Neuroimaging and Studies

Computed Tomography (CT)

Contraindications
Radiation exposure: particularly in young pts
Contrast dye: dangerous for allergies & renal disease
Contrast is only required for exotic cases: CT-A, CT-perfusion (in stroke), CT-V (thrombosis)

Density
Bone/Ca (1000 HU) > Contrast > Blood> Parenchyma > Water (0 HU) > Fat > Air (-1000 HU)

(REF: Copen Summerstock 2010)

Windows
Brain: W75, L20
Perf: W30, L36
SDH: W30, L50
Skull: W3500, L340
Soft tissue: W350, L0
CTA: W214, L88
W = width of window (contrast)
L = center of window (brightness)
(REF: Copen Summerstock 2010)

Contrast
Allergy
Anaphylaxis: no contrast ever again
Hives: pre-treatment w/steroids & diphenhydramine (takes 13 hours, call a radiologist for the regimen)

Nephrotoxicity
For pts over 60 years old, or w/a history suggesting renal impairment, serum Cr within one month is needed.
eGFR >45: no problem
eGFR 30-45: radiologist will be contacted, patient will likely need post-scan hydration
eGFR <30: no contrast

Renal protection
I.V. infusion: 154 mEq/L sodium bicarbonate in D5W solution: 3 mL/kg/h for 1 hour immediately before contrast injection, then 1mL/kg/h during contrast exposure and for 6 hours after procedure
(REF: http://www.uptodate.com/online/content/topic.do?topicKey=drug_l_z/242912&selectedTitle=1%7E150&source=search_result)

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Helical/Spiral CT

"Source (usually of x-rays) describes a helical trajectory relative to the object while a two dimensional array of detectors measures the transmitted radiation on part of a cone of rays emanating from the source. … Source and array of detectors are mounted on a rotating gantry while the patient is moved axially at a uniform rate." (REF: http://en.wikipedia.org/wiki/Spiral_computed_tomography)

16-64 multislice scanner/detectors

Non-Contrast Head

Fast (30 sec scanning time)

Hyperdensity ~ blood (evaluate distribution, compartment), 99% sensitivity to r/o sub-arachnoid hemorrhage

Hypodensity ~ edema, air

Midline shift ~ mass

Large lateral ventricles ~ CSF obstruction, degeneration

Small 3rd or 4th ventricles ~ CSF obstruction, increased ICP

Blood vessels: arteries (acute stroke, aneurysm) & veins

Hemorrhagic Transformation

HI-1: small petechiae without mass effect

HI-2: confluent petechiae without mass effect

PH-1: hematoma <30% of infarct w/mass effect

PH-2: hematoma >30% of infarct w/mass effect or hemorrhage beyond infarct

Sample Read

No obvious hyperdensities/hemorrhage, hypodensities/edema, mass, midline shift, or hydrocephalus. Ventricles symmetric. Gray-white differentiation preserved. No sulcal effacement. No vessel hyperdensities.

Posterior Fossa

Limited by beam-hardening artifacts and nonlinear volume averaging


CTA Head/Neck

Fast imaging of blood vessels (delay of ~15sec from injection, takes ~5 min)

Used for aneurysms, dissection, severe stenosis, cutoff/thrombosis, perfusion/distal vessels, prior to angio

CTV

Useful to r/o sinus vein thrombosis (young pt w/unexplained headache & papilledema)

This is essentially a CTA w/delays

CT Perfusion
Somewhat useful in ischemic stroke, but not clear how often it changes management (even as a tPA guide)

**CT-Myelogram**

**XRay**

Pneumoencephalogram – old procedure based on rotating x-ray; used to evaluate midline shift

Shunt series

**Magnetic Resonance Imaging (MRI)**

**Contraindications**

Look up devices on mrisafety.com

Pacemakers: classically no, however it may be safe w/controlled monitoring (REF: Nazarian S et al. A prospective evaluation of a protocol for magnetic resonance imaging of patients w/implanted cardiac devices. Ann Intern Med 2011 Oct 4; 155:415. [http://www.annals.org/content/155/7/415.full](http://www.annals.org/content/155/7/415.full))

Stents typically ok (info on manufacturer website), especially after 6 weeks

Clips / aneurysms OK

Ortho implants typically OK

**Severe claustrophobia**

Pretreat w/benzos (lorazepam) and/or haloperidol

**Incidental findings**

Brain infarcts (7.2%), cerebral aneurysms (1.8%), benign primary tumors (1.6%), mainly meningiomas. Prevalence of asymptomatic brain infarcts & meningiomas increased w/age, as did the volume of white-matter lesions, whereas aneurysms showed no age-related increase in prevalence. (Vernooij NEJM 2007)

**T1**

Good fine structure, high resolution.

In stroke, for hemorrhage, thrombosis

**T2**

Spin-echo sequence sensitive to water

**FLAIR (Flow Sensitive Alternating Inversion Recovery)**

T2 w/fluid subtracted (CSF)

for edema, evolving infarct

**DWI (Diffusion Weighted Imaging)**

**The key study for acute stroke**

Very good at differentiating acute vs. chronic, & embolic vs. small vessel infarcts. Ischemia is visible on DWI w/in 30 min, early DWI lesions invariably involve (irreversible) infarction.

**What it is looking for**

Cytotoxic edema that occurs when ischemia keeps neurons from making energy. w/no energy, cells can’t maintain ion hemostasis. Ions shift, taking water w/them from extracellular to intracellular space, where diffusion is more limited.

**How it works**

Two strong “diffusion sensitizing gradients” are placed symmetrically around the “180º radiofrequency pulse of a spin-echo sequence”. These gradients cause dephasing & rephasing of water protons. Protons that are dephased during the first gradient (because of normal diffusion) randomly change phase as they diffuse from one region of the magnetic field to another so rephasing is incomplete. Signal is attenuated by this incomplete rephasing. Areas of restricted diffusion retain the signal & appear hyperdense.
Differential


**ADC (Apparent Diffusion Constant)**
Quantifies the degree of water proton mobility on the edges of an infarct

What it is looking for: Incomplete energy failure – the penumbra. Studies show that on the periphery of ischemic areas DWI is restricted, but ATP production is still near normal. In these areas, cells are using anaerobic glycolysis to maintain their membranes. If energy (oxygen) is restored to these areas, cell death may be avoided.

**PWI (Perfusion Weighted Imaging)**
Looks at relative cerebral perfusion

T2 weighted images are made every 1-2 sec to monitor the signal decrease associated with the passage of a contrast bolus. with this, the time to peak, mean transit time, & relative cerebral blood flow can be calculated to identify perfusion deficits. Can be combined with DWI to calculate the PWI-DWI ratio. If PWI>DWI, expect the lesion to grow.

**Gradient Recalled Echo (GRE)**
T2 weighted MRI sequence
for hemorrhage

Detect microbleeds (dark) via focal deposits of hemosiderin (REF: Neurology 2008;70:1208-1214)


**Short Tau Inversion Recovery (STIR)**
Spine & orbit studies

Fat suppression

**DTI (Diffusion Tensor Imaging)**
For axonal tract tracing

**fMRI (Functional MRI)**
Limited clinical utility, used in research for epilepsy surgery

**Gadolinium**
Visualize vessels

Visualize breakdown in blood-brain barrier


gyral enhancement common post stroke

(REF: Patterns of Contrast Enhancement in the Brain and Meninges. http://radiographics.rsna.org/content/27/2/525.full?sid=e0259461-1de3-4d35-ae42-e601067728a3)
Dura-arachnoid pachymeningeal enhancement. (a) Diagram illustrates dura-arachnoid enhancement, which occurs adjacent to the inner table of the skull; in the falx within the interhemispheric fissure; and also in the tentorium between the cerebellum, vermis, and occipital lobes. Pure dural enhancement, without pial or subarachnoid involvement, will not fill in the sulci or basilar cisterns.

Dural tail enhancement with meningioma. (3a) Diagram illustrates the thin, relatively curvilinear enhancement that extends from the edge of a meningioma. Most of this enhancement is caused by vasocongestion and edema, rather than neoplastic infiltration. The bulk of the neoplastic tissue is in the hemispheric extraxial mass; nonetheless, the dural tail must be carefully evaluated at surgery to avoid leaving neoplastic tissue behind.

Pia-arachnoid leptomeningeal enhancement. (a) Diagram illustrates the enhancement pattern, which follows the pial surface of the brain and fills the subarachnoid spaces of the sulci and cisterns.

Cortical gyral enhancement. (a) Diagram illustrates gyral enhancement that is localized to the superficial gray matter of the cerebral cortex. There is no enhancement of the arachnoid, and none in the subarachnoid space or sulci.

Subcortical nodular enhancement. Diagram illustrates nodular lesions near the gray matter–white matter junction and one near the deep gray matter. This pattern is typical for metastatic cancer and clot emboli. Because of their typical subcortical location, metastases often manifest with cortical symptoms or seizures while the lesions are small (often <1 cm in diameter).

Smooth ring-enhancing pattern in late cerebritis and subsequent cerebral abscess. Diagram illustrates a thin (<10 mm) rim of enhancement, which is usually very smooth along the inner margin; this pattern is characteristic of an abscess. The lesion is surrounded by a crown of vasogenic edema spreading into the white matter.

Necrotic ring pattern of high-grade neoplasms. (a) Diagram illustrates a lesion with an enhanced rim that is very thick medially; the ring is thicker and more irregular than that seen in a typical abscess. The lesion is surrounded by a crown of vasogenic edema spreading into the white matter.

Fluid-secreting neoplasm (cyst with mural nodule pattern). Diagram illustrates a “cystic” mass with a “mural nodule,” which is the classic description for a pilocytic astrocytoma. This pattern is seen in a variety of fluid-secreting neoplasms, including hemangioblastoma, ganglioglioma, and pleomorphic xanthoastrocytoma.

Open ring pattern. Diagram illustrates a lesion with an incomplete rim (only part of the rim enhances). This appearance may be seen in multiple sclerosis (without mass effect as in this drawing), tumefactive demyelination (with mass effect), and fluid-secreting neoplasms (with associated mass effect and occasionally with surrounding vasogenic edema).

Periventricular pattern. Diagram illustrates thick periventricular enhancement, as shown around the right lateral ventricle. This enhancement pattern is usually neoplastic and is most commonly seen in a high-grade astrocytoma or primary CNS lymphoma. Thin periventricular enhancement, as shown around the left lateral ventricle, is usually infectious.

**MRA**

Arterial phase post-contrast

r/o stenosis or dissection

Better soft-tissue than CT-A

Intracranial MRA is via TOF (no contrast)

Neck MRA is done w/contrast (can be TOF too)

**MRV**

Venous phase post-contrast

r/o sinus/vein thrombosis

**Fat saturation/suppression**

T2 for optic neuritis
T1 for dissection

**Blood on MRI**

<table>
<thead>
<tr>
<th>Phase</th>
<th>Time</th>
<th>Hgb</th>
<th>T1</th>
<th>T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperacute</td>
<td>&lt;24 h</td>
<td>Oxy-hgb (intra-cell)</td>
<td>Iso</td>
<td>Hyper</td>
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<tr>
<td>Acute</td>
<td>1-3 d</td>
<td>Deoxy-hgb (intra-cell)</td>
<td>Iso to hypo</td>
<td>Hypo</td>
</tr>
<tr>
<td>Early subacute</td>
<td>&gt;3 d</td>
<td>Met-hgb (intra-cell)</td>
<td>Hyper</td>
<td>Hypo</td>
</tr>
<tr>
<td>Late subacute</td>
<td>&gt;7 d</td>
<td>Met-hgb (extra-cell)</td>
<td>Hyper</td>
<td>Hyper</td>
</tr>
<tr>
<td>Chronic</td>
<td>&gt;14 d</td>
<td>Hemosiderin (extra-cell)</td>
<td>Hypo</td>
<td>Hypo</td>
</tr>
</tbody>
</table>

Acute -> Sub (e->l) -> chronic (T1, T2): liDee, BiDee, BaBy, DaDa

(REF: Can Assoc Radiol J. 2009 Apr;60(2):88-90. Easy ways to remember the progression of MRI signal intensity changes of intracranial hemorrhage. PMID: 19433039. )

**Myelination**

Abnormal for cerebral white matter to have a high T2 > 1.5y

Normal T2-weighted (A–D) and T1-weighted (E–H) images at term (A, E), and at the ages of 5 months (B, F), 1 year (C, G), and 5 years (D, H).

Myelin deposition is represented by a low signal on T2-weighted images and high signal on T1-weighted images.

Note that myelination consistently looks more advanced on T1-weighted images than on T2-weighted images.

In all white matter pathologies other than deficient myelination, the signal changes are as a rule more prominent: the affected white matter has a much higher signal on T2W images and a much lower signal on T1W images than grey matter structures

A: Alexander disease (A) presents in many patients with predominantly frontal white matter abnormalities. Note the additional slight signal abnormalities in the basal ganglia.

B: The most frequent presentation of the cerebral form of X-linked adrenoleukodystrophy (B) is with a lesion in the parieto-occipital white matter. Note that two zones can be distinguished within the lesion.

C: Kearns-Sayre syndrome (C) is one of the disorders characterized by predominantly subcortical white matter abnormalities and relative sparing of the periventricular white matter. The disease also displays signal abnormalities in the thalamus (C).

D: Metachromatic leukodystrophy (D) primarily affects the periventricular and deep cerebral white matter, whereas the U-fibers are relatively spared. The stripes with more normal signal within the abnormal white matter are typically seen in certain lysosomal storage disorders (D).

E: Cortical neuronal degenerative disorders often have an ill-defined, broad, periventricular rim of mildly abnormal signal, as shown here in juvenile neuronal ceroid lipofuscinosis (E).

F: Diffuse cerebral white matter abnormalities are seen in childhood ataxia with central hypomyelination /vanishing white matter (F).

G: In cerebrotendinous xanthomatosis (G), the cerebellar white matter is usually more affected than the cerebral white matter. The cerebellum often also contains areas of low signal (G).

H: In patients with autosomal dominant adult onset leukoencephalopathy related to a duplication of LMNB1 (H), involvement of the middle cerebellar peduncles is frequently seen.

(REF: [http://www.neurology.org/content/72/8/750.full](http://www.neurology.org/content/72/8/750.full))

**Interpretation**

If DWI & PWI are normal while symptoms persist – acute ischemia is not the cause, consider unmasking old deficit.

If DWI & PWI are normal w/symptoms resolved – TIA or non-ischemic lesion.
If DWI & PWI are positive – ischemic stroke

DWI positive, PWI normal - consider status epilepticus, TGA, CJD, cerebral venous thrombosis, eclampsia

PWI positive, DWI normal – reversible ischemic lesions like TIA, vasospasm secondary to SAH, migraine

How this is likely to affect treatment in the future:
Better identification of pts who will benefit from thrombolysis (and who will have lowest risk).

PWI>DWI (large proportion of salvageable area "at risk") vs. DWI>PWI (completed infarct)
Large vessel infarcts vs. lacunar infarcts.
Smaller DWI regions will likely bleed less.

Stroke

(REF: http://www.neurographics.org/2/2/1/13.shtml)

(Lumbar Puncture (LP)

Anatomy


Spend most time positioning patient

Placement
Iliaic crests highest point identifies L4 body
Use L3/4 or L4/5 interspace, L2/L3 as last resort

Lateral recumbent
Allows measurement of opening pressure

C shape, focus on curving lumbar back out
Back perpendicular to table
Pillows between head & knees

(Sitting upright
Simpler to determine horizontal placement by sight & pt sensation

Teaching
Differential Diagnosis

Emergent

Infection

Fever

MS

Headache

Meningeal signs

Subarachnoid hemorrhage

Xanthochromia

Hgb degradation >2hr in vivo

Pink to yellow tint post of serum post centrifugation

Malignancies

Demyelinating

Guillain-Barre Syndrome

Nonurgent

Pseudotumor cerebri

Carcinomatous meningitis

Tuberculous meningitis

Normal pressure hydrocephalus

CNS syphilis

CNS vasculitis

MS

GBS

Med indications

Spinal Anesthesia

Intrathecal chemo

Intrathecal antibio

Contrast media

Relative Contraindications

- ICP

Overlying infection

Bleeding diathesis

Check coags & plt

Neuraxial techniques in obstetric and non-obstetric patients w/common bleeding diatheses. PMID: 
19608843. )

Anticoag:
**Herniation**

**CT before LP**

Concern for precipitating herniation in pts w/structural injury -> mass effect


**How to predict abnormal CT/mass effect when considering LP?**

Factors associated w/abnormal CT: 60yo, immunocompromised, h/o CNS disease, sz <1wk, focal neuro deficits

If 0 factors: 97% normal CT (1% mild mass effect)

If 1 factor: 62% normal CT/38% abnormal

**Significance of CT uncertain**

No pts who got an LP (n=289) herniated (including 5 w/mild mass effect, 2 w/mod)

Of pts who got a CT (n=235), 4 pts did not get an LP because of CT (3 w/severe mass effect, 1 mild). 2 pts w/severe mass effect herniated <1 wk (despite no LP)

Even pts w/documentated abscess have a relatively low risk of herniation (2%) (REF: N Engl J Med. 2002 Apr 18;346(16):1248-51. PMID: 11963945)


**Equipment**

**Needle**

"Atraumatic" 22G, e.g. Whitacre needle (also Sprotte, Gerlie-Marx)

- Whitacre/Atraumatic
- Quincke/Traumatic

**Standard kit**

**Extra gear**

Mask
Eye protection
Sterile Gown
Sterile Gloves
Extra Lidocaine 1%

**Ultrasound?**

(REF: Shaikh. Ultrasound imaging for lumbar punctures and epidural catheterisations: systematic review and meta-analysis. BMJ. 2013 Mar 26;346:f1720. doi: 10.1136/bmj.f1720. PMID: 23532866. [http://www.bmj.com/content/346/bmj.f1720](http://www.bmj.com/content/346/bmj.f1720))

<table>
<thead>
<tr>
<th>Wt</th>
<th>Distance from skin to ligamentum flavum</th>
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<tr>
<td>BMI</td>
<td>mm</td>
</tr>
<tr>
<td>Normal</td>
<td>&lt;25</td>
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<tr>
<td>OverWt</td>
<td>25-30</td>
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<tr>
<td>Obese</td>
<td>30</td>
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</table>


Figure 1. Transverse image of the lumbar region showing a midline dorsal spinous process with posterior acoustic shadowing and symmetric paraspinal musculature on either side.

Figure 3. Sagittal ultrasound image of a lumbar midline dorsal spinous process. Centered in image is the typical crescent shape of the spinous process with posterior acoustic shadowing.

Figure 4. Centered in the field of view is the soft tissue of the interspinous space in the sagittal plane. Note the presence of a dorsal spinous process on either side, with characteristic acoustic shadowing defining the superior and inferior borders of the interspinous space.


**Opening pressure**

Obtained in lateral recumbent position (lying on side) (REF: Loman. Effects of alteration in posture on the cerebrospinal fluid pressure. Arch Neurol Psychiatr 1934; 33: 1279–95.)


### Results

<table>
<thead>
<tr>
<th>Disease Specific</th>
<th>Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malignancy: cytology &amp; flow cytometry</td>
<td>Cytology &amp; Flow Cytometry for Leukemia/Lymphoma</td>
</tr>
<tr>
<td>Demyelinating: IgG index, oligoclonal bands</td>
<td>IgG Index, Oligoclonal Bands</td>
</tr>
<tr>
<td>Paraneoplastic/Autoimmune: autoantibodies</td>
<td>Autoantibodies</td>
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<tr>
<td>Immunocompromised: PCR for CMV, HHV6, JC virus, galactomannan for aspergillus</td>
<td>PCR for CMV, HHV6, JC Virus, Galactomannan for Aspergillus</td>
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<tr>
<td>Meningoencephalitis: Viral PCR (HSV1, HSV2, VZV, enterovirus, WNV, EBV), IgG/IgM (WNV, SLE, EBV), Bacterial PCR (tropheryma whippelli, bartonella, rickettsia), Syphilis (VDRL, Borrelia burgdorferi ELISA/Western), TB (PCR, AFB smear &amp; culture), Fungi (cx, crypto antigen, histoplasma antigen, coccidioides immunodiffusion or complement fix)</td>
<td>Viral PCR (HSV1, HSV2, VZV, Enterovirus, WNV, EBV), IgG/IgM (WNV, SLE, EBV), Bacterial PCR (Tropheryma Whippelli, Bartonella, Rickettsia), Syphilis (VDRL, Borrelia Burgdorferi ELISA/Western), TB (PCR, AFB Smear &amp; Culture), Fungi (Cx, Crypto Antigen, Histoplasma Antigen, Coccidioides Immunodiffusion or Complement Fix)</td>
</tr>
</tbody>
</table>

<p>| Type | Components | Normal (Adult) | DM | Bacterial Meningitis | Viral Meningitis | Crypto | HSV1 | HSV2 | LymA | SyphNia | Mycobacterial | Mycoplasma | Fungal | SAH | Traumatic |
|------|------------|---------------|----|---------------------|-----------------|--------|------|------|------|-------|--------------|------------|--------|--------|--------|---------|
| Clear | | 6-20 cm H2O | 0.6 serum 45-80 | 15-45 mg/dL | &lt;5/1 | 3 PMN | | | | | | | | | |
| Bacterial meningitis | &lt;200 WBC or &gt;400 RBC | &lt;40 | 23, 40 | 15 LR 0.5 | 4 LR 18, &gt;0.4 | 0.31, recovery first | | | | | | | | | |
| Viral meningitis | Usual y &gt;50% serum | &lt;150 | &lt;250-2000 | 2/3 PMN (lymph 12-24h) | | | | | | | | | | | |
| Crypto | | | | | | | | | | | | | | | | |
| HSV1 | | | | | | | | | | | | | | | | |
| HSV2 | | | | | | | | | | | | | | | | |
| LymA | | | | | | | | | | | | | | | | |
| SyphNia | | | | | | | | | | | | | | | | |
| Mycobacterial | | | | | | | | | | | | | | | | |
| Mycoplasma | | | | | | | | | | | | | | | | |
| Fungal | | | | | | | | | | | | | | | | |
| SAH | Bloody if &gt;6000 RBC, xanthochromia 2-4 h – 2-4 wk | | | | | | | | | | | | | | |
| Traumatic | No xanthochromia | 1mg/dl | 1/tube | 1 WBC/700 RBC (parrhe ratio) | | | | | | | | | | | |</p>
<table>
<thead>
<tr>
<th>Condition</th>
<th>&quot;but&quot;</th>
<th>&quot;18&quot;</th>
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<tbody>
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<td>CNS tumor</td>
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<td>Heme malign</td>
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<td>Idiopathic Intracranial Hypertension</td>
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<td>Idiopathic Intracranial Hypertension</td>
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<td>Post-LP (REF: Barry E., Hauser WA. Pleocytosis</td>
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<tr>
<td>after status epilepticus. Arch Neurol 1994;51</td>
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<td>(2):190-193)</td>
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<tr>
<td>Stroke (REF: Adams &amp; Victor, 8th ed.)</td>
<td>50-80</td>
<td>3-8</td>
<td>PMN</td>
</tr>
</tbody>
</table>


Differential for xanthochromia: SAH, protein 150 mg/dl, bilirubin >10-15 mg/dl, traumatic LP >100k RBC

Eos unhelpful? Broad differential: atypical infection, heme malign, SAH, obstructive hydrocephalus


Dementia


**Post Lumbar Puncture Headache (PLPHA)**


**Presentation**

Usually hours-3d post-LP

Worse w/standing

Commonly over frontal & occipital areas radiating to neck & shoulders

Usually severe, ‘searing & spreading’

Duration: ~25% <2d, 50% <4d, 70% <7d (REF: Vandam & Dripps 1956)

**Prevention**

Less w/"atraumatic" needle (Whitacre, 3%) than cutting (Quincke, 36%) (REF: Neurology 2005 Aug 23;65(4):510-2) (REF: AJR 2008; 190:1686-1689)

Less w/smaller bore/higher gauge needle (22G balances headache & fluid collection) (REF: Neurology. 2006 Oct 24;67(8):1492-4)

Less when bevel parallel to fibers (REF: Neurol Neurochir Pol 1999;32(suppl 6):179–182)

Less w/reinserting stylet before withdrawal (REF: J Neurol 1998; 245: 589–92)


**Treatment**
Standard analgesia (esp first 24 hr)


**Other Complications**

Backache (25%)

Radicular symptoms (15%)

Infection

Bleeding

Cerebral herniation

Epidermoid tumors

**Evoked potentials**

(REF: [http://www.uptodate.com/contents/clinical-neurophysiology?detectedLanguage=en&source=search_result&search=evoked+potentials&selectedTitle=1-150&provider=noProvider](http://www.uptodate.com/contents/clinical-neurophysiology?detectedLanguage=en&source=search_result&search=evoked+potentials&selectedTitle=1-150&provider=noProvider))

**Brainstem Auditory Evoked Potentials (BAEP)**

6 deflections in 10ms: serial ascending brainstem structures

Clearer ipsi structure

Subject: under either sedation or general anesthesia, even pentobarb (though perhaps not lidocaine & thiopental), but not in brain death

Stim: broadband monaural click stimulation is used on the ear tested while a masking noise 30-40 dB lower in intensity is used on the contralateral ear. 65-70 dB above click perception threshold, rate of 10 Hz

Electrodes: Ear lobes & Cz (vertex). Ref: A1/2 - Cz. + at Cz, therefore waveforms -, therefore upgoing

Recording: First 10ms, 2000-4000 responses x 2 separate trials

**Postulated Generators**

Wave I: Action potential of cranial nerve (CN) VIII

Wave II: Cochlear nucleus (and CN VIII)

Wave III: Ipsilateral superior olivary nucleus

Wave IV: Nucleus or axons of lateral lemniscus

Wave V: Inferior colliculus

Wave V latency shifts are used to estimate the amount of hearing loss


**Clinical Use**

Newborn screening

multiple sclerosis (MS)

acoustic neuroma

intraop monitoring during cerebellopontine angle tumor surgery

**Somatosensory Evoked Potentials (SSEP)**
Median Nerve Stim | Location | Tibial Nerve Stim
---|---|---
N5 | Median/Ulnar nerve | |
N9 | Brachial Plexus: Erb’s Point | |
N11 | Dorsal Root Entry/Dorsal column? | N22
N13 | Dorsal Column Nuclei: Nuc Cuneatus & Gracilis | N29
P14 | Medial Lemniscus | |
N18 | Brainstem vs. Thalamus | N34
N20 | 1º Sensory Cortex | P37
P22 | 1º Motor Cortex | |


Subjects: Change with sleep
Stim: Need not be supramax, dur 200-300ms, Rep 3 Hz
Filter: 10-2500 Hz
Electrodes: Arm Study: C3/4 ref to Fz; Spine Cervical 2 ref to Erb point/Brachial Plexus; Fz to contralateral cortex
Electrodes: Leg Study: L1, L3 to hip; Cz to Fpz
Clinical Utility: MS, Spinal disease, Post-arrest prognosis, Intraop SEP, metabolic: B12, thyroid

(REF: http://www.uptodate.com/contents/image?imageKey=RHEUM%2F68888&topicKey=NEURO%2FS274&rank=1-150&source=see_link&search=evoked+potentials&utmPopUp=true)


(REF: http://nelsoneeg.net/available_investigations.htm)

Figure 1 Test of somatosensory evoked potentials (SEP). This is a test of the sensory system whereby a peripheral nerve, normally the median or tibial, is stimulated with small electric shocks. The most important evoked response peaks are marked on the patient. The printout on the upper left is from an examination of a patient with cerebral anoxia damage. In SEP tests there was no response from cortical N20 bilaterally in channels 3 and 4. There was a normal response from the plexus brachialis (N9, channel 1) and cervical medulla (N13, channel 2). Note that intact P15 and N18 in channel 4 (reference electrode on the earlobe) indicate that the brainstem function in the medial lemniscus is intact. Such findings indicate that there is a poor prognosis for regaining consciousness. The printout to the right is from a patient with paraesthesia and a sense of heaviness in the right arm and leg. It shows normal left-side median SEP peaks from the brachial plexus (N9), cervical medulla (N13) and sensory cortex (N20). However, N20 latency is significantly increased after stimulation of the right side (24 ms) compared to the left (18.7 ms). Double arrows: Central conduction time (CCT = N20-N13, upper normal limit 7.2 ms) is longer in the medial lemniscus system on the right side (11.4 ms) than the left (5.4 ms). A cross-section from T2-weighted MRI at level C2 with a wedge-shaped signal increase dorsally which affects the right cuneate fasciculus (see single arrow) is also shown. The findings indicated a clinically isolated syndrome, and not a definite multiple sclerosis. The printout to the bottom right is from a patient with intermittent blurred vision in the one eye for the last couple of years. The printout shows tibial SEP with prolonged latency for the evoked potential P40 from the sensory cortex. Central conduction time is also considerably prolonged (P40-N22 = 34.1 ms, upper normal limit 22 ms). Prolonged P40 latency was also found on the right side, and both the median nerve SEP and VEPs showed bilateral latency prolongation, while BAEPs were slightly abnormal on the right side. Note normal peripheral potential from the left fossa poplitea (FoP1) and normal lumbar potential from the electrode over Th12 in the midline (N22). These findings, coupled with inter alia MRI findings and spinal fluid tests, were consistent with multiple sclerosis (REF: http://tidsskriftet.no/article/3011088/en_GB)

Visual Evoked Potentials (VEP)
Other causes: ocular conditions (such as glaucoma), compressive lesions of the optic nerve (such as pituitary lesions), and pathological conditions of the optic radiations or occipital cortex.

Other causes:

<table>
<thead>
<tr>
<th>Timing</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>N75</td>
<td></td>
</tr>
<tr>
<td>P100</td>
<td>Calcarine Cortex</td>
</tr>
<tr>
<td>N145</td>
<td></td>
</tr>
</tbody>
</table>

W shape may become more unimodal when stim from 1Hz -> 2Hz

Delays most likely 2/2 prechiasmal lesions, given monocular stimulation

Sedation & anesthesia abolish the VEP

Stim: A checkerboard pattern (or, less often, a flash) is used as stimulation, 1-2 Hz

Electrodes: Oz, O1, and O2 and with hemifield studies at T5 and T6

Subject: distance of 70-100 cm from the monitor screen, which gives a check size of approximately 30 seconds of visual angle. The vision should be corrected to the extent possible in case of a visual problem.

Recording: 1-200 Hz, 250 ms, 50-200 responses x 2 trials


**Advantages**

It is more sensitive than MRI or physical examination for prechiasmatic lesions

It is an objective and reproducible test for optic nerve function

The abnormality observed persists over long periods

It is inexpensive as compared with MRI

Under certain circumstances, it may be helpful for positively establishing optic nerve function in patients with a subjective complaint of visual loss; a normal VEP virtually excludes an optic nerve or anterior chiasmatic lesion


**Pathology**

**Biopsy**

Brain

Nerve

Muscle

**Patterns of Injury**

(REF: Robbins)

**Hypoxic-Ischemic**

FIGURE 28-13Cerebral infarction. A. At low magnification it is possible to see the demarcated areas of an acute infarction. In the underlying white matter, the areas of infarction are well shown by the myelin stain. B. Acute ischemic injury causes diffuse eosinophilia of neurons, which are beginning to shrink. C, Infiltration of a cerebral infarct by neutrophils begins at the edges of the lesion where vascular supply has remained intact. D, After about 10 days, an area of infarction is characterized by the presence of macrophages and surrounding reactive gliosis. E, Remote small intracortical infarcts are seen as areas of tissue loss with residual gliosis.
**Subacute and chronic neuronal injury**

Cell loss and reactive gliosis (e.g., ALS)

**Neuronal Inclusions**

- Aging
- Infections
- Metabolic
- Neurodegenerative

**Gliosis**

Gliosis (or astrogliosis) is the most important histopathologic indicator of CNS injury, regardless of etiology, and is characterized by both hypertrophy and hyperplasia.

**Alzheimer type II astrocyte**

Not AD, rather metabolic, esp ammonia/urea

gray-matter cell with a large (two to three times normal) nucleus, pale-staining central chromatin, an intranuclear glycogen droplet, and a prominent nuclear membrane and nucleolus. Its name is a misnomer, as it is mainly seen not in Alzheimer disease but in individuals with long-standing hyperammonemia due to chronic liver disease, Wilson disease, or hereditary metabolic disorders of the urea cycle

**Rosenthal fibers**

Thick, elongated, brightly eosinophilic, somewhat irregular structures that occur within astrocytic processes, and contain two heat-shock proteins (B-crystallin and hsp27) as well as ubiquitin. Rosenthal fibers are typically found in regions of long-standing gliosis; they are also characteristic of one type of glial tumor, pilocytic astrocytoma

**Reactions of Microglia to Injury**

Microglia are mesoderm-derived cells whose primary function is to serve as a fixed macrophage system in the CNS. They share many surface markers with peripheral monocytes/macrophages (such as CR3 and CD68). They respond to injury by (1) proliferating; (2) developing elongated nuclei (rod cells), as in neurosyphilis; (3) forming aggregates about small foci of tissue necrosis (microglial nodules); or (4) congregating around cell bodies of dying neurons (neuronophagia). In addition to resident microglia, blood-derived macrophages are the principal phagocytic cells present in inflammatory foci.

**Structures**

**Rosettes & Pseudo-rosettes**

<table>
<thead>
<tr>
<th>Rosette Type</th>
<th>Associated Tumors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Homer Wright rosette</td>
<td>Neuroblastoma, medulloblastoma, primitive neuroectodermal tumor, pineoblastoma</td>
</tr>
<tr>
<td>Flexner-Wintersteiner rosette</td>
<td>Retinoblastoma, pineoblastoma, medulloepithelioma</td>
</tr>
<tr>
<td>True ependymal rosette</td>
<td>Ependymoma</td>
</tr>
<tr>
<td>Perivascular pseudorosette</td>
<td>Ependymoma, medulloblastoma, primitive neuroectodermal tumor, central neurocytoma, glioblastoma, monomorphous pilomyxoid astrocytomas</td>
</tr>
<tr>
<td>Pineocytomatous rosette</td>
<td>Pineocytoma</td>
</tr>
<tr>
<td>Neurocytic rosette</td>
<td>Central neurocytoma</td>
</tr>
</tbody>
</table>

(REF: [http://www.ajnr.org/content/27/3/488.full](http://www.ajnr.org/content/27/3/488.full))
Rosette:

Diagram of a typical rosette demonstrating a halo of cells surrounding a central lumen. Adapted from Ellison et al (2004)34 with permission.


Photomicrograph from a PNET demonstrating multiple Homer Wright rosettes. The halo-like cluster of cells in each rosette surrounds a central area of fiber-rich neuropil (H&E; original magnification 400×).

Diagram of Flexner-Wintersteiner rosette. A halo of cells surrounds a largely empty central hub. Small cytoplasmic extensions from the cells project into the lumen. Adapted from Ellison et al (2004)34 with permission.

Photomicrograph from a retinoblastoma showing multiple Flexner-Wintersteiner rosettes. The halo-like cluster of cells in each rosette surrounds a nearly empty appearing central lumen containing fine cytoplasmic processes (H&E; original magnification 400×). Photomicrograph generously donated by Dr. Morton Smith, Ophthalmic Pathology, Washington University, St. Louis.


Photomicrograph from an ependymoma showing several true ependymal rosettes. The halo-like cluster of cells in each rosette surrounds an empty central lumen (H&E; original magnification 400×).


Photomicrograph from an ependymoma showing 2 prominent perivascular pseudorosettes. The halo-like cluster of cells in each rosette surrounds a blood vessel. Note the several smaller true ependymal rosettes (H&E; original magnification 200×).

Diagram of neurocytic rosette. This rosette is similar to the Homer Wright rosette, but the central fiber-rich neuropil island is larger and more irregular. Adapted from Ellison et al (2004)34 with permission.

Photomicrograph from a central neurocytoma containing an irregularly shaped neurocytic rosette with central neuropil (H&E; original magnification 400×).

Other Studies

Labs

Electrolytes
BUN/Cr
Glucose
CBC/Diff
LFTs
NH3
Infectious
Demyelinating
Coagulation
Urine

Ultrasound

Transcranial Doppler

Contraindications

Virtually none
Findings
Quick, bedside test of flow in the cranial vault
Finds blood vessels & gets pulsed flow in each blood vessel
Can provide evidence for emboli, stenosis, vasospasm, hemorrhage

**Carotid Ultrasound**

Contraindications
Virtually none

Findings
Measures internal carotid in the neck: peak systolic & end diastolic velocity, & ratio of middle to distal carotid velocities
Can look at soft tissue, intima, plaque
Large clinical trials of carotid stenosis are based solely on ultrasound (REF: N Engl J Med. 1991;325:445-453)

**Cerebral Angiography**

Contraindications
Bleeding
High creatinine
Recent dye exposure

Findings
Direct visualization of arterial or venous system
Gold standard for vasculitis, aneurysm, AVM
Allows catheter based interventions for aneurysms & ischemic stroke

Other Complications
Infection
Stroke
Bleeding / blood vessel puncture
Femoral bleeding
Contrast nephropathy

**EEG**
See sz section

**EMG/Nerve Conduction Studies**
See peripheral section