Neurosurgery Gray Matter
A General Overview

Produced with the Ad-Hoc Committee on Neurological Surgery, Undergraduate Medical Education Council of State Neurosurgical Societies
Overview

- Neurological Exam
- Head Trauma
- Cerebrovascular
- Spine injury
- Degenerative Spine Disease
- Peripheral Nerve
- Brain Tumors
- Hydrocephalus
Neurological Examination
Cranial Nerve Exam

II  hold up two fingers, cover one eye, vice verse
III  follow fingers
IV  follow fingers, downward and medially
V  sensory forehead, maxilla, chin
VI  follow fingers, laterally
VII  smile, wrinkle forehead
VIII rub fingers together next to ear
IX  say “ahhhhh” -- palate elevation
X  swallow
XI  shrug, turn head side-to-side against resistance
XII stick out tongue and wiggle side to side
### Sensory and Motor

<table>
<thead>
<tr>
<th><strong>SENSORY</strong></th>
<th><strong>MOTOR</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>C3 back of ears</td>
<td>C5 Lift arm at shoulder</td>
</tr>
<tr>
<td>C5 sternal notch</td>
<td>C6 Flex at elbow</td>
</tr>
<tr>
<td>T4 nipples</td>
<td>C7 Extend at elbow</td>
</tr>
<tr>
<td>T10 belly button</td>
<td>C8 hand grasp</td>
</tr>
<tr>
<td>L4 medial malleolus</td>
<td>L4 ankle dorsiflex</td>
</tr>
<tr>
<td>L5 dorsal foot</td>
<td>L5 great toe extension</td>
</tr>
<tr>
<td>S1 lateral foot</td>
<td>S1 ankle plantarflex</td>
</tr>
</tbody>
</table>
# Motor Strength

<table>
<thead>
<tr>
<th>Grade</th>
<th>Strength</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No contraction</td>
</tr>
<tr>
<td>1</td>
<td>Flicker or trace contraction</td>
</tr>
<tr>
<td>2</td>
<td>Movement with gravity eliminated</td>
</tr>
<tr>
<td>3</td>
<td>Movement against gravity</td>
</tr>
<tr>
<td>4</td>
<td>Movement against resistance</td>
</tr>
<tr>
<td>5</td>
<td>Normal strength</td>
</tr>
</tbody>
</table>
Reflexes

- Biceps: C5, 6
- Triceps: C6, 7
- Knee: L2, 3, 4
- Ankle: L5, S1, S2

Biceps m.
Triceps m.
Quadriceps m.
Gastrocnemius m.
**Brainstem Exam**

- **Pupils** - CN II, III
  - unilateral dilated, non-reactive - focal mass lesion/herniation w/ compression of CN III
  - bilateral fixed, dilated - diffusely increased ICP

- **EOMI** - CN III, IV, VI

- **Corneal** - CN V, VII
  - positive test means intact pons

- **Doll’s eyes** - CN VI, VIII

- **Cold Calorics** - CN VI, VIII

- **Gag** - CN IX, X
  - positive test means functioning upper medulla
Head Injury
Epidemiology

- Head injury is common
  - 150,000 patients die from traumatic injuries each year in the United States. Half of these deaths are the result of head injury.

- Head Injury Hurts
  - Over 200,000 people in the United States are disabled due to the effects of head and spinal cord injury.
Classification of Head Injuries

- Mild, Moderate or Severe, based upon post resuscitation GCS score.
- Penetrating or Blunt injury
- Mass lesion or diffuse axonal injury
Pathophysiology

- **Primary Injury**
  - The injury to the brain which occurs as a result of the initial trauma. This may be a blunt injury, penetrating injury, hypoxic or hypotensive injury.

- The only treatment is prevention.
Secondary Injury:

- Damage to the brain which is ongoing. This type of injury is potentiated by hypoxia, hypotension, intracranial hypertension, and inflammatory cascades initiated at the time of injury or soon thereafter.

Secondary injury is treatable.
### GLASGOW COMA SCALE (GCS score 3-15 based on best motor, verbal, eye response)

<table>
<thead>
<tr>
<th>MOTOR Activity</th>
<th>EYE Opening</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 obeys commands</td>
<td>4 spontaneous</td>
</tr>
<tr>
<td>5 localizes pain</td>
<td>3 to command</td>
</tr>
<tr>
<td>4 withdraws to pain</td>
<td>2 to pain</td>
</tr>
<tr>
<td>3 flexion of arms to pain (Decorticate)</td>
<td>1 none</td>
</tr>
<tr>
<td>2 extension of arms to pain (Decerebrate)</td>
<td></td>
</tr>
<tr>
<td>1 none</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>VERBAL Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 oriented</td>
</tr>
<tr>
<td>4 confused</td>
</tr>
<tr>
<td>3 inappropriate</td>
</tr>
<tr>
<td>2 incomprehensible</td>
</tr>
<tr>
<td>1 none</td>
</tr>
</tbody>
</table>

**note: intubated patients are given the designation “T” and have a verbal score of 1.**
Max score = 15
Minimum score = 3

Decorticate - implies destructive lesion of corticospinal tracts within or near cerebral hemispheres.

Decerebrate - implies lesion in diencephalon, midbrain, or pons. Can be mimicked by metabolic disorders such as hypoxia or hypoglycemia.
Preventing Secondary Injury

How do we avoid secondary injury?

• Avoid hypotension
• Avoid hypoxia
• Identify and treat intracranial mass lesions
• Identify and treat patients with intracranial hypertension.
The Role of Hypotension

How bad is hypotension in patients with severe head injury?

REALLY REALLY REALLY REALLY BAD!

- TCDB: 717 patients
- A SINGLE observation of SBP<90 mm Hg DOUBLED mortality, increased in hospital morbidity, and significantly influenced final outcome.
Hypoxia: Another Bad Thing

- Also bad for head injured patients
- Effects not as profound as hypotension
- Defined as apnea or cyanosis in the field or a PaO₂ of < 60 mm Hg by ABG
Intracranial Hypertension

- Two Causes:
  - Mass Lesion
  - Cerebral edema

- Must make diagnosis because treatment is radically different.

- Both may lead to same effects:
  - Herniation syndromes
  - Decreased CBF
Intracranial Hypertension

- Mass Lesions:
  - Evolve quickly
  - Lateralizing Signs
  - Pupillary Changes
  - Early herniation possible
Intracranial Hypertension

- Diffuse Axonal Injury
  - Depressed level of consciousness
  - No localizing signs
  - Herniation syndromes and peak ICP delayed
Herniation Syndromes

- **Subfalcine:**
  - “Midline Shift,” or movement of medial frontal/parietal lobes underneath falx cerebri.
  - No direct injury attributed to herniation itself, a sign of mass effect.
Herniation Syndromes

Transtentorial/Uncal
- Movement of medial temporal lobe over tentorial edge, compressing brainstem.
- Usually the result of a lateral or anterior temporal lesion.
- Uncus becomes impacted over edge and produces medically irreversible brainstem compression.
Intracranial Hypertension

**Effects on CBF:**
- The brain usually tightly controls CBF through regulation of the tone of smooth muscle in intracranial vessels. This process is called autoregulation.
- Autoregulation may be chemical or pressure dependent.
- Following TBI, autoregulation may be impaired or lost completely.
Intracranial Hypertension

- Normally, CBF is maintained in a normal range even with wide swings in SBP.
- If autoregulation is lost, perfusion becomes completely pressure dependent.
- As CPP = SBP - ICP, increased ICP will lower CPP.
The Vicious Cycle

- Increased ICP
- Decreased CBF
- Increased edema
- Ongoing ischemia
Breaking the Cycle

- Support Blood Pressure and Oxygenation
- Treat Intracranial Hypertension
  - Remove Mass Lesions
  - Medical Management
  - The role of hyperventilation
  - The role of steroids
  - New frontiers (Tirilizad, Hypothermia, THAM, SC58125)
Initial Evaluation and Management

ABC’s: Patients with head injuries are trauma victims. Adhere to ATLS guidelines.

- Hypotension = Hypovolemia until proven otherwise.

ABCD: After ABC’s, assess disability.

- GCS score, pupillary examination, motor asymmetry, cervical spine precautions
Initial Evaluation and Management: Blunt Head Injury

- **GCS 3-8**
  - Severe head injury, requires STAT neurosurgical consult and subsequent CT.

- **GCS 9-12**
  - Moderate head injury, requires urgent CT and neurosurgical consultation based on CT findings and early clinical course. Clinical observation is critical (utility of sedation).
GCS 12-15

- Mild head injury. Patient’s other injuries may take precedence. CT scan essential for patients who are not normal. In the fully awake patient, CT may not be necessary provided that the patient can be observed, there are no focal deficits, and there are no complicating factors.
Initial Evaluation and Management

- Pupillary examination
  - Anisocoria
  - Size and shape
  - Reactivity

“Pupillary asymmetry, dilation, or loss of light reflex in the unconscious patient usually reflects herniation caused by ipsilateral mass effect.”
Motor Examination

• The best motor response is used for GCS
• Motor asymmetry should be noted
• Tentorial herniation usually causes contralateral hemiparesis, but can cause ipsilateral hemiparesis (Kernohan’s Crus Syndrome). Pupillary changes are a better localizer.
Radiographic Examination

- Skull films:
  - Don’t bother

- C-Spine films
  - Don’t Forget

- CT Head
  - Get one!

Cerebral contusion
Radiographic Examination

CT Findings in Severe Head Injury:

Mass Lesions: blood clots in various locations

- Epidural Hematoma (lentiform shape)
- Subdural Hematoma (crescent shaped)
- Intraparenchymal Hematoma (within brain)
Treatments for increased ICP:
- Elevate HOB to 30°
- Mannitol
- Hyperventilate
- Sedation
- CSF drainage
- Pressors

**Note: for increased ICP, keep patient sedated, paralyzed if necessary, and CPP above 70mm Hg.**
For increased ICP:
The goal is Cerebral Perfusion Pressure > 70mm Hg.

- Normal ICP is around 12mm Hg. Use above meds to maintain ICP under 20mm Hg in trauma patient. May need to use pressors to increase MAP and maintain CPP > 70mm Hg if unable to adequately control ICP with above interventions.
- In pts. with severe injury, CPP > 60mm Hg may be adequate
- REMEMBER -- each patient is UNIQUE!
Skull Fractures

- Open vs. Closed -- depends on overlying skin
- Linear, Stellate or Comminuted fracture line(s)
- Depressed vs. Nondepressed
- Simple fractures:
  - Also known as nondepressed fractures
  - Requires no treatment as long as there is no associated intracranial bleeding (seen when fracture line crosses major vascular channel).
  - Considered open if fracture crosses nasal sinuses or mastoid air cells.
Epidural/Subdural Hematomas

CT is Key!

- Scan all head trauma patients -- scan quickly if there is a change in baseline mental status!
- Scan children and elderly with mental status changes.
- Scan if you have the slightest suspicion of intracranial bleeding.
- And, don’t forget, SCAN! It can save the patient’s life!

Treatment: expectant if small, craniotomy if there is mass effect or change in mental status.
Epidural Hematomas

- Between inner table of skull and dura mater
- Classic lenticular shape
- Usually arise from tear of middle meningeal art.
- Classic presentation
  - follows a blow to the head and unconsciousness
  - may be a *lucid interval* of several hours
  - as hematoma enlarges, medial temporal lobe is forced over the tentorium affecting the oculomotor nerve --result is a dilated ipsilateral pupil.
- Often fatal, but curable if lesion is found quickly.
Subdural Hematoma

- Usually result from tear of bridging veins between cortex and dura., disruption of cortical arteries and cortex laceration. Follows contour of brain.
- Associated with severe head injury, so neurologic deficit often remains after clot evacuated.
- May progress over several days:
  - increasing lethargy, confusion, hemiparesis
  - may see profound improvement with removal of clot.
Subdural Hematomas (cont)

- **Acute**
  - associated with severe trauma

- **Subacute**
  - progress over several days

- **Chronic**
  - gradually enlarge, seen most often in children and elderly
Subarachnoid Hemorrhages and Cerebral Aneurysms
Subarachnoid Hemorrhage (SAH)

**Etiologies**
- Trauma – most common
- Intracranial aneurysms
- Arteriovenous malformations
Spontaneous Aneurysmal SAH

- Estimated annual rate 10-28/100,000 persons
- About 30,000 ruptures per year
  - 1/3 die before reaching hospital
  - 1/3 with serious morbidity
  - 1/3 do well
# Subarachnoid Hemorrhage

## Hunt-Hess Classification of aneurysmal SAH:

<table>
<thead>
<tr>
<th>Class</th>
<th>Symptom</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>none</td>
<td>5%</td>
</tr>
<tr>
<td>II</td>
<td>H/A, nuchal rigidity</td>
<td>10%</td>
</tr>
<tr>
<td>III</td>
<td>confusion/lethargy</td>
<td>15%</td>
</tr>
<tr>
<td>IV</td>
<td>stuporous</td>
<td>60%</td>
</tr>
<tr>
<td>V</td>
<td>comatose</td>
<td>almost 100%</td>
</tr>
</tbody>
</table>
Clinical Presentation of SAH

- Thunderclap headaches – “worse headache”
- Meningismus
- Ocular hemorrhage
  - Preretinal hemorrhage seen with fundoscopie exam
Principles of Acute Diagnosis

- Admission procedures
  - ABC’s
    - protect airway, stabilize vital signs
  - Review Past Medical History
    - identify risk factors
  - Physical Examination
    - Blood pressure, pulse rate and rhythm, cardiac examination, check for cervical, supraclavicular, ocular or cranial bruits, fundoscopic examination
Evaluation of SAH

- Non-contrast head CT
- If CT negative, then lumbar puncture
  - Finding – RBC count usually > 100,000
  - Check first and last tubes
- If any of above positive proceed with cerebral angiogram and neurosurgical consultation
Carotid Artery Disease
Carotid Artery Disease

Carotid Endarterectomy

- Multiple large randomized controlled studies have evaluated CEA.
- Significant benefit (vs. best medical therapy) for symptomatic lesions with stenosis >50%.
- Moderate benefit for asymptomatic lesions with stenosis >60%.
- Benefit dependent on low surgical morbidity/mortality
Carotid Artery Disease

**Epidemiology**
- Atherosclerotic plaques begin to form at 20 yrs. of age.
- Risk of stroke correlates with degree of stenosis

**Presentation**
- Stroke, TIA, amaurosis fugax
Carotid Endarterectomy and Removed Plague
Spinal Cord Injury
Spinal Cord Injury

**Epidemiology**

- 10,000 per year in US
  - Traumatic para- and tetraplegia range from 10-50 new patients per million individuals
  - Mostly young healthy individuals
Deficits caused by SCI are caused by damage incurred at the time of injury, as well as progressive damage that occurs hours to days following injury. The initial injury is termed “primary” injury, ongoing processes are termed “secondary” injury.
Secondary Spinal Cord Injury

- Trauma to the spinal cord triggers a series of biochemical events which play out over a period of hours to weeks following injury.
- These events cause local and systemic effects, some of which are amplified by positive feedback cycles.
Secondary Injury After SCI Leading to Cell Death

- Oedema, tissue ischaemia and anoxia
- BBB breakdown and cell membrane injury
- Release of excitotoxins from damaged and hyperactive cell

Cell Death
Preventing Further Spinal Cord Injury

**ABC’s:**
- Oxygenate, avoid hypotension

**Immobilization:**
- Prevent further mechanical damage to spinal cord

**Methylprednisolone:**
- May ameliorate some effects of secondary injury cascades
Treatment of Spinal Cord Injury

- **Decompression**
  - Alleviate effects of compression on local blood flow

- **Stabilization**
  - Prevent further damage due to pathological movement of spine

- **Mobilization**
  - Preserve and improve residual function through aggressive rehabilitation
Degenerative Spine Disease
Degenerative Disc Disease
Clinical Presentation

- Myelopathy VS Radiculopathy

**Myelopathy**
- Spinal cord compression
- Upper motor neuron findings
  - Brisk reflexes
  - Spastic gait

**Radiculopathy**
- Nerve root compression
- Lower motor neuron findings
  - Diminished reflexes
  - Dermatomal findings
  - Isolated muscle weakness
Low Back Pain (LBP) and Radiculopathy

LBP is extremely common

- Lifetime prevalence 60-90%
- Annual incidence 5%
- 85% no specific diagnosis
- 90% with LBP improve within 1 month with or without treatment
- Only 1% have radicular symptoms
Different nerve roots have different motor and sensory functions, and these functions are relatively stereotypical. The history and physical examination of a patient are usually adequate to diagnose a nerve root compression syndrome.
Spinal Anatomy:

Lumbar Segment, top view

- Spinal Cord
- Nerve Root
- Annulus
- Nucleus Pulposus
- Intervertebral Disc
- Vertebral Body

L5 Medial

S1 Lateral
Lumbar Radiculopathy from Herniated Disc

- Presentation
  - Positive straight leg raise
  - Radicular symptoms
    - L45 HNP
      - L5 dermatome
      - Weakness EHL
    - L5S1 HNP
      - S1 dermatome
      - Gastroc. Weakness
      - Absent ankle jerk
Lumbar Radicular Syndromes:

**L3 Radiculopathy (Rare):**
- Pain anterior or anterolateral thigh
- Quadriceps weakness
- Diminished patellar reflex

**L4 Radiculopathy (3-10%):**
- Pain across knee, to medial malleolus
- Quadriceps weakness, maybe foot dorsiflexor weakness
- Diminished patellar reflex

**L5 Radiculopathy (40-50%):**
- Pain lateral thigh/leg into top of foot
- Stereotypical sensory loss in first webspace
- EHL weakness and tibialis anterior (foot drop)
- Medial hamstring reflex diminished

**S1 Radiculopathy (40-50%):**
- Pain and sensory loss extend down posterior leg into lateral aspect of foot, lateral malleolus
- Weak plantar flexors of foot
- Achilles reflex diminished
Cervical Herniated Disc

Presentation
- Cervical radiculopathy
- Neck pain
- Myelopathy
Cervical Radicular Syndromes:

**C6 Radiculopathy (19%)**:
- Sharp stabbing pain in lateral forearm, thumb and index finger (six shooter).
- Biceps weakness
- Loss of biceps reflex
- Dermatomal sensory loss

**C7 Radiculopathy (69%)**:
- Pain into 2nd and 3rd fingers
- Forearm extensors or triceps weak
- Loss of triceps reflex
- All fingertips may be involved in paresthesias

**C8 Radiculopathy (10%)**:
- Hand intrinsic weakness
- Medial hand pain (4th and 5th finger) and sensory changes
Cauda or Conus Syndromes

- Caused by large mass in canal which compresses the conus or cauda equina
- Slightly different presentations
- Slightly different prognosis
- Many similarities: Bilateral leg symptoms, bowel and bladder dysfunction, “saddle” sensory loss.
- Both are neurosurgical emergencies.
Lumbar Spinal Stenosis

- Presents with “Neurogenic Claudication”
- Often occurs in patients with congenital stenosis
- Usually a disease of the elderly
- Responds well to surgery
- May be difficult to distinguish from vascular claudication
### Claudication

<table>
<thead>
<tr>
<th>Neurogenic claudication</th>
<th>VS</th>
<th>Vascular claudication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory loss dermatomal</td>
<td></td>
<td>Sensory loss stocking/glove</td>
</tr>
<tr>
<td>More variable pain onset</td>
<td></td>
<td>Usually very reproducible pain onset</td>
</tr>
<tr>
<td>Standing to rest not sufficient</td>
<td></td>
<td>Standing to rest is OK</td>
</tr>
<tr>
<td>No pallor on foot elevation, pulses OK</td>
<td></td>
<td>Pallor with foot elevation, diminished pulses</td>
</tr>
<tr>
<td>“Anthropoid” posture</td>
<td></td>
<td>Normal Posture</td>
</tr>
<tr>
<td>Improves/stable with stationary bike test (Stretch ligament in flexion opens canal)</td>
<td></td>
<td>Made worse by stationary bike test</td>
</tr>
</tbody>
</table>
Cervical Stenosis

Presents with neck pain and myelopathy
• Gait disturbances
• Hand dysfunction
• Bowel and bladder problems
• Hyper-reflexes
Plain films are a cheap and easy screening tool.

Plain films lack specificity and sensitivity.

Plain films deliver 1000X the dose of radiation to the genitals as does a CXR.

- Need to justify use: AHCRP
Who to Image:

- In the absence of “Red Flags,” no one requires plain films during the first 6 weeks of pain.
- If imaging is required due to presence of “Red Flag,” or prolonged symptoms, consider MRI.
## Imaging “Red Flags”

<table>
<thead>
<tr>
<th>Category</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Trauma</strong></td>
<td>• Major Trauma&lt;br&gt;• Minor Trauma in older or osteoporotic patients&lt;br&gt;• Steroid use&lt;br&gt;• Osteoporosis&lt;br&gt;• Age &gt;70</td>
</tr>
<tr>
<td><strong>Tumor</strong></td>
<td>• Age &gt;50 or &lt;20&lt;br&gt;• History of CA&lt;br&gt;• Unexplained weight loss</td>
</tr>
<tr>
<td><strong>Infection</strong></td>
<td>• Fever, chills weight loss&lt;br&gt;• Risk factors for spinal infection (IVDA, UTI, immunosuppression)&lt;br&gt;• Pain worse when supine or at night</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td>• Progressive neurological deficit&lt;br&gt;• Cauda equina syndrome</td>
</tr>
</tbody>
</table>
Indications for MRI or Surgical Referral

- Neurological Deficit
- Pain lasts beyond 6-12 weeks
- “Red Flag”
- Recurrent pain
- Findings on imaging studies
Peripheral Nerve Disease
Peripheral Nerve Entrapment Neuropathies

- Compression of peripheral nerve from external forces or nearby anatomical structures
- Pain is the most common symptom
  - Frequently worse at night
  - Often retrograde radiation from point of entrapment
  - Tenderness at point of entrapment
Entrapment Neuropathies

Carpal tunnel
- Compression of median nerve at wrist
- Tinel’s sign
- Phalen’s test
- Symptoms palmar thumb, index, middle finger
- Hand weakness

Tardy ulnar palsy
- Compression at elbow
- Pain, numbness little finger (ulnar distribution)
- Froment’s sign
- Claw deformity
- Atrophy interossei muscles
Brain Tumors
Brain Tumors

- Two types – primary and metastatic
- Presentation
  - Head aches
  - Nausea and vomiting
  - Seizure
  - Focal neurological deficit
Primary Brain Tumors

Epidemiology

- 5 per 100,000 individuals per year
- 10,000 new cases per year in US
- Rarely metastasis outside brain
- Very poor prognosis for malignant tumors
Metastatic Brain Tumors

Epidemiology

- 60,000 – 70,000 new cases per year
- Increase incidence in smokers
Brain Tumors in Children

- 22 new cases per million each year
- Second leading cause of cancer death in children
- More often in posterior fossa
- Present with sign of increased intracranial pressure
  - Hydrocephalus
  - Papilledema
  - Head ache
  - Nausea vomiting
Hydrocephalus
DEFINITION:
Diverse group of conditions which result from impaired circulation and resorption of CSF.
CSF Formation

- CSF IS PRIMARILY FORMED BY THE CHOROID PLEXUS.
- NORMAL CSF PRODUCTION: 20 ml/h., about 500 ml/day, total volume 150 ml. Therefore must circulate 3 times per day.
Types of Hydrocephalus

- OBSTRUCTIVE OR NON-COMMUNICATING (OBSTRUCTION WITHIN THE VENTRICULAR SYSTEM)

- NON OBSTRUCTIVE OR COMMUNICATING (MALFUNCTION OF ARACHNOID VILLI)
SYMPTOMS:

- IRRITABILITY
- POOR APPETITE, DECREASED FEEDING
- LETHARGY
- VOMITING
- IN OLDER PATIENTS:
  - HEADACHE
  - CHANGES IN PERSONALITY
  - ACADEMIC DETERIORATION
Clinical Manifestations

**SIGNS:**
- Anterior fontanelle wide open and bulging, increased head circ.
- Dilated scalp veins
- Setting sun sign
- Brisk tendon reflexes, spasticity
- Possibly clonus
Shunts

- **Shunts**: the main form of therapy for hydrocephalus
- **Types**: peritoneal; atrial; pleural; ureteral; gallbladder; subarachnoid
- **Disadvantages**: mechanical and infectious complications
What’s a Shunt?

- Ventricular catheter
- Reservoir
- One-way valve
- Peritoneal, pleural, or atrial catheter
Diagnosis of Shunt Failure Guidelines

- **Symptoms:** Headache, nausea, vomiting, lethargy, increased head circumference, fontanelle, wound drainage, MOTHER KNOWS BEST!

- **What about fever?**

- **Studies:** Head CT, shunt series, shunt tap, abdominal ultrasound, nuclear medicine scan, ICP monitoring
Eval. of Shunt Malfunction

**H & P**
- reason for initial insertion
- date of last revision and reason for revision
- fontanelle tension if < 1 year old

**SYMPTOMs of Acute ↑ ICP**
- headache, N/V, diplopia, lethargy, ataxia, seizures

**SIGNs of Acute ↑ ICP**
- papilledema, upward gaze palsy, visual field deficits
- Infants: bulging fontanelle, prominent scalp veins
Shunt Malfunction

- Radiographic studies:
  - Shunt series
    - A/P and Lateral skull
    - low CXR or Abdominal CXR
  - Head CT without contrast
  - COMPARE to old CT for evidence of increased ventricular size.
- Treatment: shunt revision may be necessary up to several times per year.
Questions?

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