistance</td>
</tr>
<tr>
<td>Sitting Position Minimum 20 minutes 3x/d</td>
<td>Sitting Position Minimum 20 minutes 3x/d</td>
<td>Sitting Position Minimum 20 minutes 3x/d</td>
<td></td>
</tr>
<tr>
<td>Sitting on edge of bed if can move arm against gravity</td>
<td>Sitting on edge of bed if can move arm against gravity</td>
<td>Active Transfer to Chair (OOF) minimum 20 minutes/day if can move leg against gravity</td>
<td></td>
</tr>
</tbody>
</table>

Morris P. Crit Care Med. 2008;36:2238

Results

- ICU LOS
  - Protocol: 5.5 days
  - Standard: 6.9 days
- Hospital LOS
  - Protocol: 11.1 days
  - Standard: 14.5 days
- Reduced sedation levels
- Increased activity sessions during ICU stay
- Cost savings
- No adverse events during mobility session
**Keys to Successful Progressive Mobility Plan**

- Strong nursing support
- Strong physical therapy program
- Guidelines for referral to PT/OT
- Strong interprofessional program
- Staff, patient and family education
- Appropriate resources and equipment
- Daily During Rounds/Report: Did my patient achieve his/her maximal mobility activity today?
  - Was a goal established?
  - Did everyone know the goal?
    - Mobility is everyone's responsibility

**In-Bed vs Out-of-Bed Mobility**

**In-Bed**
- Passive ROM
- Turning
- **Hemodynamic training**
- Dangling
- Active strengthening exercises

**Out-of-Bed**
- Standing at bedside
- Sitting on a regular chair
- Sitting in a cardiac chair
- Walking
Barriers to Progressive Early Mobility

- Severity of disease
- Severity of weakness
- Premorbid level of function
- ICU culture that promotes bed rest
- Nutritional state
- Sleep deprivation
- Level of delirium
- Pain
- Obesity

Absolute Contraindications to Progressive Mobility

- Patients on neuromuscular blockade
- Hemodynamic instability requiring escalating dose or multiple vasopressors
- Significant oxygenation dysfunction requiring high level of oxygen
- Unstable fractures
- Cerebral edema with uncontrolled intracranial pressure
- Active bleeding
- Intra-aortic balloon pump in femoral artery
- Pacer dependent with transvenous temporary pacemaker
- ECMO with femoral cannulation
- Femoral arterial sheath
- Open chest/open abdomen
Universal Falls Precautions

**Hourly rounding to include the five Ps:**

- **Pain:** Assess the patient’s pain level. Provide pain medicine if needed.
- **Personal Needs:** Offer help using the toilet; offer hydration, offer nutrition, empty commodes/urinals.
- **Position:** Help the patient get into a comfortable position or turn immobile patients to maintain skin integrity.
- **Placement:** Make sure patient’s essential needs (call light, phone, reading material, toileting equipment, etc.) are within easy reach.
- **Prevent Falls:** Ask patient/family to put on call light if patient needs to get out of bed.

- Patient with diabetic foot and suspected bone infection.
- Significant uptake in the soft tissue in the plantar aspect of the foot suggestive of cellulitis.
- Focus of abnormal activity in the talus (consistent with talar bone osteomyelitis).
Osteomyelitis

• Bone normally resistant to bacterial colonization
  – Trauma, surgery, foreign bodies disrupt integrity
  – Bacteria can grow in microfilm in prosthetic joints
  – Hematogenous bacteremia (vertebrae)
  – Major cause is Staph aureus

• Antibiotics and surgical interventions

Osteomyelitis

• Antibiotics for 4 to 6 weeks
  – Parenteral and oral
  – Rifampin is used with other antibiotics if a biofilm is involved

• Cierny-Mader Staging System
  – Stage of osteomyelitis
  – Physiologic status of the host
Osteomyelitis

- **Surgery**
  - Drainage
  - Extensive debridement
  - **Management of dead space** (soft tissue and bone left behind after debridement)
  - Adequate soft tissue coverage
  - Restoration of blood supply

  - Bone revascularization after debridement takes 4 weeks

---

**Depression and Anxiety in Cardiac Patients**

- **Depression**
  - Approximately 1 in 5 patients hospitalized with MI have major depression. There is also evidence that depression continues for several months after discharge (Fihn et al., 2012; Bush et al., 2005).
  - There is strong evidence that patients who are depressed post MI have a higher rate of mortality from both cardiac and non-cardiac causes (Bush et al., 2005).

- **Anxiety and Stress**
  - In post MI patients, interventions to reduce stress can reduce recurrent cardiac events by as much as 35-75% (Gibbons et al., 2002).
Anxiety

- Apprehensive anticipation of future endangerment or misfortune accompanied by a feeling of dysphoria or physical symptoms of tension.

- In ICU setting can have many sources
  - Unstable physiology
  - Pain
  - Apprehension regarding the unknown
  - Impending procedures
  - Seclusion from loved ones
  - Undiagnosed psychiatric disorder

- Sign and Symptoms
  - Cognitive
  - Behavioral
  - Physiologic – Cardiac, Respiratory, Neuromuscular, GI and GU

Management of Anxiety

- Anticipate patient needs

• Nonpharmacological
• Reassure
• Explain
• Encourage verbalization
• Family presence
• Music therapy
• Pet therapy

- Pharmacological
• Benzodiazapines
• Sedatives / hypnotics
• Pain management
Workplace Violence

• One of most accurate predictors of violence is past behavior

• Risk factors
  – Decline in family and community support
  – Easier access to weapons
  – Mental illness
    • Poor impulse control predicts violence
  – Gang prevalence
    • Male, travel in groups, heads down, combat gear can be sunglasses, baseball caps, jackets, vests, or scarves over faces

Workplace Violence

• High risk patients / families
  – Patients admitted with abuse or in an abusive relationship
  – Patients with intoxication or experiencing withdrawal
  – Trauma patients
  – Patients with impaired neurological function or cognition
  – Grieving families

• Disgruntled employees also a source of workplace violence

• AWARENESS IS KEY TO PREVENTION
Delirium Assessment and Management

• **ACUTE** change in consciousness
• Accompanied by inattention AND change in cognition OR perceptual disturbances
• Hyperactive delirium
  – Agitation, restlessness, emotional liability
• Hypoactive delirium
  – Flat effect, withdrawal, apathy, lethargy, decreased responsiveness
• Mixed

**WHAT NOT TO SAY?**

*Well there is a full moon tonight!*  
*This just happens to the elderly when you change their environment!*  
*They will go out of the house!*
Pre-hospital Risk Factors for Delirum

- Baseline cognitive impairment / dementia
- Pre-existing chronic illness including hypertension
- Age > 65 years
- Depression
- Tobaccoism
- Alcoholism

Of the pre-existing risk factors history of dementia, alcoholism, or hypertension are the strongest pre-disposing factors.

Hospital Risk Factors for Delirium

- Diagnosis of congestive heart failure, sepsis (or hyperthermia), or other medical diagnosis with a high severity of illness.
- Prolonged immobility or restraint use.
- Substance withdrawal.
- Intracranial trauma or lesions.
- Use of opioid analgesics and sedatives.

Of the hospital risk factors, severity of illness is most strongly associated with the development of delirium. Benzodiazepine use may also increase the risk for delirium in the critically ill adult patient. Immobility and the use of restraints can contribute to the risk for delirium as well.
Patients most commonly develop delirium 24 to 72 hours after entering the ICU.

It is important to recognize that benzodiazepine withdrawal is a cause of delirium. Therefore, although benzodiazepines are generally avoided in an effort to reduce delirium, they should not be discontinued in patients who routinely take these medications at home.

Prevention

- Analgesia first, sedation second, is a recommended strategy to reduce the amount of required sedation in patients receiving mechanical ventilation.
- In critically ill patients receiving mechanical ventilation, dexmedetomidine infusion rather than benzodiazepine infusion may be associated with lower rates of delirium.
- Daily awakening trials should be performed in mechanically ventilated patients.
- Sleep rest promotion is a nursing driven intervention. Strategies include light and noise control, and clustering of activities to minimize stimulation during normal sleeping hours.
- Early progressive mobility in critical care is one of the most important strategies for the prevention of delirium.

(Barr et al., 2013)
Key Choice / CNEA

Identify the cause using **THINK** pneumonic

- **T**oxic situations
  - CHF, shock, dehydration
  - Deliriogenic meds (tight titration of sedatives)
  - New organ failure (e.g., liver, kidney)
- **H**ypoxemia
- **I**nfection/sepsis (nosocomial)
- **I**mmobilization
- **N**onpharmacologic interventions (Are these being neglected?)
  - Hearing aids, glasses, sleep protocols, music, noise control, ambulation
- **K**+ or electrolyte problems

Expected Practice

- Implement delirium assessment for all critically ill patients using validated tools such as the Confusion Assessment Method for the ICU (CAM-ICU) or Intensive Care Delirium Screening Checklist (ICDSC) [Level B]

- ABCDE Bundle of Care
Key Choice / CNEA

Richmond Agitation Sedation Scale (RASS)
Score Term Description

- +4 Combative
  - Overtly combative, violent, immediate danger to staff
- +3 Very agitated
  - Pulls or removes tube(s) or catheter(s); aggressive
- +2 Agitated
  - Frequent non-purposeful movement, fights ventilator
- +1 Restless
  - Anxious but movements not aggressive vigorous
- 0 Alert and calm
- -1 Drowsy
  - Not fully alert, but has sustained awakening (eye-opening/eye contact) to voice (>10 seconds)
- -2 Light sedation
  - Briefly awakens with eye contact to voice (<10 seconds)
- -3 Moderate sedation
  - Movement or eye opening to voice (but no eye contact)
- -4 Deep sedation
  - No response to voice, but movement or eye opening to physical stimulation
- -5 Unarousable No response to voice or physical stimulation
Procedure for RASS Assessment

• Observe patient
  – Patient is alert, restless, or agitated. (score 0 to +4)
• If not alert, state patient’s name and say to open eyes and look at speaker.
  – Patient awakens with sustained eye opening and eye contact. (score –1)
  – Patient awakens with eye opening and eye contact, but not sustained. (score –2)
  – Patient has any movement in response to voice but no eye contact. (score –3)
• When no response to verbal stimulation, physically stimulate patient by shaking shoulder and/or rubbing sternum.
  – Patient has any movement to physical stimulation. (score –4)
  – Patient has no response to any stimulation. (score –5)

ABCDE Bundle

• A wakening and B reathing Trial Coordination (the Wake Up and Breathe Protocol)
• C hoice of Sedative
• D elirium Detection
• E arly Progressive Mobility and E xercise
• F amily
Post ICU Syndrome (PICS)

• Life-altering condition experienced by patient and family
• Complex syndrome occurring after critical illness encompassing new or worsening impairments in
  – Physical status
  – Cognitive function
  – Mental health
• Implementation of SCCM Pain, Agitation and Delirium Guidelines intended to decrease incidence of PICS
• Originally included ABCDE Bundle
• Now ABCDEF
  – Recognizes importance of family

Patient’s Speak

http://www.icudelirium.org/testimonials.html
ALCOHOL WITHDRAWAL

Can occur within hours of cessation – usually 2-3 days
Usually lasts for 48-72 hours but could be longer

- A. Cessation of (or reduction in) alcohol use that has been heavy and prolonged.
- B. Two (or more) of the following, developing within several hours to a few days after criterion A:
  - (1) Autonomic hyperactivity (eg, sweating or pulse rate 100/min)
  - (2) Increased hand tremor
  - (3) Insomnia
  - (4) Nausea or vomiting
  - (5) Transient visual, tactile, or auditory hallucinations or illusions
  - (6) Psychomotor agitation
  - (7) Anxiety
  - (8) Grand mal seizures

Signs of Alcohol Withdrawal

- C. The symptoms in criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- D. The symptoms are not due to a general medical condition and are not better accounted for by another mental disorder.
Alcohol Withdrawal Delirium

- Disturbance of consciousness (i.e., reduced clarity of awareness of the environment), with reduced ability to focus, sustain, or shift attention.
- A change in cognition (such as memory deficit, disorientation, or language disturbance) or the development of a perceptual disturbance that is not better accounted for by a preexisting, established, or evolving dementia.
- The disturbance develops in a short period (usually hours to days) and tends to fluctuate during the day.
- There is evidence from the history, physical examination, or laboratory findings that the symptoms in criteria A and B developed during, or shortly after, a withdrawal syndrome.

Treatment

- Control agitation
  - Rapid control reduces adverse events
  - Sedative hypnotics are preferred
    - Reduce mortality, Reduce duration of symptoms, Fewer complications
    - Diazepam (5mg)
    - Lorazepam 1-4 mg q 5-15 minutes
    - Haloperidol can be used with above
Other Treatment Strategies

• Neuroleptic agents
  – In conjunction with Benzos when agitation, perceptual disturbances or disturbed thinking are not adequately controlled with benzos.

• Beta blockers
  – To assist with BP and HR control

• Parenteral administration of thiamine
  – At least 3 days
  – 100mg daily

Nursing Considerations

• Close monitoring
• Vital signs
• Include pulse oximeter when significantly loaded with benzodiazapines
• Quiet Room
• Good light and environmental clues – clock and calendar
• Restrains if needed to protect the patient
• Monitor fluid and electrolyte balance
• Treating other comorbidities
• Psychosocial support
Nicotine Withdrawal

- Effect of nicotine on the brain is very rapid and short acting
- Smoking must be repeated to maintain a steady state and avoid withdrawal
- Smoking withdrawal syndrome last for 1-4 weeks

Withdrawal Symptoms

- Agitation and irritability
- Restlessness and difficulty concentrating
- Anxiety
- Depressed mood
- Increased appetite
- Insomnia
- Intense urge to smoke

Hospitalized patients may benefit from short term use of benzodiazepines

Post Traumatic Stress Disorder

- After shocking, dangerous, or frightening event
- Symptoms usually begin within 3 months but can begin years afterwards
- Must last > 1 month and interfere with work or relationships
  - 1 or more re-experiencing symptom (i.e. flashback)
  - 1 or more avoidance symptom (i.e. avoid thinking about)
  - 2 or more arousal and reactivity symptoms (i.e. easily startled)
  - 2 or more cognition and mood symptoms (i.e. feelings of blame)
- Can recover in 6 months or can become chronic
- Antidepressants and psychotherapy
Suicidal Ideation

• Estimated 1 million worldwide suicides per year (potential for under reporting)
• Estimated 2.1 to 18.5% of persons have seriously considered suicide
• Suicidal attitudes and ideation can be precursors to suicide
• 14 suicidal attitude scales and 15 scales for suicidal ideation in the literature (2015 meta-analysis)
• No gold standard for assessment scale
• Direct inquiry recommended

Barriers to Self-Care Management

• Higher acuity
• Multiple needs
  – Co-morbidities
• Shorter LOS
• Noncompliance
• Transportation issues
• Financial concerns
• Depression / anxiety
• Lack of knowledge
• Literacy
• Multiple medications
• Fear of medication side effects
• Living alone (lack of social support)
• Memory problems
Transitions of Care

- Hand offs
- Medication reconciliation
- Coordination / facilitation of care
- High risk populations
  - Elderly
  - Chronic disease states
- Models for transitions of Care
  - Dr. Eric Coleman
There is joy in our work when our work makes a difference!!