



## Understanding Management of Neurological Emergencies

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## Managing a Train Wreck



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## General Physiology Cranial Vault

- Intracranial cavity: 1500 ml
  - Brain (87%) 1300 ml
  - CSF (9%) 140 ml
  - Blood (4%) 60 ml
- 420 ml CSF made per day thus adults cycle through CSF 3x per day

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## General Physiology Intracranial Pressure (ICP)

- ICP is pressure within the cranial vault
- ICP varies with activity but normal ICP in resting patients is around 0-10 mm Hg
- ICP > 20 while at rest is considered elevated
- Several processes can elevate ICP
- Elevated ICP adversely affect the brain function and outcome because of decreased cerebral perfusion

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## General Physiology Cerebral Perfusion Pressure

- CPP = Mean arterial pressure (MAP) - ICP
- Maintaining cerebral perfusion is a very important priority in the management of CHI
- CPP of less than 70 mm Hg in Adults and 60 mm Hg in children is generally associated with a poor outcome
- In the presence of elevated ICP, it is absolutely important to maintain normal or supernormal BP unless a clinical source of cerebral hemorrhage is identified
- Watch out for drugs that may lower BP (i.e. Propofol)

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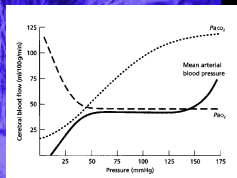
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## General Physiology Cerebral Blood Flow (CBF)

- $CBF = CPP / CVR$  (cerebrovascular resistance)
- Normal CBF is approximately 50 ml/100g of brain / min
- Autoregulation maintains CBF fairly constant within a Systolic Blood Pressure (SBP) range of 50-160 mm Hg unless there is elevated ICP
- When oxygenation / CBF falls substantially
  - Cushing's Triad
    - HTN
    - Bradycardia
    - Respiratory Irregularities




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## General Physiology Cerebral Blood Flow (CBF)

- Ischemic penumbra 8 – 23 ml / 100g brain / min
- At 23 ml / 100 g of brain / min functional impairment ensues
- Less than 8 ml / 100 g of brain / min results in cell death and irreversible damage

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## General Physiology Cranial Volume

- Since the cranium is non-expansile, the total volume of intracranial contents must remain constant - Monro-Kellie Doctrine
- Addition of a mass such as a hematoma results in removal of an equal volume of CSF and venous blood up to a point
- Once this compensatory mechanism is exhausted, there is exponential increase in ICP

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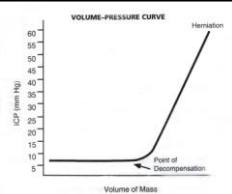
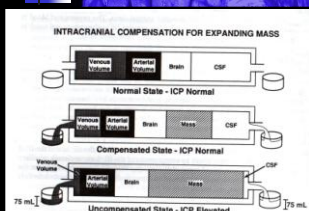
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## General Physiology Pressure Volume Relationship



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## General Physiology Fluids and Physiology

- 3 fluid compartments
  - Intracellular
  - Extracellular
    - Interstitial
    - Plasma
- Location of Ions
  - K<sup>+</sup> intracellular
  - Na<sup>+</sup> extracellular
  - Separation of ions creates electrochemical gradient
  - Gradient set up by Na/K ATPase and maintained by impermeability of cellular membrane to these ions
  - HSD used in shock/trauma as volume expander

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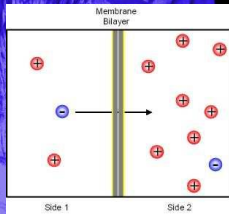
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## Calculating OSM

- $2 \times \text{Na}^+ + \text{BUN}/2.8 + \text{Glu}/18$
- Normal 280 – 290 mOsm/L
- Serum OSM changes maximally with increases in Na<sup>+</sup> and less so with glucose which is co-transported and BUN which can freely cross membranes
- Other solutes cause osmotic changes as well
- The difference between the calculated (expected) and the measured serum osm is known as the osmolar gap (mannitol, toxins, etc.)
- 200 ml 20% mannitol = 100 ml 7.5% Na<sup>+</sup> in 6% Dextran Solution (Hypertonic Sodium Dextran)



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## Brain Physiology and Tonicity

- Blood Brain Barrier
  - Tight junctions
  - Not water impermeable but solute impermeable
  - Fluid shifts with osmotic agents occur in areas with intact BBB
  - Osmotic agents may lodge in areas of incompetent BBB worsening ICP
    - Thought to occur with larger molecules (mannitol)
- The effects of cellular dehydration are seen principally in the CNS, where stretching of shrunken neurons and alteration of membrane potentials from electrolyte flux lead to ineffective functioning

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## Effects of Tonicity on Cells

(a)  
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## Causes of Elevated ICP

- Mass Lesion
  - Blood
  - Neoplasm
- Edema
  - Extracellular
    - Vasogenic – leaky vessels / tumor
    - Vasomotor instability – trauma / reactive hyperemia from CVA
    - Non Vasogenic
  - Intracellular / Cytotoxic
- Hydrocephalus

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## So Far...

- Brain in a fixed volume compartment (calvarium).
- As contents in calvarium increase, pressure increases exponentially.
- As pressure increases, blood flow decreases and ischemia occurs.
- Increased ischemia results in increased edema and the cycle continues to increase ICP.
- Understand the mechanisms of edema and the physiology behind them.
- Evaluate patients often using physical exam and image as necessary.
- Maintain ACLS protocol throughout evaluation and treatment.

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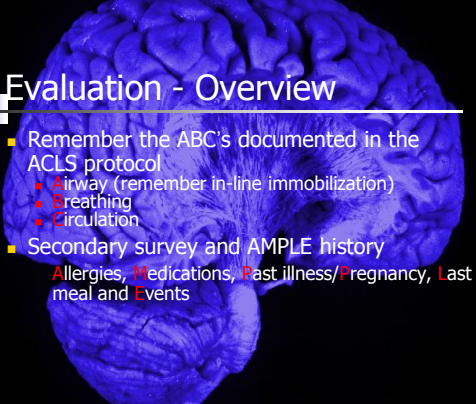
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## Evaluation - Overview

- Remember the ABC's documented in the ACLS protocol
  - Airway (remember in-line immobilization)
  - Breathing
  - Circulation
- Secondary survey and AMPLE history
  - Allergies, Medications, Past illness/Pregnancy, Last meal and Events

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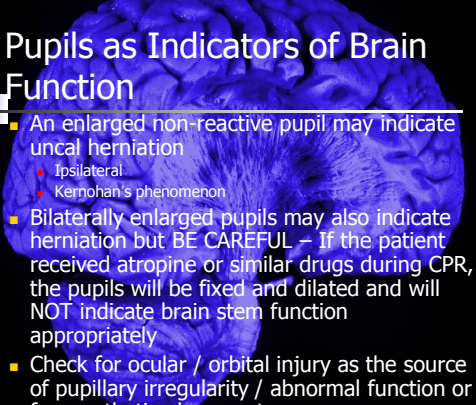
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## Pupils as Indicators of Brain Function

- An enlarged non-reactive pupil may indicate uncal herniation
  - Ipsilateral
  - Kernohan's phenomenon
- Bilaterally enlarged pupils may also indicate herniation but BE CAREFUL – If the patient received atropine or similar drugs during CPR, the pupils will be fixed and dilated and will NOT indicate brain stem function appropriately
- Check for ocular / orbital injury as the source of pupillary irregularity / abnormal function or for prosthetic placement

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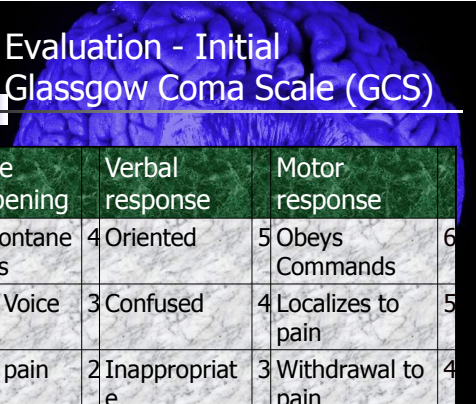
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## Evaluation - Initial Glasgow Coma Scale (GCS)

Eye Opening	Verbal response	Motor response	
Spontaneous	4 Oriented	5 Obeys Commands	6
To Voice	3 Confused	4 Localizes to pain	5
To pain	2 Inappropriate	3 Withdrawal to pain	4

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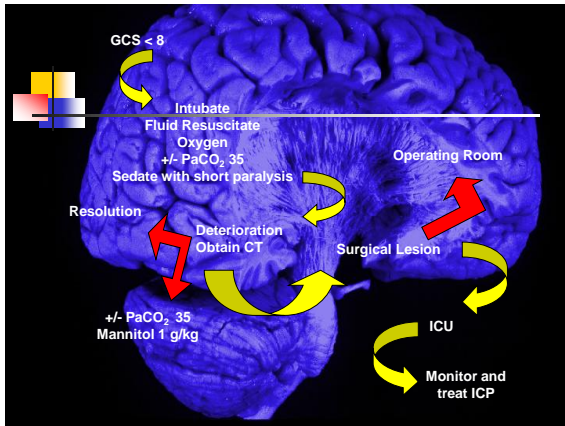
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### Evaluation- Short Term

- Q 1 hour neurological checks watching for decline in mental status or seizures
- Repeat CT scan if condition deteriorates or if hemorrhage / edema noted on CT as part of routine follow-up at a scheduled interval

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### Invasive ICP Monitoring

- A patient's ICP is monitored because elevated ICP will reduce cerebral blood flow
- The consequences of decreased CPP are hypoxia and subsequent stroke
- Stroke then leads to increased swelling and further elevation of ICP
- This vicious cycle is extremely difficult to rectify and often results in poor patient outcome
- Typically ICP > 20 mmHg sustained for over 5 minutes requires treatment

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## Invasive ICP Monitoring

- CT and MRI not able to determine pressure, imaging can infer high ICP
- Place ICP device when INR < 1.3 for ventriculostomy and < 1.5 for strain gauge monitor
- Platelets > 100k
- Indications – GCS < 7
  - Eyes closed
  - No verbal (includes if ET tube placed)
  - Localizes to noxious stimulus

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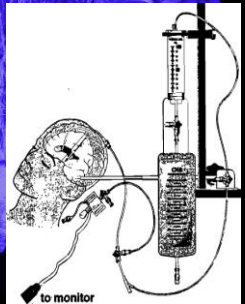
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## Invasive ICP Monitoring

- Strain Gauge ICP Monitor
  - Codman
    - Works by altering small resistance changes in electrical current = higher pressure = less resistance
  - Camino
    - Works by altering light path through a fiber optic cable
- Ventriculostomy




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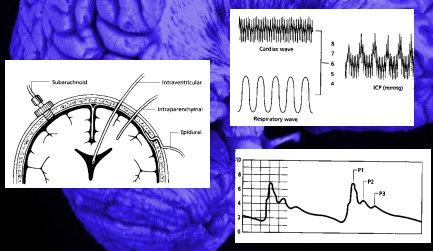
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## Invasive ICP Monitoring




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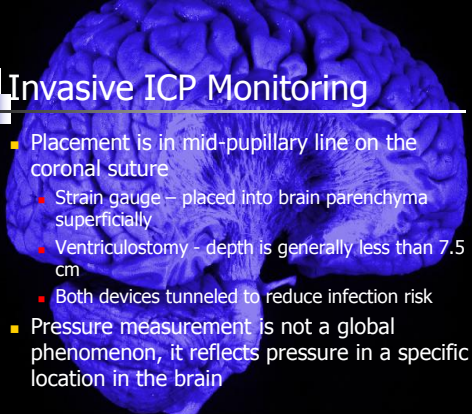
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## Invasive ICP Monitoring

- Placement is in mid-pupillary line on the coronal suture
  - Strain gauge – placed into brain parenchyma superficially
  - Ventriculostomy - depth is generally less than 7.5 cm
  - Both devices tunneled to reduce infection risk
- Pressure measurement is not a global phenomenon, it reflects pressure in a specific location in the brain

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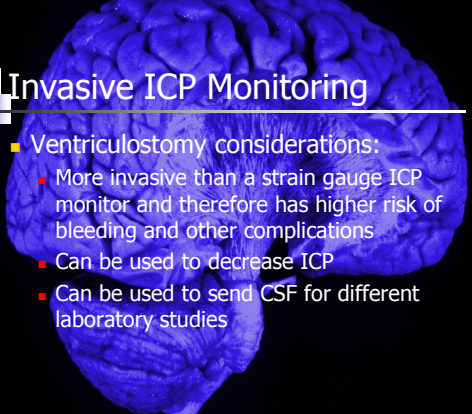
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## Invasive ICP Monitoring

- Ventriculostomy considerations:
  - More invasive than a strain gauge ICP monitor and therefore has higher risk of bleeding and other complications
  - Can be used to decrease ICP
  - Can be used to send CSF for different laboratory studies

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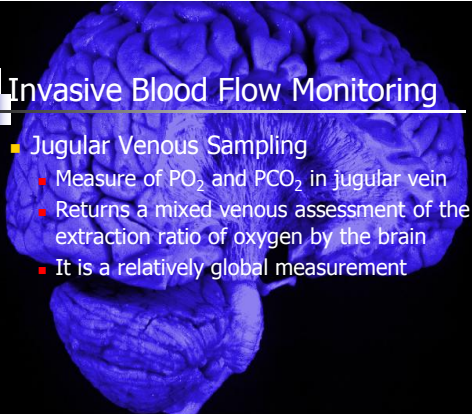
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## Invasive Blood Flow Monitoring

- Jugular Venous Sampling
  - Measure of  $PO_2$  and  $PCO_2$  in jugular vein
  - Returns a mixed venous assessment of the extraction ratio of oxygen by the brain
  - It is a relatively global measurement

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## Invasive Cerebral Chemistry Monitoring

- LICOX
  - An implanted device similar to a strain gauge monitor but much larger
  - Measures ICP, pH, O<sub>2</sub> content
  - Is a local measurement only and will generate false data if placed into injured brain. Best placed in normal, functioning brain for routine assessment

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## So Far...

- Best evaluation of patient status is the *physical exam*.
- If physical exam limited or if there is a physiological question that needs to be answered continuously, consider invasive monitoring strategies.
- Invasive monitoring is not without risk.
- Choose the monitoring strategy based on risk benefit ratio for that patient and the physiological needs of the situation.

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## Initial Management Strategies

### *General*

- Maintain adequate nutrition
- Range of motion via PT / OT
- Infection Surveillance
- DVT prophylaxis with venodynes (SCD's) at a minimum / SQ heparin (LMWH etc.)

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## Initial Management Strategies

### *Ventilation*

- See that patient is protecting their airway
- Check that patient is moving air appropriately (chest rise and fall, adequate respiratory rate)
- Sedatives on board?
- Pulse oximetry
- ABG to evaluate  $PO_2$  and  $PCO_2$ 
  - Goal  $PO_2 > 60$
  - Goal  $CO_2$  33 – 37
  - Hypocapnia increases ischemia risk but may be useful as an adjunct to emergent surgical decompression
- Better to intubate rather than risk poor ventilation

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## Initial Management Strategies

### *Ventilation*

- Remember, intubating a patient often involves sedative and paralytic agents that will have serious implications when trying to assess neurologic function later on
- Thus, if it is absolutely necessary to intubate a patient, use short acting medications
- An adequate intubation history is imperative (What drugs were used? When were they last used on the patient?)

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## Initial Management Strategies

### *Blood flow basics*

- Elevate HOB  $> 45$  degrees if adequate blood pressure present to prevent intracranial venous pooling
- See that the neck is not kinked as this may increase ICP by resisting venous outflow in the jugular veins
- Avoid internal jugular central lines if possible – especially introducer / Swan-Ganz catheter. (better used in subclavian vein)
- Avoid vasodilating agents such as nitroprusside unless absolutely necessary
- Maintain adequate MAP – may need ICP device for this for goal CPP  $> 70$ mmHg. Typically SBP  $> 90$  mmHg

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## Initial Management Strategies

### *Steroids*

- Not definitively established in adults
- Not for cytotoxic edema (stroke/trauma)
- Potential candidates are those with organisms on gram stain, especially with high ICP (Clin Inf Dis, 1993; 17:603-610)
- Dexamethasone 10 mg every 6 hours for 4 days improved outcome in patients with cloudy CSF, bacteria on gram stain, or CSF WBC count  $>1000/\text{mm}^3$  (NEJM, 2002; 347:1549-1556)

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## Initial Management Strategies

### *Temperature*

- Keep temperature  $< 37^\circ\text{C}$  (normal)
- Hypothermia
  - Use in patients already ventilated
  - May need paralytics to keep from shivering which will raise ICP.
  - Risks include decreased cardiac output, coagulopathy, pancreatitis

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## Initial Management Strategies

### *Electrolytes*

- Normal  $\text{Na}^+$  level 135 - 145
- Keep glucose normal – elevated glucose worsens cerebral edema and ICP
- Maintain normal levels of bicarbonate and  $\text{CO}_2$ 
  - $\text{CO}_2$  crosses BBB and gets converted into  $\text{H}^+$  and  $\text{HCO}_3^-$
  - The lower pH will increase pressure (cause vasodilatation) and will cause potentially irreversible damage / denaturation.

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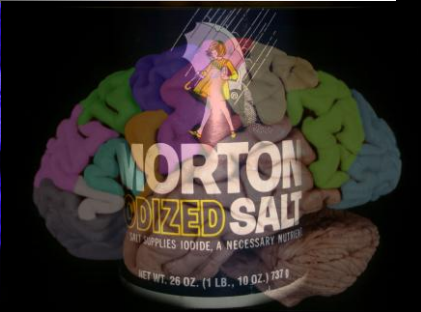
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## Brain Physiology and Tonicity



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## Brain Physiology and Tonicity

- Since  $\text{Na}^+$  is extracellular,  $\text{Na}^+$  will equilibrate between interstitial fluid and plasma
- If a relatively small amount of BBB is incompetent but most is not, the increase in  $\text{Na}^+$  (or other extracellular osmotic component) will effectively dehydrate cells in the intact regions
- When large areas are effected the reverse may occur and ICP may paradoxically increase
- This may be partially responsible for the rebound effects of bolus osmotic therapy

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## Brain Physiology and Tonicity

- After an hour of hypernatremia, intracellular organic solutes are generated in an effort to restore cell volume
- If shrinkage is severe enough, stretching and rupture of bridging veins causes subarachnoid hemorrhage
- Cerebral edema ensues if water replacement proceeds at a rate that does not allow for excretion of accumulated solutes

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## Osmotic Approaches to Decreasing ICP

- Diuresis / Indirect Increases in Extracellular Osmolarity
  - Mannitol
  - Urea
  - Lasix / HCTZ
- Direct Increases in Extracellular Osmolarity
  - Mannitol
  - Hypertonic Na<sup>+</sup>
  - Hydroxyethyl Starch (HES) or Colloid

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## Diuresis

- Decrease water free water volume
- Retained solutes draw more fluid back in to the extracellular space resulting in decreased intracellular water and hopefully decreased ICP
- Kidneys must work exceptionally hard to retain solutes against gradients set up by the diuresis of plasma volume (OSM > 320 can cause ATN and renal failure that usually resolves over time) however, dead nephrons can remain dead nephrons
- Mannitol can have direct toxicity to the renal tubules
- When plasma volume is low, renal blood flow is decreased and thus less O<sub>2</sub> is delivered to the cells to produce ATP for the ATPase Na<sup>+</sup>/K<sup>+</sup> pumps that are actively trying to reabsorb Na<sup>+</sup> to maintain plasma volume
- Eventually renal failure will occur

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## Direct Increase in Plasma Tonicity

- Albumin / HES / Na<sup>+</sup> etc.
- Cause diuresis by directly expanding plasma volume
- Increase in blood flow to kidneys and potentially blood pressure increases GFR and diuresis
- Increase in Atrial Natriuretic Factor (ANF) released by atria due to stretch from increased plasma volume
- If Na<sup>+</sup> is used, ATPase will slow down (less metabolic demand since plasma volume is high and Na<sup>+</sup> is high
- Na<sup>+</sup> is thus spilled into the urine

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## Difference Between Hypovolemic and Hypervolemic Hyponatremia

- Diuresis
  - Higher renal metabolic demand with lower renal blood flow
- Hypervolemia
  - Lower renal metabolic demand with higher renal blood flow

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## CNS Benefits of Hypervolemic Hyponatremia

- Improved CBF
  - Decreased ICP
  - Increased MAP
    - Vasospasm
    - Ischemia
- Unlike mannitol or colloid solutions,  $\text{Na}^+$  is dialyzable by CVVH (as is Urea)

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## Problems With Osmotic Therapy

- Rapid changes in tonicity can lead to CPM, especially with  $\text{Na}^+$ 
  - Do not let  $\text{Na}^+$  change by more than 12 mmol/L up or down each day
  - Most CPM cases in thiamine deficient patients (i.e. EtOH abuse) and in patients with rapid correction of chronic hyponatremia
  - Patients that develop sudden DI even with  $\text{Na}^+$  of > 170 mmol/L often do not develop CPM but the risk does remain
- Consider maintenance drip of hypertonic solution as opposed to bolus doses

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## Problems With Osmotic Therapy

- Rebound effect after osmotic bolus or sudden cessation of continuous osmotic agent
- Rebound effect thought to be worse with bolus  $\text{Na}^+$  than with other osmotic agents (Javid 1964)

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## Benefits of Hypertonic $\text{Na}^+$

- Improved gas exchange
- Improved cardiac output
- Improved MAP
- Hemodilution / Rheostatics benefit
- Immunomodulation

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## Adverse Effects of $\text{Na}^+$

- Electrolyte derangements
  - Mild acidosis that is often clinically insignificant
  - Concomitant dose with THAM or acetate to normalize pH 2<sup>nd</sup> to hyperchloremic acidosis
- Congestive Heart failure
- Bleeding diathesis
- Phlebitis
  - Use hypertonic  $\text{Na}^+$  through a central line only unless using 1.8%  $\text{Na}^+$  solution

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## So Far...

- Maintain normal physiological parameters for most things.
- Cause certain metabolic derangements in a controlled fashion to help manage ICP but do so to preserve other organs as best as possible.
- Use basic physiology to decide when to treat and what to treat with.

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## Sedation

- Decreases brain metabolism with only certain sedative medications
- Relaxes patient / less agitation
- If ventilator dependent, can help blunt coughing / "bucking the ventilator"

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## Paralytic Medications

- Forces muscle relaxation
  - No valsalva
  - No muscle related vascular tone
  - No ventilator / ventilation ICP related problems
- Must have 1-2 twitches to avoid myopathy
- Must limit length of use and concomitant use with steroids to help avoid myopathy

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## Barbituate / Propofol Coma

- Decreases cerebral metabolic rate
  - Decreases neuron function directly
  - Decreases temperature which indirectly decreases neuron function
  - CSF has 3 components
    - Choroid plexus 70%
    - Transudate 18%
    - *Metabolic production of water*
- Forces full relaxation of CNS if burst suppression obtained
- Neuroprotective – decreased demand for resources

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## So Far...

- HOB > 30 degrees
- Neck Straight
- Maintain normothermia – hypothermia in rare instances
- Maintain normal Na<sup>+</sup> level
- Use hypernatremia / mannitol when ICP is being measured or as a bridge to when ICP can be measured
- +/- usage of other chemistry measuring devices for all patients but in certain situations these devices may be valuable
- Place ICP device when necessary

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## Conclusion

- ICP management issues can be basic or very complex
- Control those things that are within your realm
  - Elevate head of bed
  - Head should be mid-position
  - Ensure appropriate ventilation
  - Normalize temperature
  - Normalize electrolytes
- Call for help when you're not sure what to do next

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