Shaken Baby Syndrome
“Silenced Angels”
Anesthesia Implications
Case Study

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Outline

• Overview of Shaken Baby Syndrome
• Case Study: Pediatric Head Trauma
• Anesthesia Implications
• Problem-Based Learning with Audience Participation
• Questions & Answers
What Type of Cases will we See?

- Orthopedic
- Neurosurgery
- Ophthalmic
- Trauma
- General Surgery: Feeding Tubes etc.
- Sedation for LP: r/ o Meningitis
History

- More than 1000 children die each year in the US as a result of child maltreatment (McClain, 1993)
- Of these at least 250 are victims of Shaken Baby Syndrome
- 1996: First National Shaken Baby Conference
Although the SBS was not formally described until 1972 by John Caffey, it can be traced back at least 500 years.

Nostradamus (1555) made a prophecy relating to subdural hematomas and their relationship to trauma and abuse.
History Lesson

- James Parkinson (1799): well known for describing “shaking palsy”
- Became of the first physician to publicly condemn child abuse
- He addressed the issue of head trauma and its implications directly
- “Dropsy of the Head”: subdural hematoma
Caffey
“Whiplash Shaken Infant Syndrome”

- 27 Cases
- 15 cases were found to be attributed to a nurse, Virginia Jaspers: Newsweek Magazine, 1956
- They tied this in to Nurse Jaspers’ massive physical traits. (6’, 220 lbs)
Shaken Baby Syndrome

- Reflects a subset of injuries caused by abusive head trauma (AHT) and is a part of Silver’s (1962) “Battered Child Syndrome”
- There is a pattern of abuse injuries associated with SBS with and without impact
Medical Components

- Retinal Hemorrhages
- Subdural or Subarachnoid Hemorrhage
- Associated Fractures
- Other External Physical Findings

It is the often the absence of external signs of abuse that makes the early diagnosis of SBS so difficult
Incidence

- Difficult to ascertain
- The perpetrator is reluctant to provide an accurate history
- Most children with injuries seen with SBS without obvious external trauma are presumed to have been shaken (Hadley et al., 1989)
Risk Factors

- Perps described as having reversed nurturing needs
- They are looking to be nurtured by their infants
- When this does not happen, abuse can occur
Risk Factors

- Ex-preemies or children with handicaps
- People who have admitted to shaking: a child have stated that they were not trying to harm the infant, but were trying to make the baby stop crying
| Perpetrators                  | Males (60-70%)  
|                             | Mother’s Boyfriend (34%)  
|                             | Baby Sitter (4-30%)  
|                             | Mother (6.5%)  |
| Family                      | Single Parent  
|                             | Maternal Age <18 yrs  
|                             | Poor Socioeconomics  
|                             | Maternal Education <HS  
|                             | Known to Social Service  |
| Victim                      | Age <1 yr  
|                             | Male Infants  
|                             | Prematurity/ LBW  
|                             | No PNC  |
Physical Risk Factors of Infants

- Small Body Size
- Large ratio of head (~ 25% of body wt.)
- Weak neck muscles
- Lack of head control
- Un-fused sutures
- High brain water content
- Less myelination of nerve cells
- Large subarachnoid space
Seasonal Variations?

- HMC: “Christmas” 2002
- 5-6 babies with SBS under 8 week of age
- ? Seasonal Variations
- More Common during Holidays?
- Stressors: financial, depression, burdens
Mechanisms of Injury

- Infants are more susceptible to whiplash shaking injuries than older children.
- Relatively large heads supported by weak neck muscles which increases their head movement during shaking.
- Their unmyelinated brain, soft sutures, open fontanelles, and relatively increased CSF result in a brain that is more vulnerable to injury.
Clinical Presentation

- Can be very non-specific
- The infant may present in a coma, with a bulging fontanelle, or with subtle signs such as vomiting, irritability, seizures, poor feeding or failure to thrive
- Some are misdiagnosed as: meningitis
- A “bloody” Tap may be thought to be related to the tap itself rather than a sign of a subarachnoid bleed (also HSV)
Clinical Presentation

- Some argue that retinal hemorrhages were pre-existing from birth trauma
- 14-40% of newborns sustain retinal hemorrhages at birth (Budenz, 1974)
- These usually resolve in several days and certainly within 2-3 weeks
- After the neonatal period any finding of retinal hemorrhage should suggest abusive head trauma
Lab Data

- May present with clotting dysfunction which is reflective of DIC secondary to intracranial trauma
- Mild-moderate anemia
- LP: will be bloody
- Leukocytosis, Electrolyte Disturbances
Making the Diagnosis

- CT is the method of choice for initial imaging. It readily identifies lesions requiring emergency surgery.
- MRI has shown to detect 50% more subdural hemorrhages than CT and can detect smaller injuries missed by the CT.
- The cost and availability of an MRI makes it more useful as a second study.
Prognosis

- Victims of SBS suffer significant morbidity and mortality
- Ludwig et al. (1984) showed a 15% mortality rate and a 50% morbidity rate in their review of 20 cases
- Permanent brain damage, hydrocephalus, developmental delay, blindness, deafness, paralysis and mental retardation have been noted
Long-Term Medical Burden

- High incidence of mortality and morbidity
- SBS accounts for 10-12% of all child-maltreatment deaths
- 25% of victims of SBS will die
- Of those that survive 57% will have neurological complications
- Including: severe motor deficits, seizures, developmental delay, and blindness
Biomechanics of Abusive Head Trauma

- Precise mechanisms of neurological injury remains unclear.
- Injuries occur from rapid and repetitive flexion, extension, and rotation of the head and neck around a relatively stable torso with (Duhaime) or without impact (Dias).
Biomechanics

- The injuries with SBS are not likely caused by falls from short distances, rough horseplay, swinging or rocking baby
- Infants’ brains have a higher water content and less myelination than the adult brain, it is more gelatinous and is easily compressed and distorted from shaking
Shaking Event

FIGURE 4
Spinal cord injury

Violent shaking can cause trauma directly to the spinal cord resulting in apnea and cardiovascular collapse.
Shaking Event
Skeletal Findings

- Subperiosteal hemorrhage is common in child abuse
- The periosteum which surrounds long bones has two layers
- With trauma, the periosteum is stripped from bone and blood accumulates between the cortex of the bone and the periosteum
- The osteogenic layer responds by laying down a thin layer of periosteal new bone
Periosteal New Bone

Subperiosteal Hemorrhage
Diaphyseal Fractures

- Spiral fractures can occur with long bones (femur or humerus)
- In the non-mobile infant, this is highly suggestive of abuse
- In a toddler learning to work: can be Toddler’s Fracture: non-abuse
- Distal clavicular fractures can result from traction of twisting forces applied to the arm
Spiral Fractures
Clavicular Fractures
Rib Fractures

During violent shaking, the perpetrator’s hands wrap around the child’s thorax (left). Note that the fingers are located over the spinal column, where the vertebrae act as a fulcrum, resulting in posterior rib fractures (right). (Reproduced with permission from Lauridsen J, Levin A, Parrish R: Animated Graphic Demonstration of Shaken Baby Syndrome (CD-ROM). Ogden, Utah: National Center on Shaken Baby Syndrome, 2000)
Rib Fractures

Courtesy of Children’s Hospital, Boston: Child Protection Team
Skull and Scalp Injuries

- Infants with significant impact injuries to the head frequently exhibit swelling of a portion of the scalp.

- The majority of skull fractures in abused children occur during infancy (80-85% under age 2), (Lazoritz, 2001).

- Skull fractures do not heal by callus formation thus making the DX difficult.
Skull Fractures

Fracture

Infant skull
What are Patients with Skull Fractures at Risk For?

- A. Meningitis
- B. Intracranial Bleeds
- C. Mike Tyson Syndrome
- D. A&B
Example of Mike Tyson Syndrome
Definition: Volatile Temperament from Repeated Head Trauma

- Mike As Baby
- Mike Now
Leptomeningeal Cyst
AKA “Growing Skull Fracture”
Extra-Axial Hemorrhage

- In the skull three membranes surround the infant’s brain
- Immediately covering the surface of the brain is the pia arachnoid
- CSF is within the subarachnoid space which lies between the pia arachnoid and the dura arachnoid
- The subarachnoid space is normally larger in infants than in older children
Subdural Hemorrhage
Subdural Hematomas with SBS
Post-Mortem View
CT Findings Subdural Bleed & Cerebral Edema
MRI Findings

Coronal View
Parenchymal Brain Injuries

- Subarachnoid hemorrhage usually occurs in association with parenchymal brain injury
- Subarachnoid & epidural hemorrhage are both relatively uncommon with child abuse
- They can, however, occur with associated parenchymal brain injuries with SBS
Shearing Injuries

- Results from angular acceleration during shaking or from blunt impact
- Commonly found at the gray-white junctions
Cerebral Edema

- Results from various mechanisms but is probably an indirect pathophysiological response to head trauma (Aldrick, 1992)
- There is also associated hypoxic-ischemic injury with SBS (apnea). This is caused by increased ICP/ hypo-perfusion
- Diffuse cerebral edema from hypoxic-ischemic injury carries a grave prognosis
- May produce a “Mass-Effect”
- Edema may be present on CT after 2-3 hours “or” not until 1-2 days (when they usually present in ER)
Cerebral Edema with SBS
No Space Between Ventricles
Ophthalmic Manifestations

- Retinal hemorrhages are the most common ocular finding with SBS.
- Cannot be dated clinically.
- Non-ophthalmologists have great difficulty in seeing RH with direct exam and undilated pupils.
- RH can cause loss of vision, but the most common cause of blindness with SBS is a direct bilateral injury to the visual pathways of the brain.
Retinal Hemorrhages

- Occurs in 50-100% (other studies 75-90%) in patients with SBS
- There are different layers of the retina that may be affected
- Hemorrhages include: splinter, dot-blot, and large blot
- Large dome-like bleeds have been observed in the macular area
RH

- Extremely common with birth (50% @ 1st DOL)
- Influenced by age of infant, type of birth, and parity of the mother
- Appears to be less frequent in premature infants (although they are at higher risk for abuse)
- By 3 weeks: RH are generally not r/ t birth unless the birth was extremely traumatic
Iatrogenic Causes of RH

- ECMO
- Heparinization, alterations in cerebral blood flow, major vessel ligation, mechanical ventilation, and hypoxia could contribute to RH
- AVM or Aneurysms could also cause RH
Other Causes of RH

- Meningitis
- Accidental trauma “rarely” causes RH
- Chest trauma can cause RH
- Some RH can be caused by CPR
- Some patients with persistent ROP may have RH and Vitreous hemorrhages
- SIDS: extremely unlikely to cause RH itself or from CPR in these cases
Adult RH Causes

- Valsalva maneuvers
- High-Altitude Hypoxia
- Bungee Cord Jumping
- Whip-Lash Injuries (MVA)
Retinal Hemorrhages

Normal

Abnormal
Medical Management

- Challenging and Frustrating
- Significant brain injuries resulting in life-long physical and cognitive abnormalities
- Death
- History is inaccurate which may delay treatment
- Emotionally taxing to staff
Brain Injury

- Injury caused by direct trauma
- Hypoxic-ischemic injury
- Secondary injuries as a result of the trauma and hypoxic-ischemia
Treatment

- ABC
- Endotracheal Intubation for Glasgow < 8, apnea or cardiac arrest
- Avoid hypoxia and hypercarbia
- Hemodynamic monitoring: Arterial BP
- Maintain BP: give initial NS Bolus 20cc/kg
- Keep ICP <20 (1-15 mm Hg)
- Surgery for Trauma, Bleeds etc.
Treatment

- If infant requires >40-60cc/kg fluid then inotropic/vasoactive support is needed
- Dopamine, Epi, NE, Vasopressin *gtts etc.
- This is necessary to maintain adequate CPP (CPP=MAP-I CP)
- CPP: minimally accepted 60mm Hg (adults 70mm Hg)
- Follow Neuro status
- Manage Seizures (fosphenytoin, benzodiazepines, phenobarbital etc)
- *note: hyponatremia described in Peds. Literature
DX Tests to Monitor

- CBC with diff, coags, Fibrinogen, ABGs, LFTs, Lytes, Amylase, Lipase, BCX, UCX, CSF, CRP
- CXR, f/u CT, MRI
- CT of Abdomen
- Skeletal Survey
- EEG
- ICP, MAP, CPP
- Many Systems Involved: Interdisciplinary Approach
Specific Goals

- Maintain Cerebral Perfusion Pressure
- Minimize ICP: Monitor CSF drainage, replace cc:cc
- sedation, decrease noxious stimuli, and osmolar therapy
- Maintain osmolarity: 300-310 mosm/l
Changes in Carbon Dioxide have a profound effect on cerebrovascular responses

With hyperventilation, cerebral blood flow decreases which decreases ICP...but at the risk of cerebral infarction with prolongation

Until recently (last 5 years), hyperventilation was a mainstay in therapy

Currently: Maintain Low-Normal PCo2
Ventilation

- We used to provide temporary hyperventilation: (maintain PC02 low 20’s)
- Studies have shown that it worsens the outcome
- Can cause ischemia
- Goal: maintain PCO2 low normal range (35-45)
- Hypercapnia should also be avoided: may increase ICP
Treatment

- Manage Seizures
- Obtain EEG: Barbiturate-induced coma reduces cerebral metabolism and can be achieved by seeing a "burst" suppression on the EEG
Temperature

- Reducing body temperature reduces cerebral metabolic rate significantly.
- There is also evidence for additional benefits to ischemic brain tissue.
- Hypothermia: Runs the risk with neutropenia, coags, and infection. The patient lacks the capacity to elicit a proper immune response. Underlying infection may be missed due to the inability to mount a fever.
Numerous lines of evidence indicate that cerebral ischemic insults disrupt normal respiratory activity in mitochondria.

Glutamate mediated intracellular calcium accumulation and free radical generation are thought to be major mechanisms that contribute to cell death in hypoxic-ischemic brain injury.

Study Demonstrated: L-carnitine protects against glutamate- and KA-induced neurotoxicity.
Surgical Interventions

- Mass lesions require surgery
- ICP Bolt placement for EVD (extra-ventricular drain): collects CSF
- Evacuation of Bleed
- Subtemporal decompression is used for patients refractory to conventional therapy, (Chambers, 2003)
- Another expert does not recommend this for shaken infants, (Cho et al., 1995)
End of Part I
Trivia Question
What Syndrome Do All of These People Have?
Answer:
“The Mick Jagger Syndrome”
Please
“Curb Your Enthusiasm”
Part II
Case Study
Problem-Based Learning
It is 5PM one winter evening a few weeks before Christmas. You have not done your Peds rotation. You are getting ready to leave clinical and have plans for holiday “cheer” and Carols with friends. Suddenly you hear overhead: “Pediatric Trauma Level One: ETA 5 Minutes”. Your heart begins to pound. You look at your preceptor and via mental telepathy you both run to the trauma bay. No caroling for you this evening. The patient arrives via Life Lion in the trauma bay.
Case Study November, 2006

- 1 y/o male presented to Community Hospital with seizures, change in mental status, fever, and lethargy.

Was that a trauma page?
Patient Information

- 1 y/o male, 10 kg, ex 25 weeker, NICU X 3 months, Vent X2 months with diuretic therapy & 02. hx of BPD, RAD, GERD, mild developmental delay, HX of PDA: s/p Closure with Indomethacin at birth.

Patient Information

- Arrived Boarded and Collared, CT: shows: HUGE Sub-Dural Bleed and sub-arachnoid bleed, patient is on IVF @ 60cc/ hr of D5 ¼ NS + 20meqkcl/ Liter, s/ p 3 boluses of NS: 20c/ kg in the ER for “shock” on Room Air, Pupils: Right Pupil dilated, you see conjunctival hemorrhages OU

- left pupil 3mm sluggish, Anterior Fontanelle bulging, mottled, cap refill >6 seconds, poor peripheral pulses, circumoral cyanosis, decerebrate posturing
Labs

- PT: 16, INR: 2.0, PTT: 50
- Lfts: elevated, Albumin: 4.8
- CXR: hypoventilatory changes
- ABG: 7.27/ 50/ 70/ 18/ BD - 10, ICA++: 0.59
- BGM: 60
- (note: these labs were from 2 hours ago)
- NA: 124, K: 6.0, Cl: 120, Co2: 10, Bun 25, Cr: 1.5, Glu: 58
- Ca: 7, wbc 22, hgb: 7.8, Hct: 24, Plt: 125
- Neut: 85, L 15, Bands 25
- EKG: shows mild peaked T waves approaching sinus brady
- IV Access: 22 gauge PIV X1 in Right AC
Question
What are some Causes of Hyponatremia?

- 1. Dehydration
- 2. Meningitis
- 3. Subarachnoid Hemorrhage
- 4. SI ADH and Cerebral Salt Wasting
- 5. All of the Above
Progression of Case

- The patient is intubated using STP and Rocuronium

- The child is started on a fentanyl gtt @ 2 mcg/ kg/ hr, PRBC (10cc/ kg) are put on a pump over 2 hours, FFP 10cc/ kg, and treatment is initiated for hyperkalemia. The Ab CT shows a duodenal hematoma, the repeat CXR: confirms ETT placement w/ atelectatic areas, the child receives a 4 Fr. Rt. Femoral CVL and placement is confirmed via Xray (you can use the line), an Arterial Line is placed
Progression of Case

- There is a depressed skull fracture, the repeat head CT shows cerebral edema with a brain stem shift, OGT is placed and abdomen is decompressed: you get fresh blood. You repeat the trauma panel of labs + Blood Cultures. You also give Tylenol 15mg/ kg/ PR.

- CXR: Several Rib Fractures (new)
Peds Doses of Blood Products

1. Plt: 1 unit/ 10kg if <10kg: 1 Unit: 
   note:  1 unit ~50cc
2. PRBC: 5-20cc/ kg
3. FFP: 10-15cc/ kg
4. Cryo: adult: 10units (15cc/ unit)
5. Peds Cryo: <1yr: 10cc/ kg, >1 yr: 1 unit/ 5kg
6. 5 % albumin: 5-15cc/ kg
Questions

- What implications does a depressed skull fracture have on a febrile patient and what would you do and why? What med and dose?

- This child’s Temp is 40 °C: Is this the time for a lumbar puncture?
Answer

- Patient At Risk For Meningitis
- Treatment:
  - Decadron: 0.6mg/ kg divided by 4 = 1.5mg IV X1
  - Ceftriaxone: 100mg/ kg X1 (1gm)
  - Vancomycin: 15mg/ kg X1 (150mg)
- Give in That Order
- Note: no steroids if HSV is suspected or less than 6 weeks
- NO LP !!!!
Questions

Group Participation

1. What would you give immediately to treat the cerebral edema? What IVF would you run? And Why?

2. What is in LR that is bad for this patient? How much per Liter? (actually 2 things may be bad)

3. What does giving hypotonic fluid to this pt. contribute to?
Mannitol To Treat Cerebral Edema: 0.25-0.5gm/kg over 20 minutes (use filter)

K+ in LR: Bad for this Patient: Hyperkalemic Already, Lactate may be bad

Hypotonic Fluids Contribute to Dilutional Hyponatremia

Fluids Containing Dextrose Contribute to Cerebral Edema in the Trauma Patient
Progression of Case

- The child is transported to the OR. Neuro Surgery and General Surgery are Ready
- The neuro surgeon begins the emergency craniotomy to evacuate the bleed and place bolt with EVD.
- Vent Settings: 1.0 FiO2, TV: 90, Peep: +3, Rate: 18  The child begins to seize. Urine is dilute and large volume now...
Questions
Group Participation

1. What is the best treatment for pediatric seizures?
2. What could be the cause of the seizures? List some neuroprotective measures in the OR?
2. What is the different between phenytoin and fosphenytoin? What is the loading dose? When can you check a level?
Answers

- Seizures: Lorazepam 0.1mg/ kg (dilute), Midazolam: 0.1mg/ kg
- Causes: Fever, Meningitis, Hyponatremia, Bleed etc.
- Neuroprotective: temp, Isotonic Fluids, ?steroids, anticonvulsant load, EEG suppression (meds)
Pediatric Status Epilepticus

- Lorazepam (0.05-0.1 mg/ kg IV/ IO slowly infused over 2-5 min) has rapid onset and long duration of anticonvulsant action. It is preferred over diazepam.
- Midazolam (0.1-0.2 mg/ kg IM) is most effective when IV or IO access is not available. Midazolam is the only benzodiazepine that can be administered safely intramuscularly with equivalent rapid onset and moderate duration of action.
Phenytoin (18-20 mg/kg IV/IO) or fosphenytoin (15-20 mg/kg IV/IO) loading doses: These long-acting anticonvulsants usually are infused if benzodiazepines do not stop the seizures. A full loading dose should be delivered unless the patient is known to have a current therapeutic level.
Intravenous Phenytoin vs Fosphenytoin

- Fosphenytoin is a water-soluble prodrug of phenytoin.
- It is rapidly converted into phenytoin in vivo by phosphatase enzymes.
- The half-life of this conversion is 8-15 min and is independent of the plasma concentrations of either fosphenytoin or phenytoin.
Intravenous Phenytoin vs Fosphenytoin

- Phenytoin has very poor water solubility. It requires slow infusion in glucose free solutions to avoid precipitation.
- Fosphenytoin is supplied in phenytoin equivalents (PE) to obviate the need for learning new dosage schedules or calculating equivalent dosages.
- In general, has less CV effects (arrhythmias, hypotension)
<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Fosphenytoin</th>
<th>Phenytoin</th>
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<tbody>
<tr>
<td><strong>Routes of Administration</strong></td>
<td>IV or IM</td>
<td>IV</td>
</tr>
<tr>
<td><strong>Time to Max Serum Level</strong></td>
<td>20 Min</td>
<td>20 min</td>
</tr>
<tr>
<td><strong>IV Solution Compatibility</strong></td>
<td>Dextrose or NS</td>
<td>NS Only</td>
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<tr>
<td><strong>Max IV Infusion Rate</strong></td>
<td>150mg PE/ min</td>
<td>50mg/ min</td>
</tr>
<tr>
<td>Side Effect</td>
<td>Fosphenytoin</td>
<td>phenytoin</td>
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<tr>
<td>------------------------------------------------------</td>
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<tr>
<td>Local pain or burning</td>
<td>1%</td>
<td>37%</td>
</tr>
<tr>
<td>Hypotension</td>
<td>2%</td>
<td>13%</td>
</tr>
<tr>
<td>itching / burning (transient, not serious)</td>
<td>9%</td>
<td>0%</td>
</tr>
<tr>
<td>Purple glove syndrome (see below)</td>
<td>none reported</td>
<td>3 to 7%</td>
</tr>
<tr>
<td>1000-mg IV loading dose</td>
<td>$90.00</td>
<td>$6.72</td>
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</table>
The patient now has large U/O has DILUTE Urine (that is a hint)...but think out of the box..Vitals: HR: 55, BP: now low 90/55, Sats: 98%, Temp: 38.9 °C, CVP: 3

We gave Versed: 1mg IV X1, fosphenytoin 20mg PE/ kg/ X1...Still Seizing?  YES...
The labs come back:

- **ABG:** 7.35/ 28/ 450/ 15/ BD-10, ICA++: 0.4
- **Wbc:** 36K, hgb: 7, Hct: 22, plt: 60, N: 90, L 30, M 4, Bands 40
- **CRP:** 10  CVP: 2-3, Serum Osmolarity: 600mmol/ L
- **Pt:** 14, INR: 1.8, PTT: 35
- **Na:** 118, K: 3.8, Cl: 130, CO2 15, Bun 35, Cr: 2.8, Ca+: 5.6, Glucose: 30
Questions
Group Participation

What are the implications of these labs?

What is your plan?
Case Progression

- 10cc/ kg PRBC, 1 unit platelets, 3% hypertonic Saline: 2cc/ kg (slowly): seizures stop...

- IVF are continued @ NS 1 ½ MI VF (60cc/ hr) to change to ½ NS in 4 hours. We also gave Decadron: 0.15mg/ kg/ dose X1...why?

- Decreased FiO2 to 0.6%

- Atropine: 0.2mg IV X1
Case Progression

- D25% : 2cc/ kg then recheck BGM
- The child is given: Vanco: 15mg/ kg and Ceftriaxone 1 gm
- The Crani is done. The general surgeon is evacuating the bleed and repairing the duodenum. (Now...here’s the rub)
- The patient has insensible losses from the open gut, bleeding etc. Despite this, the patient is putting out large amount of clear urine and has electrolyte disturbances.
3% Hypertonic Saline

- 2cc/kg until Seizures Stop
- Infusion Rate: 1cc/min
- Do not exceed 12cc/kg
- 1meq/2cc
- Note: check institution policy
- *Can cause central pontine demyelination
Question
What possible factor is most likely not contributing to the higher urine output?

- A. DI
- B. Fluid Resuscitation
- C. Improved Renal and Splanchnic BF
- D. SIADH
New Lab Data

- **New Labs:**
  - Hgb: 11, Hct: 30, Coags: normal
  - Vitals: HR now: 150, BP: 88/46 and dropping, Sats: 99%
  - PH 7.28, PCo2: 30, Po2: 300, BD: -12, HCo3: 8, K: 4.0, Ca: 9
What Differential Diagnosis is possible to explain the dilute urine?

- A. High Output Renal Failure
- B. DI
- C. Cerebral Salt Wasting Syndrome
- D. Fluid Overload
- E. All of the Above
What is the Diagnosis R/ T the hypovolemia, acidosis, and dilute urine output?

- A. Cerebral Salt Wasting Syndrome
- B. DI
- C. SI ADH
- D. Too many hotdogs at Three Rivers Stadium
Diagnosis
Cerebral Salt Wasting Syndrome

- We determined through concise examination of our data that this child has Cerebral Salt Wasting. Fluid Restriction is NOT Correct. Can cause cerebral infarctions. With SIADH (Faser et al.) fluid restriction of $\frac{3}{4}$ MIVF improves hyponatremia. In patients with SAH and undiagnosed CSW fluid restriction is detrimental. Therefore, look at all the parameters.
Diagnosis
Cerebral Salt Wasting Syndrome

- With **SIADH** you have euvolemia, normal of increased CVP, normal HR, normal hct, increased EC volume, normal alb, decreased bun/cr, normal K+.
- With **CSW**: you have: decreased EC fluid volume, increased albumin, increased hct (we saw that after transfusions), increased Bun/ Cr, normal or increased K+, decreased CVP and BP, and increased HR.
We proceeded with the latter part of the surgery, did not restrict fluids and the NA was improving. By 10PM: The NA was 130 (up from 124 from 3PM in the ER: initial labs) The patient was on NS @ 1 ½ MIVF to change to ½ NS by 10PM in PICU. The BP and HR were approaching normal and we gave a 5% albumin bolus 10cc/ kg. The child was stabilized and transported to the PICU intubated with a bolt and EVD.
Case Progression

- No vasopressors were on board. (good for organ perfusion...the issue was volume) The child was extubated 3 days later (was started on the head injury protocol @ HMC). He then went to the rehab unit and has some minor deficits. (hard to judge because his baseline was delayed). Time will tell.
Cerebral Salt Wasting Syndrome

- CSWS is defined as “true” hyponatremia which occurs when there is a primary loss of sodium into the urine without an increase in total systemic volume. It is related to acute or chronic damage of the central nervous system.

- The most important component of this case is the timing of the injury. You don’t usually see CSW right away much like with NAT (non-accidental trauma)... The child usually doesn’t present to the ED right away, thus, delaying the diagnosis due to late clinical manifestations.
When further patients were studied (eg. SAH, head injury), plasma volume was found to be reduced, ADH levels were appropriate for serum osmolality and patients did not respond to fluid restriction as expected for SIADH.

If hyponatremia is due to CSW, fluid restriction may actually aggravate the clinical condition (esp in vasospasm of SAH) and lead to cerebral infarction.

- patients respond to salt and volume replacement (ie. opposite treatment!)
Causes of CSWS

- Humeral mechanisms: increase in circulating atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are thought to be contributing factors to the development of CSWS. Brain insult from the following disease processes are thought to also increase the risk of developing CSWS:
  - Sub-Arachnoid Hemorrhage
  - Intracerebral Hemorrhage/ Stroke
  - Cerebral Neoplasm/ Intracranial Surgery
  - Increased Intracranial Pressure
  - Tuberculous Meningitis
Signs and Symptoms of CSWS:

- Physical signs of CSWS: associated with severe hyponatremia or intravascular depletion.

- Hypovolemia (low CVP), shock SX
- Absence of Weight gain
- Orthostatic tachycardia/ hypotension
- Increased capillary refill time/ increased skin turgor
- Dry mucous membranes
- Sunken anterior fontanel (in infants)
- Large Volume of Dilute Urine
Hallmark: Low EC Volume with CSWS

- Differentiating the Diagnosis of CSWS and SIADH:
  - Identical acute cerebral insults may cause either SIADH or CSW. The clinical manifestation of both conditions can be virtually identical. The only true discriminative feature is that extracellular volume is “expanded” in SIADH and is “low” in CSWS.
<table>
<thead>
<tr>
<th><strong>CSWS</strong></th>
<th><strong>SI ADH</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>U/O high, dilute</td>
<td>U/O low, conc.</td>
</tr>
<tr>
<td>High Serum Osm.</td>
<td>Low Serum Osm.</td>
</tr>
<tr>
<td>Urine NA: high</td>
<td>Urine NA: high</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>Euvolemic</td>
</tr>
<tr>
<td>Serum Na+: low</td>
<td>Serum Na+: low</td>
</tr>
<tr>
<td>TX: NS (fluid)</td>
<td>TX: Fluid Restriction</td>
</tr>
<tr>
<td>Replete Sodium</td>
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</tbody>
</table>
Treatment of CSWS:

- Making the distinction between CSW and SIADH is of particular importance with regard to therapy. The following treatment regimen is recommended for treatment of patients who are suffering from CSWS:
  - Treat the underlying neurological process
  - Volume replacement (to maintain a positive salt balance)
  - IV Hydration with 0.9% NaCl infusion
  - IV Hydration with hypertonic 3% NaCl infusion
  - Colloids may be effective to absorb third-space fluid
  - Blood products may be useful for volume expansion
  - Urine replacement (cc for cc) and CSF: if EVD in place
  - Positive sodium balance
  - Fludocortisone (enhances sodium reabsorption, can cause hypokalemia)
<table>
<thead>
<tr>
<th>Clinical Manifestations to Monitor</th>
<th>CSWS</th>
<th>SIADH</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECF volume (the primary way to differentiate SIADH and CSWS)</td>
<td>Decreased</td>
<td>Increased</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>Increased</td>
<td>normal</td>
</tr>
<tr>
<td>Albumin concentration</td>
<td>Increased</td>
<td>normal</td>
</tr>
<tr>
<td>BUN/creatinine</td>
<td>increased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Potassium</td>
<td>normal or high</td>
<td>normal</td>
</tr>
<tr>
<td>Uric acid</td>
<td>normal or low</td>
<td>Decreased</td>
</tr>
<tr>
<td>Treatment</td>
<td>normal saline</td>
<td>fluid restriction</td>
</tr>
</tbody>
</table>
Pennsylvania State Program

• “Pennsylvania Shaken Baby Syndrome Prevention and Awareness Program”

• Mark S. Dias, M.D., FAAP: Neurosurgeon: HMC

• Dean J. Bonsall, M.D.: Pediatric Ophthalmologist

• Kelly Cappos, RN, BSN, CPUR, CLNC, HMC

• Carroll Rottmund, RN, BSN, CCRN, CLNC, HMC
In 2002, the 42 hospitals that provided maternity services in Central Pennsylvania were asked to partner together and participate in a shaken baby education, research, and child abuse prevention effort. The ultimate goal is to decrease the incidence of infant abusive head trauma statewide!
Dr. Dais and the shaken baby team were awarded a $2.8 million dollar grant from the Centers for Disease Control (CDC) in October 2007 to continue the hospital-based program.

The ultimate goal is to decrease the incidence of infant abusive head trauma statewide!
Moral of the Story
Don’t Shake or Drop your Baby
One Hundred Years from now
It will not matter
what kind of car I drove,
What kind of house I lived in,
how much money was in my bank account
nor what my clothes looked like.
But the world may be a better place because
I was important in the life of a child.

Forest E. Witcraft

Thank you
Marie