Care of Patients with Altered Consciousness

Types of Neurological Insults
ROUGHLY neuro insults can be lumped into just a few broad categories:
- Ischemic Damage (Stroke or Mechanical)
- Hemorrhagic Damage (Traumatic or Spontaneous bleeds.)
- Inflammatory Damage (Vasculitis, Infection). Less likely, more difficult to determine.
- Presence of foreign mass (Tumors)
- What do they have in common?
- All of the insults cause alteration in normal brain function ultimately by diminishing or obliterating blood supply to a specific brain region or by distorting the anatomy via compression and causing malfunction of neuronal pathways. Brain is dependent on constant supply of glucose and oxygen...even small disruptions can cause damage. A prolonged injury can lead to profound and permanent deficits.

Terminology
- Consciousness = Defined as a state of awareness of self and the environment that includes cognitive and affective mental functions. An alteration of consciousness impairs the ability to interact with the external and internal environment and the ability to respond to change.
  - awareness- do you know the world is there?
  - arousal- are you awake? Arousable to pain, voice, not at all?
  - attention- can you focus on a task/object/idea? Can you do something?
- Alteration in Consciousness
  - confusion
  - lethargy (difficult to arouse to verbal stimuli)
  - stupor (needs vigorous tactile stimuli to arouse)
  - obtundation (non responsive)

Continuum Prognosis of brain injury (near total to brain death)
- Near total (example was someone who pretty much seems OK but may have a few residual problems)
- Brain death (body is still alive, heart is still beating, but the brain is dead).
- The mechanism of injury and the severity of injury as well as the aftercare determine where you are going to land on this continuum.
  - Coma
  - Persistent Vegetative State
  - Locked In Syndrome (a type of coma in which the pt is completely aware but totally paralyzed)
  - Brain Death

Critical Focus for the Patient with Altered LOC
- Declining level of consciousness - needs immediate attention! The brain wins every single time when there is a struggle between organs in the body.
  - Airway – can the patient maintain an airway (GCS <8 = intubate)
  - What is happening with blood pressure - Higher BP is needed to perfuse the brain. BP management is extremely important!
  - Overall goal: control swelling and maintain perfusion in the brain to minimize injury

Cerebral Physiology
- Metabolism (brain needs constant supply of glucose, oxygen and removal of waste products)
- Blood Flow (use the CPP to monitor blood flow)
- Cerebral Perfusion Pressure (CPP)
- Cerebral Spinal Fluid (CSF)...acts as the waste-removal system and cushioning for the brain.

- Cerebral Physiology: Metabolism
  - Brain uses energy (glucose) and oxygen at a very high rate. (15% of body’s metabolism)
  - Neuron activity requires that energy and oxygen must be delivered on a second-by-second basis
• The brain CANNOT live more than 4-6 minutes without oxygen. Cell death occurs rapidly with decreased delivery.
  • however, hypothermia can lengthen this time period.
• Only 2 minute supply of glucose stored in neurons

• **Cerebral Physiology - Cerebral Blood Flow and Metabolic Control**
  • Takes 15% of Cardiac output
  • Metabolic Issues:
    • PCO2 (Normal is 35-45)
      • Causes vasodilatation of cerebral blood vessels and increases blood supply. In the head, when you get vasodilation, you actually get an INCREASE in the blood supply.
      • Increases carbon dioxide levels have been shown to increase swelling in the brain and the TBI patient must be "hyperventilated" (maintained with purposely decreased levels of CO2) to prevent brain swelling. This will blow off the CO2 to allow blood vessels in head to **vasoconstrict and decrease blood supply (which decreases ICP)**
    • H+ Ions decrease pH → stimulate respiratory drive
    • Oxygen deficiency: Cortical neurons very sensitive to drops in O2 → causes changes in LOC very quickly. This is very important for any type of patient...If you’ve got somebody who is otherwise "pretty normal", who all of a sudden starts to get a little loony...check an ABG even if O2 looks ok. Even slight drops can cause neurological changes.

• **Cerebral Physiology: Arterial Blood Pressure & Sympathetic Nervous System Control**
  • Blood Flow: MAP 60-140mm Hg (140 is way too high)
  • Cerebral Arteries constrict with increased MAP and dilate with decreases MAP = Autoregulation. Brain tries to autoregulate, but when you have an injury the SNS takes over...
  • SNS takes over when Autoregulation fails due to low or high pressures (but doesn’t autoregulate properly)
  • Vasospasm of Cerebral vessels can alter flow
    • spasm after hemorrhage into Sub-arachnoid space. When you hav blood that has leaked out of the vessels the blood sitting on the bessel is very irritaing to them, the vessel spasms, ayou get decreased blood flow downstream from there and you get signs of stroke.

• **Cerebral Physiology: Cerebral Perfusion Pressure (CPP)**
  • CPP = MAP-ICP ICP is the amount of pressure being exerted on teh brain. The CPP will fall as the ICP increses.
  • Normal ICP is 15-20...if you have a lot fo swelling, teh ICP will go up. Say it goes up to 30...the brain can’t push the scull out, it’s going to collapse soft tissue of teh veels, and you’ll hav decreased blood flow. If MAP falls, there is less pressure driving blood up to the brain.
  • Average CPP Pressures: 60-80 mm Hg. At less than 50 you get ischemia, but can reverse if you get on it quickly. If less than 40 for any length of time you’ll see death of neurons. Over 80 you get hyperemia...can also cause damage.
  • CPP falls as ICP increases
  • CPP falls as the MAP decreases
  • If the CPP falls too low the brain is deprived of O2
    • CPP < 50 mm Hg = ischemia
    • CPP < 40 mm Hg = death of neurons
    • CPP >180 = hyperemia

• **Cerebral Physiology: Inter-cranial Pressure (ICP)**
  • The ICP is the measure of pressure with in the skull that is being exerted on the soft tissues and CSF in the ventricles.
  • Normal ICP is less than 15 cm H2O. For practical purposes, we uses less than 20 as the normal. It’s another number to tell us if pressure in the brain is increasing.
  • An increase in ICP represents an increase of pressure. It MUST be controlled.
  • Monroe-Kellie Hypothesis
    • Total volume must remain the same
    • You’ve got blood, brain, CSF...those are the only things that are supposed to be there. If anything extra gets in there (tumor or contusion or blood that has leaked out of vessel), this throws things off and ICP will go up.
Why Does ICP Rise?

- Intracranial Compliance
  - Small changes in volume of all the things in your brain halt compensation (i.e., obstruction of CSF outflow). When you sneeze, ICP shoots up greater than 100, it's instant...brain gets a little squirt of CSF that runs down your spine/drainage system. It's a very temporary, automatic compensatory mechanism. What happens when compensation fails, you start seeing problems...can come from bleeding, tumors, obstructive venous outflow (clotting of the large veins that drain the blood from the brain...so all that blood that gets pumped into brain can't escape). The CSF vein is VERY narrow and susceptible to infections such as in meningitis...the CSF can't flow down the spinal cord and be reabsorbed like it's supposed to.

- Obstructed Venous Outflow
  - Various positions and activities impair venous return

- Cerebral Edema
  - Cytotoxic effects
  - Vasogenic effect from injury

What do you think are signs of increased ICP? Every cell lives for oxygen...symptoms do you see with brain cells that are not getting enough oxygen?

- Headache
- If caused by a tumor or bleed, may see lateralizing symptoms? (don't know what this means)
- Confusion?
- He didn’t really answer this question....so look in Big Red!

Herniation

- Herniation...A MEDICAL EMERGENCY. This is when you start to see pupil changes.
- Shifting of the brain from one compartment of high pressure to one of lower pressure...This is a late sign.
- Causes pressure on the medullary area of the brain stem structures and an ↑ in neurological deficits or death can occur.
- Pupillary changes—unequal, unilateral then bilateral, → Cushing’s Triad: bradycardia, systolic hypertension, widening pulse pressure.

NCLEX REVIEW QUESTION

Which nursing assessment finding would identify the earliest indication of increasing intracranial pressure?

- 1. Temperature over 102°F (you can see any and all types of VS changes)
- 2. Change in level of consciousness (Rationale: as cerebral hypoxia develops the patient becomes restless and drowsy well before any other signs and symptoms of increasing intracranial pressure are present).
- 3. Widening pulse pressure (that's toward the end game, not an early sign)
- 4. Unequal pupils (not an early sign)

NCLEX REVIEW QUESTION

A patient is admitted for closed head injury following a motor vehicle accident (MVA). Which intervention should the nurse plan to include in the patient’s care to minimize increased intracranial pressure?

- 1. Maintain the Head of the Bed (HOB) at less than 10 degrees.
- 2. Group all nursing activities and leave the patient undisturbed for 2 hours.
- 3. Maintain alignment of the head and neck. (positioning the client to maintain the head and neck alignment will facilitate venous drainage and prevent increased intracranial pressure due to venous congestion.). Sit them up greater than 30 degrees and keep head straight! Even if in full spinal precautions, tip the entire bed up.
- 4. Elevate the patient's legs.

Incorrect Answers Rational

Answer 1 is incorrect because the HOB should be maintained at greater than or equal to 30 unless contraindicated.

Answer 2 is incorrect because grouping multiple nursing activities like turning, bathing, linen change, suctioning will dramatically increase intracranial pressure for an extended amount of time. To minimize increases in intracranial pressure nursing activities should be planned to give the client 30-60 minute breaks between activities. The intracranial pressure...
will increase with any activity, but the pressure will have an opportunity to decrease to safer levels if activities are not clustered. So partially correct...it is important to LIMIT stimulus, but you wouldn’t let a freshly injured pt sit for hours without being monitored.

**Answer 4** is incorrect because positioning to prevent increases in intracranial pressure is focused on alignment of the head and neck and elevation of the HOB at 30 degrees.

**QUICK REVIEW**

↑ ICP can be caused by...:
- ↑ brain volume
- ↑ blood volume
- ↑ CSF

• **Cerebral Physiology: A note on CSF....**
  - Clear fluid secreted by the choroidal plexus in the ventricles—subarachnoid—spinal column.
  - It bathes the brain and spinal column, acting as shock absorber
  - Helps remove waste products from the blood.
  - Constantly being produced and reabsorbed by the body (about 800 mL/day.)

**Invasive Monitoring: Measure ICP using an EVD**

- External Ventricular Drain. Drill a small hole at a 90-degree angle at level of ear ...if there’s pressure, the fluid will come out under it’s own volition. There are HUGE problems if it comes out too fast, so do not place the drain lower than the level of the ear...it would drain too fast and cause huge huge problems. Also need to level it, zero it...usually these are tunneled underneath the skin, so not a huge infection risk...but still need to watch. They do daily cultures of the fluid to monitor for infection.
  - Therapeutic drainage of CSF
  - Measure ICP continuously
  - Placed into the ventricles (most often the lateral)
  - Can be used to monitor ICP and to remove CSF and blood from the ventricle
  - Risks of EVDs include: infection, clogging, bleeding along insertion track, creation of hydrocephalus, and pneumocephaly.
  - NEVER flush one of these toward the brain...always make sure the stopcock is closed to the brain. ONLY FLUSH OUT!
  - A little less invasive...
    - Fiber optic Catheter (Camino Bolt)
    - Most often placed just past the dura resting in a few mm’s in the brain tissue opposite the injury. As the heart beats and blood supply comes up to the brain, teh brain shivers a bit with each beat. We can look at this movement of the brain to determine ICP.
    - Advantages- Decrease risk of infection, may be more accurate when ventricles are empty or compressed.
    - Disadvantages- fragile (cable is fiberglass), expensive, cannot drain CSF when ICP’s climb.

**Neurological Assessments**

- Least invasive and most common way to monitor for ↑ ICP
- Hourly assessments unless condition warrants otherwise
- Bedside exam at change of shift...when going out or coming in...do the exam with nurse who is coming on or off. There are some subjective differences in the way people do exams. This gets everyone on the same page.
- Document accurately
- **TIME = BRAIN**
Assessing Level of Consciousness (LOC)

- Standard Bedside Neuro Exam should include a Glasgow Coma Scale check:
  - Total Score = 3-15
- Eye Opening Score
  - 4= open spontaneously
  - 3= eye opens to voice
  - 2= if I have to pinch you or sternal rub (to pain)
  - 1= no eye opening
- Verbal Score
  - 5= oriented (alert and oriented x3)
  - 4= confused
  - 3= inappropriate words (somebody who is just mumbling real words, may be out of context)
  - 2= incomprehensible sounds (grumbling, etc...)
  - 1= no response
- Motor Score
  - 6= follows commands (don’t use ‘squeeze my hand’, b/c this is a reflex. ask them to show you a thumbs up, two fingers, wiggle something.
  - 5= localizes (when asking somebody to localize, you are presuming they are not following commands and you’ve had to cause a painful stimuli...sternal rub pt should try to stop you or move toward the stimulus...coming across midline is a good sign that your brain knows there is pain on that opposite side of injury/paresis. You can use lower extremities as well...pt may try to kick or reach down)
  - 4= withdrawal (if you do a sternal rub and they have withdrawal or flexion it looks a lot the same. the way you can test withdrawal is to pinch fingernails or toes...they will pull away.
  - 3= flexes (if flexion, they should just flex, not pull away)
  - 2= extension (whether doing central or peripheral pain, will extend)
  - 1= no response

Mental Status
- Glasgow Coma Scale (GCS)
- < 10 is concerning!!!!!!
- Look at the scale (dead=3)

Level of Consciousness
Single most important indicator of neurological status

Verbal Exam
- Effects on speech can range from mechanical (stroke, paralysis) to cognitive (damage to speech or hearing centers)
- Assess for orientation to self, place, time, etc.
- Learn to recognize receptive and expressive aphasia. Almost always are expressive aphasias (speech is difficult to initiate, and writing may be difficult as well, language is reduced). NOTE: Some patients will appear confused simply because they cannot process sound or cannot verbally express clear thoughts. Simple yes/no questions may be easier for them to respond to.

Motor Exam
- Evaluate function/movement/strength of extremities comperar right to left
- If patient does not follow commands:
  - Sternal Rub or Supra-orbital pressure may be used to assess response to central stimulus.
  - Peripheral (nail bed) pain may be used if patient has no central response.
- Decorticate (flexion)
- Extension (will arch and come up off bed)
Pupil Exam (tells us the least info!)
- Note the size, shape, reactivity to light.
- Movement is the most important...we are testing cranial nerves with this.
- When looking to see if pupils are blown, we are looking at 3rd cranial nerve.
- Dilated pupils occur on side of injury b/c the oculomotor nerve is the only one that runs straight back without any components of it crossing over to the other side. If there pupil is blown, we know something is pushing on that nerve (blood or brain...probably herniation and this is a LATE sign).
- Note patients gaze (fixed, disconjugate)
- Vision Field
- EOM's (extra ocular movements)...looking for eyes to move together.
- Can see weird-shaped pupils with older ptsw ho have had cataract surgery.
- Pupils
  - Extra-ocular movement:
  - Corneal reflex...brush very corner of eyeball with sterile gauze, should blink
  - Doll's eyes (see handout)
  - Cold water calorics...60ml syringe of ice-cold water...will see “eye bobbing” if brain stem is intact.

Critical GCS Changes
- A decrease of 2 points or more warrants an immediate call to the MD.
- Even small changes are important! Your early intervention can mean the difference between a good and bad outcome!
- Knowing your patients baseline is critical.

Critical Vital Sign Changes (can be anything!)
- Temperature ↑ or ↓
- Respirations will be pretty normal until serious stem damage.
  - Cheyne stokes and Central Neurogenic Hyperventilation
  - ↑ RR
- Cushing’s Triad (pending herniation)
  - Widened pulse pressure
  - Bradycardia
  - Slowed respirations

Other Causes of Altered LOC (there are a LOT of reasons for a person to be “whacked out”, this is just a sampling)
- Metabolic Disorders
  - Electrolytes (Na,K,Mg, PO4)
  - Acid/Base Imbalance (respiratory or renal)
  - Liver Failure (NH3)
  - Hypoglycemia or Hyperglycemia (very common cause!)
  - Hypoxia (bleed, respiratory failure)
  - Overdoses (narcotics, antidepressants, ETOH)
  - Sepsis (infection)
- Tumors
- Seizure

Types of Head Injuries
- Ischemic Stroke
  - Ischemic Stroke is the third leading cause of death in the United States killing 160,000.
  - The greater tragedy is the remaining 500,000 patients who will survive their stroke but go on to have significant neurological deficits. Care for these folks is extremely expensive!
  - Occurs when arteries become occluded by clots or by gradual narrowing secondary to atherosclerotic plaques (narrowing is more common than clots thrown via A-fib)
  - This represents about 85% of stroke.
• Clots generally are of a cardioembolic source, but can be created by arterial dissection or abnormal clotting diseases.
  • Common Stroke Symptoms (either ischemic or hemorrhagic!)
  • Acute onset of lateralized weakness/numbness/tingling
  • Acute onset of confusion, difficulty speaking
  • Acute onset of trouble walking, loss of balance/coordination
  • Acute onset of severe HA
• Early detection is key...CODE GREY
• If a patient makes it to the ER in under 3 hours and qualifies for TpA, it can be administered to attempt to revascularize the occluded vessel.

NCLEX TIP! AN ABSOLUTE CONTRAINDICATION TO TPA IS A BLOOD IN THE BRAIN ON A HEAD CT.

Treatment/Prevention of Ischemic Stroke
• If stroke secondary to cardioembolic source, patient will require anti-coagulation or anti-platelet therapy
• If stroke secondary to atherosclerotic disease, will need some level of anti-platelet therapy (ASA, Aggrenox, Plavix)
• Will need rigorous control of risk factors include HTN, Hyperlipidemia, DM
• PT/OT/ST for Rehab

Hemorrhagic Conditions
• Cerebral Aneurysm (can fix these!)
  • Weakness in vessel wall generally thought to be congenital in origin, but is often seen in conjunction with hypertension HTN.
  • HUGE headache when this happens...usually those who make it to the hospital will not have deficits
• Subarachnoid Hematoma
  • Subarachnoid hemorrhage (SAH) implies the presence of blood within the sub-arachnoid space from some pathologic process.
  • That process is usually a ruptured aneurysm or AVM (arterio/venous malformation)
  • Headache due to meningial irritation (painful to have blood on meninges), presence of blood also cause of vasospasm
  • An estimated 10-15% of patients die before reaching the hospital. Mortality rate reaches as high as 40% within the first week. About half die in the first 6 months.
  • Findings:
    • Most commonly occur when an aneurysm has ruptured and bleeding has begun
      
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<tr>
<th>Early</th>
<th>Late</th>
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<tr>
<td>Headache</td>
<td>Lethargy</td>
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<tr>
<td>Violent Headache</td>
<td>↓ LOC</td>
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<td>Neck pain</td>
<td>Focal neurological deficits</td>
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<td>N/V</td>
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• Aneurysm Treatment: Coiling vs. Clipping and placement of EVD for Hydrocephalus
  • Risks of Coiling or Clipping
  • Vasospasm (causes ischemic stroke)
  • Bleeding
  • Nursing Care: Assess for changes in Neuro status, monitor blood pressure to maximize CPP.
  • Vasospasm: Triple H Therapy: Hypervolemic expansion (flood with fluid), hypertension, and hyperosmolar therapy to increase CPP. Nimodipine a Ca channel blocker is also ordered to decrease smooth muscle contraction of the blood vessels that may increase vasospasm. Vasospasm status lasts a few weeks...so if you can keep them stable through this they may walk out of the hospital doing pretty good.

AVM’s (arterial venous malformations..these are congenital malformations where arterial beds are supposed to meet up)
• Pre and Post Cerebral Angiogram of a repaired AVM (see picture)
• Cut off blood supply with super glue...then go in with surgery to cut out the “rats nest”.

Facts about Head Injuries
- 50% of patients die from traumatic brain injury before they even reach the hospital
- Most common causes are MVA's, assault and falls, other causes are aneurysm rupture, congenital malformations and stroke.
- Drugs and ETOH frequently associated with occurrence.

Phases of Head Injuries
- Primary
  - Initial injury
  - Results in tissue or vascular disruption
  - Irreversible
- Secondary (swelling)
  - Results from subsequent events or complications
  - Ischemia is the end result

Traumatic Injuries
- Epidural Hematoma
- Subdural Hematoma
- Intracerebral Hematoma
- Skull Fractures
- Diffuse Injuries

Epidural Hematoma (fixable with usually NO complications if treated quickly)
- Epidural hematoma (EDH) is a traumatic accumulation of blood between the inner table of the skull and the dural membrane.
- The inciting event often is a focused blow to the head, such as that produced by a hammer or baseball bat.
- In 85-95% of patients, this trauma results in an overlying fracture. Blood vessels in close proximity to the fracture are the sources of the hemorrhage.
- Because the underlying brain has usually been minimally injured, prognosis is excellent if treated aggressively.
- EDHs are usually stable, attaining maximum size within minutes of injury
- Surgical treatment usually consists of placement of burrholes and/or evacuation of the clot.
- Observation while the clot reabsorbs is the most common treatment.

Subdural Hematoma (a “bigger deal”...see with trauma and old alcoholic guys, or older folks who are on plavix and bump their head a bit)
- An acute subdural hematoma (SDH) is a rapidly clotting blood collection below the inner layer of the dura but external to the brain and arachnoid membrane. (he said it was a slow leak?)
- May not show any symptoms b/c it is a slow leak and brain may compensate. First signs may be clumsiness.
- Venous bleeding of bridging veins dissects the arachnoid away from the dura.
- Acute SDH’s carry a 60-90% mortality.
- Chronic SDH seen in elderly, particularly those with a history of coumadin therapy or ETOH abuse.
- Subdural Hematoma Treatment
  - Can possibly do burrhole if necessary...but the leak may still be going. A lot of time just leave them alone b/c the blood should be reabsorbed.
  - Obtain an immediate head CT scan in patients with head trauma who experienced clear loss of consciousness, are symptomatic, are disoriented/amnesic, or have any focal neurological signs.
  - Consider intubation when GCS score is less than 12 or other indications are present; this guarantees airway protection during the diagnostic workup.
  - Check labs, especially INR, and reverse with Vit. K and FFP prn. (VERY IMPORTANT!)
  - The lesion may represents clotted blood, if so a burr hole is not curative, and emergent craniotomy is necessary.
**Intracerebral Hematomas**

- ICH/Hemorrhagic Stroke used interchangeably.
- In ICH, bleeding occurs directly into the brain parenchyma.
- The usual mechanism is thought to be leakage from small intracerebral arteries damaged by chronic hypertension.
- Patients with intracerebral bleeds are more likely to have headache, altered mental status, seizures, nausea and vomiting, and/or marked hypertension; however, none of these findings distinguish reliably between hemorrhagic and ischemic strokes.
- In addition to the area of the brain injured by the hemorrhage, the surrounding brain can be damaged by pressure produced by the mass effect of the hematoma.
- Traumatic ICH (multiple areas of bleeding) see picture

**ICH Treatment**

- Careful BP monitoring is important (mainly if not a traumatic one, but caused by HTN). Goal is NOT to decrease BP, but to keep BP pretty darn high to keep brain perfused (the chances of bleeding from same site is almost nil). No controlled studies define optimum BP levels.
- Greatly elevated BP is thought to lead to re-bleeding...so keep at or slightly under 200.
- Stroke patients may lose their cerebral autoregulation of cerebral perfusion pressure.
- Although BP elevations may risk further hemorrhage, too rapid or aggressive BP lowering may compromise cerebral perfusion.
- Current guidelines for BP management in hemorrhagic stroke were developed by consensus. Current recommendation is to treat BP if the systolic is greater than 200 mm Hg or the diastolic is greater than 100 mm Hg on repeated measurements over 30-60 minutes. Readily titratable medications are recommended. Labetalol or nitroprusside are 2 options.
- Rarely surgical evacuation of a clot/infarcted brain tissue or removal of a bone flap is required to relieve pressure and prevent herniation. Will only do if life/death situation. Fungination = mushroom cap...don’t roll them on their side...don’t poke the brain (duh). It is a last-ditch effort. Skin is closed over the top...and acts as a barrier for infection.

**Diffuse Brain Injuries**

- Concussion
  - Rapid movement or sharp blow acceleration/deceleration injury
  - Temporary loss of consciousness
- Post-Concussive Syndrome
  - Results in mild neuro impairment, amnesias, irritability and insomnia. (N/V, headache)
  - Recovery is usually full
- Diffuse Axonal Injury (these are terrible and global...render you completely “jacked up”)
  - High speed and rotational acceleration forces
  - Non-rigid brain moves forcibly within the rigid skull
  - Most severe form of brain injury
  - Shearing of neuronal connections, microscopic damage to to axons.
  - Brain stem remains intact, but the rest of “you” has been wiped out.
  - Most common form of persistent vegetative state.
  - Diagnosis of exclusion. Often used to explain lack of consciousness when there is no other explanation.
- Fractures
  - Depressed
    - Variety of types
    - Results from high velocity contact over skull
    - Infection and seizures common complicatons
  - Basilar Skull (less obvious). The dura that increases the CSF gets torn easily here...
    - Periorbital ecchymosis (raccoon eyes)
    - Mastoid ecchymosis
  - CSF leaks
    - Clear fluid from nose (Rhino) or ears (Oto)
    - Check fluid using an Accu-Check. Glucose levels should be about 2/3 of the Blood glucose level.
• CSF will “halo” on a paper towel, sheet, tissue. This halo affect is created by the less dense parts of the CSF traveling further along the porous membranes of the fabric. (Will be darker with a lighter halo around it...drool, sputum, and blood will not do this!)
• A leak is serious b/c now there is a direct route if infection into the brain.
• Space occupying lesions (in this case, meningioma). If you are going to get a brain tumor, you want meningioma...very slow-growing, usually pretty easy to get out.

NCLEX REVIEW QUESTION
The client is admitted for c/o headache, new onset (less then 2 hours) left hemiparesis, aphasia, and an imaging report indicating a positive Computed Tomography (CT) scan of the head. The RN knows that this indicates:
• The client has a hemorrhagic cerebral vascular accident (CVA) and should receive a thrombolytic agent.
• The client has an ischemic cerebral vascular accident and should have a thrombolytic agent.
• The client has a hemorrhagic cerebral vascular accident and should not receive a thrombolytic agent.
• The client has an ischemic cerebral vascular accident and should not receive a thrombolytic agent

Case Study
Ray is 25 year old Caucasian male admitted 3 days ago for unrestrained MVA involving ETOH abuse and a telephone pole. He suffered multiple facial injuries. He received an emergency tracheostomy in the field for crushed larynx and in currently in the NSICU in C-Spine precautions, with a ventriculostomy drain to keep ICP at less the 15cm. His ICP is draining 12-18cc/hr (this is the high side of normal, but OK). His vital signs are stable. His is on vent at PC 32, peep 5 rate 24, PS, Fio2 55%. His gases are have pH 7.45, HCO3 22, PaC02 34, PaO2 87.

Case Study Questions:
• What type of brain injuries is he at greatest risk for? hemorrhagic type injury
• What is the goal for his ICP monitoring reading? want it to be under 20
• What is the nursing management to maximize his recovery? maintain O2 and ventilation, maintain perfusion, constant neuro evaluation with hourly assessments.

Herniation
• Herniation (most common= Uncal herniation, unilateral mass usually of the temporal lobe increased ICP pushes lower temporal lobe (uncas) down into midbrain putting pressure on cranial nerve #3.
• Central herniation downward displacement of hemispheres, basal ganglia, and diencephalon through the tenorial notch ...death
• Shifting of brain tissues from an area of high pressure to low pressure.
• There are several types of herniations, but all result in destruction of large portions of the brain, most commonly the brain stem.

Diabetes Insipidis (“Dumping Dilute Urine)
Lots of water in the urine. Head injury that affects pituitary can cause this.
• ↑ urine output (> 200cc/hr for two hours)
• Urine specific gravity < 1.005
• ↑ serum osmolality (pt is dehydrated so serum is very concentrated). Death occurs pretty rapidly when serum osm goes up. Normal is around 280-ish...as it starts to get higher, you get significant damage.
• extreme thirst
• management: Vasopressin (Pitressin) Antidiuretic hormone, increases reabsorption of H20 at distal renal tubules.

SIADH (Syndrome of Inappropriate Antidiurectic Hormone)...opposite of Diabetes Insipidus
• ↓ serum osmolality (dilute blood because of high H20)
• hyponatremia (low Na, diluted) (this will lead to seizures if it’s a fast drop or a slow big drop)
• ↓ urine output (400-500 cc/24 hrs)
• weight gain
• Focus Question: What would you expect the urine osmolality to be?_______________
Management of SIADH
- fluid restriction
- sodium replacement
- diuretics (Lasix) (last resort)

Seizures (very common with traumatic injury)
- Caused by lesion itself or mass effect of the lesion.
- Most common first medication is Dilantin (also use Cerebrex?), loaded with 1 gm IV, then 300 mg PO QHS
- Works by increasing seizure threshold
- Acute seizures treated with IV Ativan. If seizure doesn’t stop, need to paralyze and sedate (put on vent!)

Case Study Continued
Case Study: You have been caring for Ray for 2 hours and note that his urine output last hour was 370, and this hour 360. You look back on his urine output for night shift and note that he has had outputs of 200-300 for the last 6 hours. He has not been given a diuretic.
What is Ray at risk for? Diabetes Insipidus
What should you do with this information? Check the urine specific gravity
What lab tests would you anticipate? Serum osmolality, electrolytes, urine sp.gr.
Ray’s serum osmolality comes back at 329 (high), Na (146) (high) and his urine specific gravity is 1.002 (low). What treatment would you anticipate? hormones! Needs ADH!

Our patient...
Bob is a 22 y/o male who was sitting on his porch, minding his own business drinking a 40 when a couple of big dudes came up to him for no reason and proceeded to beat him about the head with spare tire irons. Bob had them all whipped until that last big dude sucker punched him. A concerned neighbor called the EMS after she noticed Bob seemed to be taking a nap on the lawn after the rowdy fun he was having with his pals. He was summarily whisked away to the UCDavis Med center. After a brief stay in the ER (ha ha) he’s admitted to the NSICU for observation.

Nursing Management of Neuro patients
- The treatment proceeds in a stepwise manner, from least invasive to most invasive.
- The patients presentation determines where they will start out at along the continuum.
- Most treatment is geared to decreasing the ICP, maintaining cerebral perfusion, decreasing ischemia and maintaining body functions for patients who need ventilation and sedation.

Our patient’s injuries...
- Scattered frontal lobe ICH’s on the left (frontal and temporal)
- R humerus fx
- Multiple bruises and lacerations
- He is in a C-spine collar until his spine can be cleared (can’t do this until sober)
- Currently on RA with no s/s of respiratory distress
- HR = 80 with a NSR
- Blood Alcohol = 220
- In the ER he is noted to be difficult to arouse (is this because of alcohol to head injury?). When he’s awake AAox3, PERRL and brisk, moving all extremities (MAE) 5/5 except for his RA which is in a sling. He quickly falls asleep without stimulation (alcohol or head injury?).
  - strength score 5=strong, 4=a little weak, 3=can move against gravity, 2= can drag hand but can’t lift, 1=trace movement...little bit of movement in toes if you ask, 0=none.
- He also seems quite irked.
**NCLEX QUESTION**
If Bob's LOC continued to wax and wane, what would your first priority be?

- a) Control the bleeding from his lacerations.
- b) Soft cast his fractured humerus
- c) Start a Normal Saline bolus maintain his cerebral perfusion pressure.
- d) Intubate in order to secure his airway

**Signs and Symptoms of increasing ICP**
- Increased Headache, N/V, photophobia, decreased alertness, decreased strength, agitation, somnolence, neck stiffness/pain, change in pupils or vision fields (late sign!), altered respiratory pattern, DI, change in vital signs.

**Bob's current complaints**
- C/O pain from HA (6/10) and pain in his arm (5/10)
- He doesn’t like the Foley
- He wants to call his mom.
- He is a bit fidgety and restless, says the pain is keeping him from resting.

**How can we help our buddy Bob?**
- Decrease Environmental Stimuli (underrated but very important!)
  - Keep room dark
  - Keep room quiet
  - Keep room cool
  - Limit visiting
  - Group nursing care
  - Positioning: HOB @ 30 degrees, neck strait
- PRN Sedation, Pain Control and Meds for N/V and Seizure Prevention
  - Morphine/Dilaudid
  - Ativan/Versed/Haldol
  - Zofran, Tigan, Compazine
  - Dilantin 1 gm loading dose, then 300 mg IV TID
- Caution must be used, as these treatments may sedate a patient. Without a neuro exam it is hard to follow your patients course. Over sedation will earn you a trip to CT!

**A few hours later your neuro check on Bob shows......**
- He is unarousable. Will barely eye open to pain
- Will only mumble incomprehensible sounds.
- Will localize briskly with the left extremities but only weakly on right (injury to left side of brain has caused damaged d/t more bleeding or more swelling)
- Bob is still breathing, but is becoming sonorous (snoring) and needs a couple litres of 02 to keep Sats > 92. If someone who wasn’t snoring starts, this is not a good sign.
- You notify the MD and get a stat head CT which shows significant expansion of the intracranial bleed with 4-5 cms of shift and compression of the ventricles. (see picture →)

**EVD placement**
- At the point where a patient is requiring prn meds, or rapidly decompensates, an EVD might be placed either to monitor ICP or drain hydrocephalus created by the bleed. You don’t see the worst of the swelling until about 48 hours, so you know more swelling is coming with this guy.
- This may be done at the BS, but often is done in the OR.
- If done at the bedside, the RN is responsible for setting up the drainage system.
EVDs and the Bedside RN

- Once the EVD is placed, it is the RN’s responsibility to manage it.
- The MD will write orders, ie.:
  - EVD @ EAC, OTD for ICP > 20 x 5 minutes. (external auditory canal)
  - EVD clamped at EAC, check ICP q1hour. Notify MD if > 20
- The RN must zero the EVD at each shift and check the waveform to assess its accuracy.
- Accurately record the ICP and CSF drainage. Sometimes the drainage requires hourly notation.

Bob’s got an EVD now.

- Bob’s ICP is initially high, but after drainage, his pressures fall to < 20. Bob begins to wake up a bit. He is now eye opening to voice, will occasionally follow commands. (This doesn’t last usually more than a couple hours)
- This lasts a couple of hours. Bob’s ICP begins to climb, he becomes restless and agitated. PRN meds for sedation are not bringing his ICP down.
- Bob is now levitating (thrashing a lot). Will drive BP up, his ICP’s are jumping into the 30’s. Though his CPP’s are okay at this point, he still needs to be better controlled.

When heavy sedation is required....

- Morphine, Versed and Vecuronium Drips may be ordered. (more current is Propofol...clears in about 10 mins for the neuro exam and put them back down again)
- When a patient is paralyzed you must be sure to provide appropriate eye, skin, and bowel care. Careful attention to DVT prophylaxis is also necessary.
- Of course, if you paralyze a patient, they must be ventilated.....
- The primary complication of decreased LOC is inability to protect the airway and aspiration or regurgitated stomach contents and/or foreign bodies in oropharynx

Ventilating the Head Injury Patient

- If the patient is not paralyzed and breathing on their own, sedation for comfort should be used.
- If the pt is vented, then the nurse will have to carefully monitor ABG results, and titrate the vent settings appropriately

ABG goals for Neuro patients

- Often times the MD will ask that you adjust the vent to achieve a goal PCO2 of 30-35. WHY? if you write for a narrow range, you’ll get a narrow range. CO2 causes vasodilation...if you vasodilate you are delivering MORE blood to brain, which raises ICP. Our goal is to keep ICP down. So you want the pt to blow off CO2 in order to keep ICP down.
- You can change the rate, TV, an I:E time to achieve this goal.
- Maintain PO2 levels at normal or better, usually > 80

ABG for Bob

- PO2 60, O2 sats 96, CO2 26, ph 7.53, HCO3 22
- Vent settings 100% FiO2, Peep 5, PS 10.
- Pt extremely agitated, but not following commands. His current RR is 28 and he is pulling TVs of 600-700.
- The CO2 and pH is no bueno. How will you fix it?
  - Give morphine to sedate...no changes to vent will help at this point. Paralyze him, then make changes to vent.

Bob, Bob, Bob.....

- Bob is now vented, paralyzed and sedated. Another head CT reveals that his ventricles are almost “empty”. You no longer have the option of removing fluid to make room to decrease pressure b/c all the fluid is already gone. Your dampened ICP waveform confirms this.
- Your head of bed is raised is 30 degrees and the neck is strait to facilitate CSF drainage. (your would have done this earlier but Bob wouldn’t cooperate.)
- Yet his ICP continues to rise....uh-oh!
**Osmotic Diuretics**
- Will pull free water out of the brain tissue itself and excrete it.
- So, you’ve got your patient sedated, ABG dialed in, but you ICP is still too high. The EVD won’t drain, the HOB is elevated, you’ve done all you can do, what now?
- The MD may order you to administer Mannitol or 3% Normal Saline. With mannitol, the sugar will eventually be deposited into brain tissue and eventually pull fluid back into the brain.
- Osmotic Diuretics work by creating a hyperosmolar state in the blood. As it passes the brain, water is removed and edema improves.
- Must monitor Sodium level and Serum Osmolality carefully!
- Current studies suggest that you not allow the Na to be greater than 150, but generally the Serum Osm. > 320 is the true cut of, despite Na levels of 160-180.
- Mannitol
  - Is a sugar solution
  - Administered by weight (gm/kg)
  - Must be administered quickly by hand pump in order to be effective. Use central line or large bore peripheral IV, just pump it in as fast as possible. Will see diuresis almost immediately.
  - May require cc/cc replacement due to profound diuresis. Be ready!
- 3% Normal Saline
  - Ordered in 100 or 200 cc doses
  - Elevation in Na and Serum Osm. levels transient,
  - Not quite the diuresis seen with mannitol.
  - Still, must monitor carefully, especially with repeated doses.
  - Frequent rapid infusions of hypertonic saline have been associated with pontine demylenations. So, don’t do this too quickly b/c it removes myelin sheath from neurons in pons. TERRIBLE!!!

**Bob is getting pickled…**
- After alternating doses of Mannitol and 3%NaCl you manage to get his ICP’s down for a few more hours.
- Now your lab results show a Na of 163 and a Ser. Osm of 325. You’ve hit ceiling for osmotic diuretics, cannot do anymore. This option is now exhausted.
- To compound matter, he’s lost several liters of fluid during diuresis and now his BP is falling.
  - Blood Pressure Management
  - Remember CPP = MAP-ICP
  - Bob’s BP = 112/56 MAP = 70
    - ICP = 25   CPP = 45
  - Is this OK? NO! This is leading toward ischemia and brain death!
  - we want to get BP up, but don’t want to give more fluid b/c this will increase ICP. We will give neosynephrine

**Neosynepherine** (shunts blood from peripheral into central)
- Alpha-adrenergic drug ordered to keep CPP> 70.
- Dosed in mcg/min, starting dose 50 mcg, titrate by 20 mcg up to 300 mcg.
- Peripheral vasoconstriction spares cerebral circulation.
- Very rapidly cleared, just seconds to see effects.
- Decreases peripheral pulses, can cause bowel ischemia (could lead to ostomy). At high doses fingers and toes will become cold, blue, even necrotic. Severe vasoconstriction occurs at higher doses...you’ve gone too far when fingers are blue. Will become very hard to find artery for ABGs, so I hope you already have a good line in)
- You’ve done great, but…..ICP still too high.

**Bob’s current status:**
- Paralyzed and sedated
- On max dose of neosynepherine
- Na and Serum Osm. Levels too high to give more osmotic diuretics.
- ABG with in desired parameters.
- ICP wave dampened and EVD not draining (tells us ventricles are “empty”)
- ICP = 28  Map = 82  CPP = 54....there’s one last tier of medical management for our buddy Bob...
Barbituate Coma and Diprivan are the next tools in your toolbelt!

- Pentobarbital or Diprivan gtt.
- These drugs essentially cut the brain off from the outside world. If the brain is not receiving input, the "processing" requirement is reduced, ergo the oxygen, energy and waste removal requirement is reduced.
- Hopefully if we decrease stimuli we decrease energy demands and decrease ischemia. Can be effective in young, healthy patient. We use an EEG to determine if they are processing anything.

Bob is in la-la land.

- Bob is now in a deep chemical coma. This is determined by examination of the bedside continuous EEG. This simple EEG registers brainwave activity. The absence of activity indicates coma induction.
- Despite this, Bob’s ICP is still high. You’ve done all you can do. Now its time for the surgeons to go work.
- Surgical Intervention
  - Craniectomy- Removal of portion of the skull to allow for brain expansion out of the cranium and reduction of pressure (funginating brain pokes out through hole)
  - Lobectomy- Removal of partial or complete lobes of the brain. Removes dead brain tissue which will create room for more swelling. Removal of necrosed tissue also reduces edema associated with the bodies response to the dead brain. It's difficult to get ONLY dead brain tissue...this is a last-ditch effort.
  - CT of patient with craniectomy (see picture)

Sometimes you just can’t do enough

Despite your best efforts, Bob has had no improvement in his ICP.

- The neurosurgery team has decided to d/c the sedation and paralytics a few days ago
- Bob’s neuro exam is now as follows:
  - extends to deep sternal pain, no spontaneous movement
  - has a cough, gag and corneal reflex
  - occasionally over breathes the ventilator (a spontaneous breath occasionally)
  - ICP’s still in mid 20’s, CPP’s still less than 60
- While you are doing your charting late in the evening you notice some drastic vital changes.....

Bob’s last hurrah...

- HR jumps to 156, then reverts to 50
- ICP shoots to 110, then a very flattened ICP waveform reads 14 (this has happened b/c a big portion of his brain just went out through the bottom of his skull...so plenty of room)
- BP on the a-line climbs to 180/110 for a moment, then falls to 100/65
- Bob’s pupils are now fixed and dilated. He has no spontaneous breaths, no movement.
- You decide now would be a good time to call the doctor. You would have called during this episode, but seeing as it all happened in less than a minute, that would have been hard.

Herniation

- Shifting of brain tissues from an area of high pressure to low pressure.
- There are several types of herniation, but all result in destruction of large portions of the brain, most commonly the brain stem.
- Signs and symptoms of impending herniation.
- Pupil changes- fixed and dilated due to 3rd nerve compression
- Elevated ICP (can go above 100)
- Increased BP/Pulse rate initially as cathecholamine surge is triggered by the event.

Brain Death

- Brain death is defined as the time when when the entire brain, including the brain stem, has irreversibly lost all function.
- Brain death is determined primarily by physical exam, but can be confirmed by imaging studies that can identify whether or not there is blood flow to any areas of the brain.
Brain Death Exam
- Cough, gag, corneal reflexes absent
- Deep pain stimulation
- Apnea test (turn off vent and look for signs of brain stem function)
- Pupil exam
- Pt. is flaccid and areflexive (no tone, no peripheral reflexes either, may see twitches in legs)
- Oculovestibular reflex- (Cold Caloric) The patient's ear canal is inspected to ensure an intact tympanic membrane and that the ear is free of wax. While holding the eyes open, ice water is injected into the ear canal. The drastic change in ear temperature will cause a violent eye twitching by the intact brain but no reaction in the brain-dead patient. This is performed in both ears.
- Oculocephalic reflex (Dolls Eyes)- The patient's eyes are opened and the head turned from side to side. The active brain will allow a roving motion of the eyes; the non-functional brain will not. The eyes remain fixed. (If your eyes don't move as you move the head, then you have problems...You want to have a NEGATIVE dolls eye's.)
- Cerebral Blood Flow Study- a simple form of angiogram. Radio-opaque dye injected and x-ray of head taken. Areas of brain still alive will show blood flow.
- EEG- shows any electrical activity in a living brain. Will be flat in a dead brain.

Bob's Brain didn't make it....
- The main focus of your nursing care at this point is helping the family work through this period.
- Discussions on the patients desires regarding donation need to be addressed, but this is something that takes some time to learn.
- Don't be disappointed!
- Many people with severe head injuries either die or have very poor function post injury.
- What we learn from each patient helps us to do a better job each time.
- Sometimes just you being there and doing your best is all you can do.