A practical Primer for Neurosurgery

The amount of information in the field of neurosurgery can be overwhelming. The following materials have been prepared for use as guidelines. While this may not be all-encompassing, it provides good guidelines and a foundation for further learning.

I. The Brief Neurological Exam
II. Notewriting (SOAP, ICU, Pre-Pop, Brief Op, Post-Op, Procedure)
III. Basic anatomy (Brain, Spine, Cranial Nerves)
IV. Imaging (CT, MRI/MRA, Angiography): Brain & Spine
V. Trauma
   a. Skull fractures, Intracranial hemorrhage, CSF leaks/pneumocephalus
   b. Spine fractures / kislocations, spinal cord injury
   c. Peripheral Nerve Injury
VI. Neurocritical care (drugs, monitors, general management guidelines for coma, SAH, SHI, SCI, CSF leaks)
VII. Neuroncology (Common tumors of brain & spine; benign, malignant, metastatic)
VIII. Neurovascular (Stroke – ischemic vs hemorrhagic; aneurysms; vascular malformations)
IX. Pediatric / Congenital (Hydrocephalus, Encephalocele/Myelomeningocele, Premature IVH, Cerebral Palsy)
X. Functional (Movement disorders)
XI. Epilepsy (temporal, extratemporal)
XII. Infections (osteomyelitis, epidural abscess, brain abscess)
XIII. Inflammatory disorders (multiple sclerosis, transverse myelopathy – learned in neurology rotation)
XIV. Degenerative diseases (brain: dementia vs delirium –learned in neurology/psychiatry rotations); (of spine: HNP, spinal stenosis); (of peripheral nerve: carpal tunnel syndrome, ulnar neuropathy)
XV. Radiation therapy (Gamma Knife, external beam)
XVI. Syndromes (requiring Neurosurgical intervention)
I. The Brief Neurological Exam

MSE – Awake, A & O x 4 (name, location, date, circumstance)
CNE – II-XII (II: Visual Acuity; III, IV, VI: EOM’s (H motion); VII: Facial (symmetric eye closure but crooked smile = central; one-sided face/eye closure weakness = peripheral); VIII: Hearing; X: Palate lifts symmetrically; XI: Shrug shoulder; XII: protrude tongue & move side to side
Motor – Check pronator drift; more detailed: UE muscle groups: D/B/T/WE/FI/G; LE muscle groups; HF/Q/ADF/EHL/APF, corresponds to CF-T1, L2-S1 nerve roots, respectively
** Grade: 5=normal; 4=slightly weak; 3=barely antigravity; 2=moves with gravity eliminated; 1=slight movement; 0=no movement
Sensory – per dermatomal chart – of note, T4=nipples; T10=umbilicus; L4=knee; Foot (medial dorsum=L4; middle dorsum=L5; plantar=S1); S2-S4=perineal sensation
Reflex-UE: Bi/Tri/BR; LE: KJ, AJ (check for ankle clonus); Babinski
**Grade: 2+=normal; 3+=hyperreflexia without clonus; 4+=hyperreflexia with clonus (specify how many beats of clonus, with >10 beats = “sustained clonus”); 1+=hyporeflexia; 0=no reflex movement
Cerebellar – FTN, gait

Variation on the brief exam

MSE: If mental status issues more concerning – naming (3 objects); short-term memory; repeat objects after 3 minutes; Long-term memory: Name first president, etc. (if more detailed desired, can do mini-mental status exam); note affect (blunted in frontal lobe injury)
CNE: Comatose patient: II, III; Pupillary light reflex; III, VI, VIII: Cold warer calorics: V, VII; Corneals: IX, X: Gag
Motor: Bulk, tone, strength of muscles
Sensory: Pinprick & fine touch, two point discrimination, proprioception
Reflex: Jaw jerk, if concern of localizing brainstem/upper cervical cord lesion: Bulbocavernosus reflex if concern of spinal cord lesion (** Rectal exam must be documented on a spinal cord/cauda equine lesion concern)
Cerebellar: Scanning speech, rapid alternating movements, intention tremor

Of use is that UMN lesions result in spastic weakness↑DTRs, clonus, ⊕Hoffman’s, ⊕Babinski and NO muscle atrophy. On the other hand, LMN lesions usually will result in flaccid weakness, ↓DTRs, atrophy and atony. Lastly, anterior horn lesions can cause fasciculations and fibrillations. Comatose patients should be checked for gag (IX, X), corneal (V, VII) and pupillary light reflexes (II, III).
When examining the child or infant, one must remember to carefully inspect the extremities, as shortened or atrophic extremities can often indicate abnormalities in the contralateral nervous system. Also, one should systematically examine the integument, for dermatological anomalies can point to certain diagnoses. The back should also be examined for defects, and the head should be palpated and fontanelles examined for tension and size. Lastly, when performing diagnostic procedures (LP, ICP monitoring) one should keep in mind the unique anatomy and physiology of the developing brain and spinal cord.

II. Notewriting

All notes must have date and time, “Neurosurgery:”, and level of person writing it: all notes must be signed and have the last name and level of person writing it spelled out legibly at the end, along with the pager number listed at the end.

H & P
CC/HPI
PMH/ROS
PSH
SH
FH
Allergies
Meds
PE: General + VS
   Neuro: MSE
      CNE
      Motor
      Sensory
      Reflex
      Cerebellar

Lungs
Heart
Abdomen
Other findings (as indicated)

Labs
Studies (Imaging)
Assessment:
Plan:

SOAP (for routine ward patient)
Subjective
Objective
Assessment
Plan

A SOAP note should take no more than 15 minutes to gather the data for a daily note. One minute for looking up and writing vital signs, 3 minutes for looking up and writing down vital signs (only note abnormalities; if no abnormalities, write “vital signs normal” Not stable (which only means they didn’t change – good or bad). An additional 8 minutes can be used to examine the patient with a focused exam (focus on the area of operation – if somebody had a back operation, you don’t need to do a cranial nerve exam). Use the final 3 minutes to write the SOAP note. Always look at the wound yourself – don not trust anybody else’s exam.

ICU Note (** Be efficient. Organized by systems – one system per line; on the left is data for that system, and on the right is the plan. Do not rewrite the plan at the end – just look at the right column for plans – these plans then often become “Orders”) – this is an exemplary; abridged format; detailed standardized forms are found in the ICU and used daily for patient care.

System
Plan

Neuro
A&O x 4: PERRL; EOMI.; Drift; MAE
? CT Scan

CV
MAP, CPP, ICP; EVD output (&level set)
? Raise EVD level

Resp
ABG on Vent settings
? Vent Changes

ID
T-max on Abx (Day : ___/___) for each Abx
? Change Abx/CVL

GI/Nutr
Feeds: ____@__ cc/hr, total ___ calories
? Change diet

Renal
UO__cc/hr
? Change ivf

Labs
BMP, CBC, Coags(+S-osm if on mannitol, Drug levels)
? Change Dose

To write these notes, become familiar with the flowsheet. ICU notes (and for that matter, patient care) is much easier when you follow a specific format as noted, by systems.

Op Note (See our standardized forms for details-this is just an abridged introduction.)
Consent properly filled out & signed, on chart?
Pre-Op Labs (CBC, BMP, Coags, Blood available (if needed), ensure Cx(-)
EKG, CXR (if indicated): Results
NPO after midnight (check if order written)
Brief Op Note (See our standardized forms for details-this is just an abridged introduction.)
Pre-Op Dx:
Post-Op Dx:
Procedure:
Surgeons:
Anesthesia (type):
EBL, I/O (from anesthesia)
Drains (+ location)
Specimens (to pathology)
Condition
Disposition

Post-Op Note
Neuro exam
VS
Labs
Studies (if indicated)
A/P:

Bedside procedure note
Procedure, technique, location (including side), performed by whom, any complications, post-procedure check.

Extubation note
Pre-extubation data (e.g. ABG, NIF/FVC); post-extubation data (O2 sat, RR, ABG after 30 minutes, etc.); A/P
III. Basic Anatomy

It’s assumed you have had a basic Neuroscience course, and have learned your anatomy there. Specifics emphasized in Neurosurgery are mentioned here.

1. Head
   a. Coronal suture-Approximately 2 cm posterior is the motor cortex (important for EVD insertion-always stay anterior to the coronal suture)
   b. Pterion-The intersection of the frontal, temporal, parietal, and sphenoid (greater wing) bones-important for a pterional craniotomy, commonly used for some skull base procedures.
   c. Frontal and temporal poles of Brain: Important sites for confusion following traumatic coup injuries; Occipital poles important contusion sites for contre-coup injuries.
   d. Central sulcus-Most likely to be seen as a symmetric line bilaterally, posteriorly on high T2-MRI cuts: critical for surgical planning, as the motor strip must not be damaged by approach.
   e. Arteries: ICA (extracranial)-recognized as not having branches (vs. ECA)
   f. ICA (intracranial)-Know all segments (petrous, cavernous, ophthalmic, intradural); know all branches (ophthalmic, superior hypophyseal, anterior choroidal, posterior communicating, MCA, ICA)
   g. Vertebral artery-Branches include anterior spinal artery, PICA (occlusion of PICA leads to Wallenberg’s lateral medullary syndrome, which spares pyramidal motor fibers, but affects sensation/proprioception/sympathetics/regional cranial nerves)
   h. Basilar artery-Branches include perforators to brainstem, AICA, SCA (most likely compressing fifth cranial nerve in trigeminal neuralgia, PCA (which normally supplies posterior medial cortex; in some cases, these are hypoplastic and fetal p-comm. Arteries supply these areas)
   i. Veins: Sinuses (Superior sagittal, inferior sagittal, straight, transverse, sigmoid, superior & inferior petrosal); Veins (internal cerebral, basal, Vein of Galen, Vein of Labbe, Vein of Trolard, internal jugular vein)
   j. Cranial nerves:

Know the names of all CN’s, exiting foraminae, and functions.
Cavernous sinus structures (ICA, III, IV, VI, V1, V2)
Optic canal structures (Optic nerve, ophthalmic artery)
CN I is the most commonly damaged in trauma (not usually tested)
CN VI paresis is a common “false localization sign” of increased ICP

2. Spine
   a. Occiput-C1-C2 relationship: Maintained by various ligaments; of these the transverse ligament is one of the most important.
   b. The spine from C3 onwards is divided into a 3-column model: the anterior column extends to the middle of the vertebral body; the middle column extends to the pedicles; and the posterior column includes the facets, laminae, and spinous processes (with the interspinous and other posterior ligaments.) Damage to 2 columns is suggestive of an
unstable injury which will likely need surgical intervention. The thoracic spine is more stable because of associated support from the ribcage.

c. The vertebral artery enters at C6 transverse foramina, travels upwards, and makes a turn between C1 and occiput to pierce the dura and form the basilar artery. The vertebral artery gives off the anterior spinal arteries (ASA) which supply the anterolateral tracts bilaterally. The radicular branch off the left T10-L2 region, which is a major contribution to spinal cord blood flow; in periods of hypotension following which patients wake up, they may be paralyzed because of spinal cord infarct from the “watershed” region of low flow at the middle thoracic cord (between the arterial feeders).

d. The central canal, a potential space, may dilate with fluid in the case of a “syrinx”; this will cause compression of the anteriorly crossing spinothalamic fibers, resulting in sensory numbness along a “patchy distribution” to pinprick, which may be un affected more distally and proximally.

e. Range of motion: 50% of head rotation is due to the interaction of C1 and C2 (hence fusing these vertebrae will reduce this motion); 50% of head rocking motion (nodding yes) is due to occipit-C1 interaction (hence an occiput-C1 fusion will impair this ability)

f. Nerve roots
Sensory are dorsal, motor are ventral
An L4-5 disc herniation affects the L5 nerve root; an L4-5 far lateral disc herniation affects the L4 nerve root.

IV. Imaging
Plain X-rays
Not generally useful for skull fractures, rarely done
Very useful for C-spine:
Generally 3 views: AP/Lateral/Odontoid; may add Flexion/Extension
Ensure can see from occiput to C7-T1 intersection;
Lateral view: Look for alignment in 4 lines: anterior vertebral body, posterior vertebral body, spinolaminar line, and clival-C2 line (of Wackenheim) & fractures
AP view: look for fractures and splaying of lateral masses of C1 vs. C2: if the sum (of both sides) is greater than 6.9 mm, transverse ligament damage is suspected and the fracture may be unstable
Look for soft tissue swelling (<7 mm at C3, >21 mm at C7).
Flexion/Extension views can be used to supplement C-spine clearance.
More detailed information can be achieved through a CT/MRI

Checking alignment on lateral, odontoid, and AP views; note the critical lines outlined on the lateral radiograph.

CT Scan
Most useful primary test of head/brain, because calcium is bright (or hyperdense) therefore, skull is hyperdense, and so is acute blood (for about 72 hours); ischemic strokes will not show up on CT scan until 12-24 hours after they occur. Water is dark (therefore ventricles are dark, edema is dark, dead or dying brain tissue is darker than normal brain.)
CT scan is a useful test for spine only if C-spine plain x-rays not revealing along with tenderness or patient is comatose; can get axial and coronal reconstructions which will allow better assessment.

MRI/A
Brain: Not as good at CT for acute hemorrhage; better than CT for posterior fossa imaging, acute stroke imaging, and older bleed imaging; Two types (T1 showing CSF dark, and T2 showing CSF (and edema) white; contrast may be injected on a T1 sequence to see what “enhances” such as brain tumor with high vascularity). An MRA is an MR angiogram, which gives a rough idea of large blood vessel flow (but is not as good a conventional angiography).
Spine: Best test for looking at soft tissue of the spine (ligaments, spinal cord, blood, abscess) same principles as brain MRI apply with respect to T1, T2, etc; sagittal/axial cuts usually performed.

Angiography (X-RAY, CONVENTIONAL)
“Gold standard” to rule out and characterize vascular problems such as aneurysms and arteriovenous malformations. The catheter is introduced via the femoral artery, and advanced to the vessels supplying the brain, after which dye is injected. Alternatively, medications (such as papaverine), glue (for embolization of AVMs), and balloon dilation (angioplasty) of vasospasming vessels can be performed during angiography.
--Not always performed first because of risk of stroke (approximately 1-3%) and dissection for cerebral angiography; spinal angiography is very rarely performed due to higher risk (approx. 8%).

V. Trauma
  a. Skull fractures
  --If skin overlying fractures is open, the debridement is indicated, with antibiotic coverage.
  --Basal skull fractures may be indicated by Racoon’s eyes (periorbital) or Battle’s sign (retroauricular); sometimes, subconjunctival hemorrhages are noted.
  --Look for CSF leak, usually rhinorrhea (from nose) or otorrhea (from ear) with basal skull fractures (no antibiotics are needed for solely a CSF leak) – For treatment of CSF leak, keep HOB elevated at 30 degrees or higher X 1-2 days; if still leaking, consider CSF diversion (via lumbar drain or EVD) X 3-5 days; if still leaking, consider endoscopic or operative repair. Confirmation of CSF leak (vs. mucus or blood) is done via glucose check (less specific) vs. chloride vs. beta-transferrin (a.k.a. beta-microglobulin) (most specific).
  --The treatment of linear skull base fractures is non surgical (look for an underlying bleed)
  --The treatment of a depressed skull fracture is solely for cosmetic value. Generally, a full thickness-depressed skull fracture in a cosmetically important region is elevated surgically.
Pneumocephalus is air in the skull; it is treated by 100% oxygen (non-rebreather), to allow diffusion of the nitrogen (which is what air mostly contains) into the bloodstream. An emergent problem is tension pneumocephalus (from ball-valve effect, similar to tension pneumothorax) must be urgently decompressed via a puncture, before a patient dies from brainstem compression.

b. Hemorrhage (Traumatic) – can occur in a plane amongst and region of the head
   --Subgaleal – between the scalp and skull; no neurosurgical treatment
   --Epidural – between the skull and Dura; usually due to middle meningeal artery tear, often associated with a temporal skull fracture (may rarely be due to venous sinus tear underlying a fracture). Appearance on a CT is biconvex (cannot assume same shape as underlying brain, because the Dura is tethered to the skull at skull bone suture sites, and hence this limits the extent of the epidural hematoma. Classic presentation is initial loss of consciousness, from which they awaken and talk, and then acutely worsen – “They’re talking and they’re dead”. Needs acute surgical evacuation, and hemostasis of the bleeding vessel; in rare cases very small bleeds can be followed with close neurological observation.
   --Subdural – More frequently associated with underlying brain injury; due to cortical bridging vein tear; can be seen in sudden acceleration/deceleration (e.g. MVA, shaken baby syndrome). Appearance on CT scan is a “crescent shape” which confirms to the underlying brain. Treatment is surgical evacuation is sizeable; otherwise can observe very small bleeds.
   --Subarachnoid hemorrhage – Trauma is the most common cause of subarachnoid hemorrhage (NOT aneurysms or AVMs); usually this is of little consequence. Appearance of a CT scan is a feathery bleed pattern which conforms to, and may be interspersed between, individual gyri or in the cisterns/subarachnoid space surrounding the brain convexity.
   --Intraparenchymal hemorrhage – Usually secondary to a contusion (or bruising of the brain substance); can blossom into a sizable bleed over 24-72 hours. Treatment depends on location and size – deep brain hemorrhages have greater morbidity when surgically removed, if important brain must be traversed to evacuate the hematoma.
   --Intraventricular hemorrhage – Rare in trauma; can lead to hydrocephalus (by blocking CSF flow) if sizeable.
   --Spine bleeds are very rare (occasional epidural/subdural bleeds have been reported); these require evacuation if patient is symptomatic.

Herniation – one of the greatest concerns with any intracranial pathology. The skull is a fixed volume, and the brain can compress and infarct itself within it. Signs leading to it include brain edema, reduced cisternal space, temporal ventricular horns being visible on imaging study. Signs of herniation clinically include unilateral III n. palsy (ipsilateral papillary dilation); a phenomenon called “Kernohan’s notch” leads to ipsilateral weakness (because of the brainstem being compressed against the contralateral tentorial edge, before the corticospinal fibers have crossed) – this is a false localizing sign, since most of us would expect contralateral side weakness from an injury. Management of ICP and herniation is considered under the “Neurocritical care” section.
Spine fractures/dislocations & Spinal cord Injury
-- The first concept is taking spinal cord injury precautions (appropriate immobilization). If spinal cord injury is suggested by neurological exam and the patient is less than 8 hours following injury, a methylprednisolone drip (30 mg/kg/hour X 1 hour loading dose, then 5.4 mg/kg/hour over the next 23 hours) is started. Plain X-rays, CT scan, and MRI are useful studies and are generally obtained ASAP. There is little evidence for immediate surgical intervention in trauma (some would intervene earlier with a disc causing injury or a bleed, vs. for a fracture/dislocation). 6 types of fracture will be considered here: Jefferson (C1), Odontoid (C2, Types 1-3), Hangman’s (C2 pars interarticularis), anterior compression fracture, burst fracture, and Chance fracture.

--Odontoid fracture (Types I-III): Type I: tip of odontoid process only; Type II: through base of odontoid; Type III: through vertebral body, below base of odontoid. Types I may be treated in a C-collar, Type III can be treated with a halo orthosis. Type II can be treated with halo orthosis if it is less than 50 and displacement is less than 6mm; otherwise, it must be treated with surgical intervention (either posterior fusion or anterior odontoid screw placement).
--Hangman’s fracture: C2 pars interarticularis fracture, which is usually treated in a C-collar if <2mm displacement is present (considered a stable fracture); if >2mm displacement or significant angulation present, reduce in halo ring with traction and then place into halo; operative intervention may be indicated if fusion in halo ring is not accomplished over 3 months.
-- Anterior compression fractures: only the anterior portion of the body is compressed; usually this causes less than 50% reduction of body height, and can be managed conservatively. (i.e. this involves the first “column” as described under the “Anatomy” section.
--Burst fracture: a two-column injury (involves the anterior and middle column); treatment is based upon angulation of vertebrae, degree of vertebral compression, and canal compromise (generally, >50% body height compression, > 30 degree angulation, and/or > 50% canal compromise will lead to surgical intervention)
--Chance fracture: A two-column injury involving the middle and posterior columns; surgical intervention may be indicated according to the 2-column model and above considerations.
-- Fracture/dislocation: this can involve all 3-columns, resulting in complete spinal cord injury. Treated operatively, although does not need to be emergent. May require combined anterior & posterior internal fixation.

c. Peripheral nerve injury
-- Three types of acute traumatic peripheral nerve injury are recognized, according to severity: neuropraxia, axonotmesis, neurotmesis. Neuropraxia preserves anatomic integrity of neurons and myelin sheaths, with recovery within hours to months (generally 2 months); axonotmesis involves axon and myelin sheath damage, with connective tissue in continually; neurotmesis involves physical separation of two nerve ends, with scar tissue developing eventually. The treatment of axonotmesis and neurotmesis is surgical
approximation of severed nerve 6 months after injury, and after confirmation of lack of function via EMG and nerve action potentials (which must be done 2 weeks after injury, to avoid denervation artifact). An exception to this treatment is a stab-type injury, which requires approximation immediately (of the nerve sheaths), to allow best chance of returning nerve function at a later time.

**VI. Neurocritical Care:** This section is divided into three types of patients who will be cared for predominantly – Severe traumatic head injury, Spinal cord injury, Subarachnoid hemorrhage. Also, some info on electrolyte abnormalities, which may be encountered frequently in the neuro-ICU.

--Severe traumatic head injury patients are, by definition, GCS <=8 (see Appendix 4a for Glasgow Coma Scale). All patients should receive cerebral perfusion pressure (CPP) management. CPP is mean arterial pressure minus intracranial pressure (CPP= MAP – ICP). Usually, CPP>70 is aimed for in adults. This can be done in several ways – (1) Increasing MAP (give fluid, including albumin and ½ normal saline, to CVP of 10 – 12, being careful not to give too much volume to older patients or patients with heart failure; then, pressors such dopamine, levophed, and neosynephrine can be added in that order while maintaining optimal volume); (2) Reduce ICP – if ICP > 15 mm Hg is noted, the EVD will drain CSF (since it is usually set to drain at ICP > 10 mm Hg; also, mannitol (via osmotic diuresis and free-radical scavenging) can reduce intracranial pressure; hyperventilation reduces pCO2 and reduces blood flow to the brain, hence reducing ICP (but pCO2 must be maintained over 30, to avoid ischemia from decreasing blood flow too much); sedation and paralytic use can also help reduce ICP, as can cooling a febrile patient. As noted from the above, a patient suffering severe traumatic head injury should receive: intubation (with ventilator), CVL, A-Line, and EVD.

--Spinal cord injury patients, if injured within 8 hours, should receive the methylprednisolone regimen (30 mg/kg X 1 hour loading dose, and 5.4 mg/kg/hour over the next 23 hours). In addition, MAP > 85 should be maintained, using the methods of volume optimization and pressors as indicated (see above severe traumatic head injury section). Hence these patients will need a CVL and A-line.

--Subarachnoid hemorrhage patients (without history of trauma), if Hunt-Hess grade I or II, do not need a ventriculostomy under most circumstances. If a patient is sleepy, not following commands, and has hydrocephalus, an EVD should be placed. In addition, Nimodipine (60 mg sublingual/po q 4 hours, hold for SBP<110), albumin (12.5 g iv q 6 hours, hold for CVP>10), and ivf (1/2 NS @ 125 cc/hr) should be ordered. These patients should have orders for angiogram (if not already performed) to determine the source of the bleed, and such patients be kept in a quiet, dark room due to their severe headache and photophobia (caused by the bleed). Two units of blood (type & crossed) should be ordered, and hematocrit should be maintained higher than 30.

--Electrolyte Abnormalities. *Hyponatremia*. In neurosurgical patients is chiefly seen in SIADH, cerebral salt wasting and postoperative hyponatremia. Tx consists of repleting sodium (VERY SLOWLY!!! Do not exceed 1.3 ± 0.2 mEq/L/hr). SIADH results in hyponatremia usually accompanied with hypervolemia and it is seen with certain malignancies (as in bronchogenic CA), meningitis, trauma, ↑ICP, tumors, s/p crani, SAH, anemia (rarely) and drugs (tegretol, diabase, oxytocin, HCTZ). 3 diagnostic criteria
must be met: hyponatremia, inappropriately concentrated urine, and no evidence of renal or adrenal dysfxn (serum Na usually <134 mEq/L and S_{OSM}<280mOsm/L) The definitive test is the water load test. It consists of asking the patient to consume 20 mL/Kg of water (up to 1500 mL). The failure to excrete 65% of the water in 4 hr or 80% in 5 hr indicates SIADH (do not perform test if Serum Na ≤ 124 mEq/L). CEREBRAL SALT WASTING is defined as renal loss of Na as a result of intracranial disease, producing hyponatremia and decrease in ECF. This inability of the kidneys to conserve Na may be either as a result of an unidentified natriuretic factor or direct neural control mechs. Tx is with NS to replace volume and maintain a positive salt balance. Hypernatremia. On the other hand, hypernatremia is most often seen in the setting of DI. DI is usually due to low levels of ADH (or renal insensitivity to it) and the central subtype is seen in familial disease (AD), post-trauma patients (including surgery), tumor induced, and neurosarcoid among others. It results in high output of dilute urine with normal to high S_{OSM} and it is accompanied by the craving of water (in danger of severe dehydration if mismanaged). Central DI may be seen following pituitary surgery as ① transient DI when the UOP is supra-normal accompanied with polydipsia, both of which normalizes 12-36° post-op; ② “prolonged” DI: when UOP stays supra-normal for a prolonged period (months) or even permanently. ⅓ of these patients will not return to normal; and ③ “triphasic response:” phase 1.- ADH reduction due to pituitary injury for 4-5 days→DI; phase 2.- cell death liberates ADH for the next 4-5 days→transient normalization or even SIADH-like water retention; and phase 3.- reduced or absent ADH secretion→ either transient DI or “prolonged.” Also seen following brain death, aneurysm (or any lesion) pressing on hypothalamus, head injury (basilar skull fx), encephalitis/meningitis, drugs (ethanol, dilantin, steroids), and Wegener’s granulomatosis among others. Hypocalcemia. Total [Ca] < 8.8mg% seen in 10-15% of ICU pts. Results from ↓ sunlight exposure, ↓ dietary intake, ↑ metabolic demand, renal/liver dz, Ca chelation (citrate from transfused blood, chelators in X-ray contrast for CT and angios), ↑ UOP (lasix). Normal [Ca^{++}] is 4.25-5.25 mg%. Special attention to acute alkalosis (it ↑ prot binding, thus ↓ Ca^{++}), patients being hyperventilated (they may develop ionized hypocalcemia with tetany despite normal [Ca]), and hypoalbuminemic patients. Usually manifests itself with cardiovascular symptoms (hypotension, arrhythmias such as brady or V-fib, failure to respond to Ca mediated drugs and QT or ST-interval prolongation), neurologic (tetany, Chvostek’s sign, Trousseau’s sign, muscle spasm and cramps, paresthesias, AMS, and seizures) and respiratory symptoms (apnea, laryngospasm/stridor). Hypercalcemia. Usually due to malignancy in older pts and hyperparathyroidism in younger ones. See reduction of neuromuscular excitability. Tx with fluids. Hyper and Hypokalemia. Nothing in particular that applies only to neurosurgical pts. These abnormalities present themselves and are treated as described in your standard surgery textbook.

VII. Neuroncology

Brain tumors can be divided into 3 main categories: Benign, malignant, and metastatic from another part of the body.
Generally, a brain tumor causes mass effect in the confines of the skull, and hence causes ICP elevation. Slow ICP increase is tolerated much better than the brain than high ICP; hence, a softball-size meningioma is sometimes found in adults without much neurological deficit, whereas a bleed the same size may cause death immediately. Classically, headache due to elevated ICP may be worse in the morning, can become progressively worse over time, and is exacerbated by Valsava maneuvers. Edema (or associated swelling) can be related to the more aggressive nature of certain tumors. In any case where significant edema or mass effect is present, a lumbar puncture should be avoided, due to risk of herniation (either uncal or tonsillar).

Benign tumors include (1) Meningioma, which typically affects 40-50 year-old females who present with a headache or seizures, is durally based, and is usually outside the substance of the brain; treatment is resection when safely possible, with degree of recurrence inversely proportional to degree of resection (otherwise these tumors can be treated by gamma knife radiation if small and possible). (2) Pituitary adenomas can cause symptoms via either the hormones they secrete (ACTH, TSH, GH, ESH/LH - usually like nonsecreting) or mass effect (which can cause bitemporal hemianopsia); they are treated usually by a transsphenoidal approach (entry through the nasal cavity, with the incision either sublabial or under the bridge of the nose). 30% recur over years, and these tumors are very rarely invasive. (3) Acoustic neurinomas are schwannomas of the VIIIth nerve (usually off the superior vestibular branch) which cause hearing loss; treatment is surgical resection (or gamma knife radiation for smaller tumors), with usually good results; (4) Hemangioblastomas are tumors of blood vessel-associated cells (often cystic), which occur with the Von Hippel Lindau complex (see the section on “ Syndromes associated with neurosurgical problems” which follows); these are benign tumors, with the preferred treatment being surgical resection. (5) Craniopharyngiomas, a suprasellar tumor, can present with hypothalamic dysfunction (such as diabetes insipidus) or optic nerve symptoms as it grows downwards; treatment is resection and/or radiation therapy; this tumor is common in childhood and in the elderly adults, and prognosis depends on treatment morbidity and recurrence. In children infratentorial (posterior fossa) primary tumors are more common than in adults. The most common primary posterior fossa tumor in children is a pilocytic astrocytoma; with complete resection, prognosis can be excellent for this benign tumor.

Malignant tumors are most commonly gliomas, which are divided into three main categories: astrocytoma, anaplastic astrocytoma, and glioblastoma. Astrocytomas have nuclear atypia and hypercellularity; anaplastic astrocytomas show the same features with the addition of endothelial cell proliferation and GBM shows all of those three features with the addition of necrosis. There is controversy as to whether any glioma should be resected in adults, especially from a deep location (many people do a biopsy to confirm diagnosis and either observe with follow-up scans or radiate). Although controversial, many neurosurgeons resect anaplastic astrocytomas and glioblastomas (usually for reduction of mass effect), followed by radiation therapy. Average survival with astrocytoma is 10 years, with anaplastic astrocytoma is 2 years, and with glioblastoma is 11 months in adults. In children, the survival is higher, with resection leading to some benefit. Common malignant tumors for children which occur in the posterior fossa
include medulloblastoma and ependymoma is resection, with gross total resection leading to better prognosis. For ependymomas, gross total resection is considered a cure. Medulloblastomas, however, require craniospinal radiation post-operatively despite gross total resection.

Metastatic tumors: Metastases from the lung, breast, and renal areas are most common tumors to invade the brain; alternatively, of all primary tumors, melanoma is the most likely to go to the brain (although not the most frequent). Metastic tumors usually occur in older adults, and are the most common tumor to occur in the posterior fossa. Typically, they are found along the grey-white matter interface, with surrounding edema and contrast-enhancement on scans. Symptoms depend upon location. Treatment is surgical resection followed by radiation for a solitary metastasis, and multiple metastatic lesions are currently treated by gamma knife. Average survival is 8 months with single metastasis following resection and radiation.

Spine tumors can be divided into 3 categories, based upon their location: extradural, intradural extramedullary, and intramedullary. Extradural tumors: In adults, metastatic tumors to the vertebral body/other elements are the most common extradural tumors; in children neuroblastoma may contribute. Breast cancer, prostate cancer, lung cancer, and renal carcinoma are the most common to cause epidural compression. One way they travel is via Batson’s plexus, a collection of epidural veins. Multiple myeloma (or in solitary form, plasmacytoma) may contribute as well. Treatment is radiation (except for renal cell, which is relatively radioresistant); surgery is indicated for stability or mass effect reduction with radiation failure/contraindication. A CT-guided needle biopsy can be performed by radiology to establish diagnosis, if none exists prior to radiation. Intradural extramedullary: In adults, the common two are meningioma and schwannoma. Meningioma occur in females 40-50 years of age (more so in the spine than the head), with radioculopathy; treatment is surgical resection. Schwannomas arise from a (sensory) nerve rootlet, and are treated by resection. Schwannomas (more than meningiomas) can possibly have a “dumbbell” appearance, with portions of the tumor extending outside the neural foramen. Neurofibromas are more intermingled with the nerve root, and hence more difficult to resect without causing significant deficit. Intramedullary: In adults, ependymoma, and hemangioblastoma are the most common intramedullary spinal cord tumors (in that order); in children, the order changes to astrocytoma, ependymoma, and hemangioblastoma. Treatment is surgical resection, which is easier for ependymoma and hemangioblastoma as a distinct plane usually found; it is more difficult for astrocytoma, which may be treated by radiation. Hemangioblastomas occur as part of the VHL complex (see section on “Syndromes associated with neurosurgical problems”).

VIII. Neurovascular
Vascular problems requiring neurosurgical treatment include treatment of ischemic stroke etiology (via carotid endarterectomy), hemorrhagic stroke management in some cases, aneurysms, and vascular malformations.
Ischemic stroke: Cartotid endarterectomy has been shown to reduce the risk of stroke if performed by experienced surgeons with low complication rates—the symptomatic study showed improvement for 70% stenosis of the ICA, whereas the asymptomatic study showed improvement for 60% stenosis of the ICA. Diagnosis by angiography (usually conventional angiograph, but sometimes MRA and/or Doppler can be used, although these can overestimate stenosis). Sources of embolic stroke can be cardiac or carotid (just distal to the CCA bifurcation), so an echocardiogram is indicated as well. Some favor heparin and/or warfarin for stroke, although this is controversial; some risk of converting an ischemic infarct into a hemorrhagic infarct exists.

Hemorrhagic stroke—usually due to hypertensive disease (most common sites are basal ganglia, thalamus, pons, cerebellum. Sometimes the hemorrhage extends into the ventricular system, leading to hydrocephalus. In such cases, EVD placement may facilitate decreasing ICP. Patients, depending upon their condition, may require intubation, EVD, and treatment with blood pressure controlled (not too high but too low, so that CCP is maintained). An additional cause of hemorrhagic stroke is cerebral amyloid angiopathy, mainly in elderly adults, which can present lobar hemorrhage.

Aneurysms—often occurring at bifurcations of intracranial vessels and representing defects in the media layer, these dilatations have a very low rate of rupture (0.05% per year for anterior circulation, for less than 1 cm.); this is 10 times as high (0.5% per year) for posterior circulation aneurysms, and may increase by aneurysm size. Female sex, hypertension, and smoking contribute to morbidity associated with aneurismal rupture according to some studies. Most common aneurism sites are anterior communicating artery, posterior communicating artery, internal carotid artery bifurcation, and middle cerebral artery, posterior communicating artery, internal carotid artery bifurcation, and middle cerebral artery. Subarachnoid hemorrhage presents as an acute onset headache (“worst in life”) with some Valsalva maneuver sometimes (like lifting a box or straining on toilet); over a third die soon thereafter. Of the remainder, different symptoms occur, depending upon aneurysm). The Hunt-hess grading system describes patient clinical status at admission (see Appendix 4d). A delayed complication which a sudden neurological deficit may occur, due to a (presumably unknown) factor in the blood. Treatment of vasospasm is triple-H (HHH=Hypertension, Hemodilution, Hypervolemia), which is done mainly via fluids; angioplasty may be indicted if HHH doesn’t work. Treatment of aneurysm is surgical clipping or endovascular coiling. Complications related to aneurysm rupture include rebleeding, cerebral ischemia secondary to vasospasm, and hydrocephalus (obstructive or communicating hydrocephalus, see “Pediatric/Congenital” section for elaboration of the difference between these two types).

Vascular malformations—there are four types of vascular malformations: arteriovenous malformations (AVMs are congenital lesions whereupon blood flows from arteries to veins, bypassing capillaries; this causes a rupture and bleed at a rate of 4% per year. There are several types of AVMs, classified by their flow state (low vs. high) or feeding vessels (dural vs. parenchymal), and gamma knife if the lesion is small/located in eloquent cortex); pre-procedure. Cavernous malformations are large-walled vascular
tumors which bleed at a rate of about 1% per year; they have a characteristic “popcorn” appearance on MRI, with a hemosiderin stain around the lesion; treatment is surgical excision. Capillary teleangiectasias are not visualized by angiography, and are solely a pathological diagnosis; these are very tiny, and little is known about them. Venous angiomas are congenital venous variations; these do not bleed, and removing a venous angioma can lead to a stroke.

IX. Pediatric/Congenital

Six common problems are considered here –hydrocephalus, encephalocele/myelomenigocele, Chiari malformations, premature IVH, cerebral palsy, and craniosynostosis.

Hydrocephalus can be due to any obstruction of the cerebrospinal fluid pathways (CSF is produced by the choroid plexus in lateral ventricles/tela choroida of the 3rd ventricle and fourth ventricle); from the lateral ventricles it flows through the foramen of Monroe into the third ventricle, through the aqueduct of Sylvius into the fourth ventricle and out the foramina of Magendie (single, midline) and Luschka (two, lateral) into the subarchnoid space where it is absorbed by arachnoid villi along the superior sagittal sinus. Any obstruction can which cause hydrocephalus along this path (except at the arachnoid granulations) is called “obstructive hydrocephalus”; if the arachnoid granulations themselves are clogged (i.e. the lateral, third, and fourth ventricles are all large) then the hydrocephalus is termed “communicating hydrocephalus.” Communicating hydrocephalus can be caused by anything which occludes the ability of CSF to flow through the arachnoid granulations (e.g. subarachnoid hemorrhage, meningitis (infectious or carcinomatous), or due to excess CSF production (e.g. by choroids plexus papilloma or choroids plexus carcinoma, two tumors originating from the cells of the choroids plexus which provide CSF). Treatment of hydrocephalus is placement of a ventriculoperitoneal (or ventriculopleural or ventriculoatrial) shunt. Shunts are prone to malfunction in children (average life is 3-5 years although there is data documenting up to 20-30 years) or infection (usually within 6 months of placement). And the most common cause of malfunction within the first six months post-shunt is infection. Increased head circumference, anterior fontanelle (bulging vs. flat), and/or nausea/vomiting/headache may suggest hydrocephalus. A CT scan (along with a shunt series or X-rays to ensure shunt tubing continually in a previously placed shunt) is important to diagnose hydrocephalus, either initially or following previous shunt malfunction. An alternative treatment is endoscopic third ventriculostomy, but this is not so effective if the arachnoid villi are the site of obstruction.

Premature IVH is defined as bleeding into the ventricles of the brain from the germinal matrix in a premature baby. The germinal matrix is a part of the brain that is active during fetal development but that disappears at about the 35th week of pregnancy. The blood vessels contained in this area are thin and vulnerable to fluctuations in blood flow.
through them, which can cause them to rupture and bleed. The younger and smaller the baby, the higher the risk these blood vessels may be ruptured, usually in the first few days of life. A rupture causes blood to flow into a ventricle or ventricles of the brain. IVH is categorized into grades of severity: grade I is considered mild, grade II moderate, and grade III & IV severe. About 50% of extremely premature babies will sustain an IVH, whereas only about 15% of older premature babies, many of whose germinal matrix has already disappeared, will have an IVH. If the IVH is classified as grade I or II, the chance that there will be long-term damage is small because the blood remains contained within the ventricles and the additional fluid does not cause excessive pressure. A grade IV IVH results from congestion to the brain tissue around the ventricles when a large IVH has occurred. This results in bleeding into the brain tissue itself with destruction of that area of brain. Lasting brain damage is almost always the result, the severity of which is determined by the extent and location of the bleeding. Since the ventricular system is obstructed by blood in these cases, temporary treatment options other than shunts are usually tried first. Interventions such as a ventricular tap, subgaleal shunt placement, or a ventriculostomy are considered. Up to a month of “temporary treatments” may be necessary before the permanent shunt can be placed.

Encephaloceles are defects in the skull which can lead to protruding brain which is often dysfunctional, named according to region (occipital, nasal, etc.). Myelomeningoceles (MMC) involve nervous tissue and meninges in a “sac” which did not properly form, and is located at the lumbosacral region. The incidence of MMC can be lowered significantly by taking folic acid before becoming pregnant. Patients with MMCs can have deficits of lower extremities or bowel/bladder. Treatment for encephaloceles and myelomeningoceles is surgical closure, with repair of the defect. A less morbid form, meningocele, involves protrusion of only meninges (and no nervous tissues) treatment is surgical repair as well. Interestingly, myelomeningoceles are almost always associated with Chiari II malformation (see next section).

Chiari Malformations: Chiari I malformation, common in incidence throughout life is a herniation of the cerebellar tonsils out of a small posterior fossa into the spinal canal; seeing the cerebellar tonsils descending below the foramen magnum on an MRI scan does not mean that patient automatically needs an operation, especially if the patient is asymptomatic and does not have a syrinx (CSF-filled central canal dilatation of the spinal cord). However, classic symptoms associated with a Chiari malformation are occipital headaches, worse with Valsalva, and possibly symptoms from the associated syrinx which may result from disruption of CSF flow (with numbness to pinprick and not to fine touch in a cape-like distribution classically attributed to syrinx if present with sensation normal distal to the syrinx). Treatment of the Chiari I malformation involves occipital craniectomy and cervical laminectomy with duroplasty, to “make more room for the tonsils and CSF flow”; this usually causes the syrinx to subside over several months. Chiari II malformations involve descent of the cerebellar vermis, and have other anatomical abnormalities (beaked tectum, hydrocephalus, bony abnormalities, etc.) associated with them. Treatment is usually placement of a ventriculoperitoneal shunt,
and if symptoms are present in the face of a functional shunt, a Chiari decompression (as for Chiari I) is performed.

Cerebral palsy: Not much can be done to reverse the supposed hypoxic insult during delivery resulting in cerebral palsy; however, these patients are more likely to have disabling dystonia. Operating for this include dorsal rhizotomy and placement of an intrathecal baclofen pump.

Craniosynostosis: Premature fusion of the sutures of the skull (sagittal most common) results in a misshapen head and craniosynostosis. Appearance depends upon which suture is involved, and can be associated with mental retardation. Treatment is surgical correction with cranial reconstruction (sometimes in conjunction with Plastic Surgery which helps with the facial features). Results can be cosmetically quite good.

X. Functional and Stereotactic

Movement disorders, pain, and biopsy are treated by this subspecialty.

Movement disorders such as essential tremor can be treated by stimulating (versus lesioning which is irreversible) a region (VL nucleus) of the thalamus; similarly, a region in the medial globus pallidum can help control rigidity and dyskinesia; most recently, a subthalamic nucleus stimulator placement can help control tremor, dyskinesia, and rigidity associated with Parkinson’s disease.

Pain is treated by lesioning at various levels of the neuraxis (e.g. dorsal root entry zone lesioning, cingulate gyrus lesion placement, motor cortex stimulation, etc.); results are variable. The pain of trigeminal neuralgia, which is a fleeting, lancinating type of pain which lasts just seconds at times, is felt more often in the V2 and V3 distributions. It has been attributed to “short-circuit cross-talk” or ephaptic signal transmission within the trigeminal nerve; in many cases it is due to compression of a blood vessel (usually a branch of the superior cerebellar artery (SCA)). Neurosurgery generally offers three types of treatments for this problem following failure on a medication (usually carbamazepine, dilantin, and/or baclofen): percutaneous rhizotomy/glycerol injection, microvascular decompression, and open rhizotomy. Success in the short term is on the order of 70-95%, but in some cases the pain can recur. Similar syndromes have been described with hemifacial spasm (from VII n. compression by a branch of the anterior inferior cerebellar artery (AICA) or glossopharyngeal neuralgia (pain with swallowing, which is sometimes associated with a compressing vessel and sometimes not). In cases where a compressing vessel is found, it is gently lifted and cotton-like pads are placed between it and the nerve; in cases where no vessel is found, nerve rootlet sectioning is considered (in trigeminal neuralgia, care must be taken not to lose the corneal reflex; in glossopharyngeal neuralgia it is important to section upper roots of X as well as IX).

XI. Epilepsy
The neurosurgical treatment of epilepsy can be divided into four categories: Temporal lobe epilepsy, extratemporal epilepsy, epilepsy partials continuants, and multifocal.

**Temporal lobectomy:** Patients whose seizures are refractory to medical therapy, whose seizures are localized by electroencephalography (EEG) to the temporal lobe (often medical temporal lobe), and who have evidence of “hippocampal sclerosis” or hippocampal atrophy on MRI scans have a greater than 90% chance of cure with temporal lobectomy; risks are low (< 5% for complications such as visual field defect, memory problems, other surgical risks). This is, by far, the most commonly performed epilepsy-related surgery, and is the most likely to result in a cure.

**Extratemporal epilepsy:** Provided non-eloquent cortex is involved, resection of seizure foci other than temporal lobe can be performed, although the likelihood of cure is not as high. “U” fibers can be interrupted in eloquent cortex regions by a technique called multiple subpial transactions. Also, seizure spread from one hemisphere (where they originate) can be prevented into another hemisphere (which is what is thought to happen in drop attacks), by a technique called corpus callosotomy (where the anterior 2/3 of the corpus calosum is cut – the posterior 1/3 is spared, to avoid a disconnection syndrome which would result in an inability of the left brain to comprehend objects seen on the right occipital cortex).

**Epilepsy partialis continuans:** A continuous partial seizure localized to one hemisphere, which is refractory to all attempted medical therapy, may benefit only from hemispherectomy. While this implies a removal of a hemisphere of the brain, anatomical hemispherectomy is rarely performed; functional hemispherectomy is performed instead, which involves disconnection of the tracts to the other half of the brain. Anatomical hemispherectomy has fallen out of favor due to the risk of subpial hemosiderosis, with associated hydrocephalus (due to the “dead space” left behind); this complication is avoided by functional hemispherectomy.

**Multifocal epilepsy:** Epilepsy from multiple foci or with foci in eloquent areas of the brain can be treated by vagal nerve stimulation. The left vagus nerve is stimulated by an implanted electrode, and while this treatment provides reduction in seizure activity, it rarely leads to cure. Right vagal stimulation is never done, since it is thought to lead to cardiac dysrhythmia.

**XII. Infections**

Osteomyelitis, epidural abscess, brain abscess, meningitis, and mycotic aneurysms.

**Osteomyelitis:** An infection of bone. Treated by iv antibiotics and removal of the bone (which is often soft and discolored). In the spine, bone removal may cause instability, hence necessitating reconstruction with graft and instrumentation.

**Epidural abscess:** Intra-spinally, this represents an emergency if a patient has neurological deficit – MRI with contrast is useful for detection, and patients must undergo urgent surgical evaluation (since venous thrombosis can result without intervention).

**Subdural empyema** - Intra-cranially, subdural empyema represents a neurosurgical emergency. CT with contrast shows a rim-enhancing collection in the subdural space.
Urgent evacuation can help prevent venous thrombosis, which in the end, is the culprit for the permanent neurological deficit by way of venous infarcts if not evacuated in time. **Brain abscess:** An abscess within the brain parenchyma. Modern management suggests aspiration of the abscess (e.g. via stereotactically guided needle or open surgery) for identification of organism. Abscess greater than 2 cm in diameter require open evacuation if possible. Otherwise, iv antibiotics targeting the organism grown from culture of the abscess should be continued until resolution. **Meningitis:** Covered in detail in the “Neurology” portion of course, it is important to remember that the organism varies with the age group, as does treatment per organism in many cases. IV antibiotics usually suffice. **Mycotic aneurysms:** Due to weakening of the wall by bacteria/other infectious agent. Usually differentiated from congenital/developmental aneurysms as these occur on distal branches, and can disappear after iv antibiotic treatment. Since the entire wall is involved, are not amenable to clipping, but if surgery must be performed, bypass or anastomosis can be considered vs. endovascular/open surgical sacrifice of the vessel (if this is a better risk/benefit-dictated option).

HIV/AIDS – diagnosed patients suffer from unique intracranial mass lesions. Three such unique lesions (associated with highest to lowest CD4 count) include toxoplasmosis, lymphoma, and progressive multifocal leukoencephalopathy (PML), respectively. Toxoplasmosis is treated by anti-fungal agents, lymphoma can be treated by chemotherapy (e.g. methotrexate)/radiation in some cases, and PML (which presents as a non-enhancing lesion frequently) does not presently have a good treatment. Toxo titers are good internal indication, but in ambiguous cases a biopsy of the lesion may be necessary to determine which of these entities is actually present.

XIII. **Inflammatory disorders**

These include sarcoidosis, multiple sclerosis, multi \ple sclerosis, tranverse myelopathy, and other. Since the majority will be covered in “Neurology” portion of your rotation, sarcoidosis will be covered in detail here.

Sarcoidosis: An idiopathic inflammatory process involving non-caseating granuloma formation. Sometimes seen in the brain (e.g. sellar region) and spinal cord parenchyma, it can be systemic or isolated to the nervous system (neurosarcoid). CSF ACE levels, if high, suggest sarcoidosis; if normal, sarcoidosis cannot be ruled out. Treatment is usually steroids or other immunosuppressive therapy (e.g. methotrexate, etc.). Differentiating inflammatory disorders from tumor (especially in the spinal cord) can be difficult. Some studies suggest that actual expansion of the cord, as measured from an MRI, show that the cord diameter is not increased in inflammatory disorders, whereas it is increased in many cases of a tumor.

XIV. **Degenerative disease**
Dementia and other chronic degenerated diseases of the brain should be covered in the “Neurology” rotation; spine degenerative process (herniated disc, spinal stenosis) and peripheral nerve-related degenerative processes (carpal tunnel syndrome, ulnar neuropathy) will be covered here.

Herniated spinal disc (herniated nucleus pulposus, or HNP) results from degeneration of the annulus fibrosus of the intervertebral discs. This causes herniation of the inner nucleus pulposus, which can compress a nerve root and cause symptoms (radiculopathy). Symptoms and signs associated with a radiculopathy include pain, numbness, and weakness along the region supplied by the nerve (for example, a C5 radiculopathy can result in deltoid weakness, and an S1 radiculopathy can result in numbness along the plantar surface of the foot with weakness of ankle plantar flexion). Alternatively, a disc can cause spinal cord compression (myelopathy). This results in hyperreflexia muscle groups innervated below the level of compression (e.g. with a C-6 central disc, hyperreflexia of the triceps and brachioradialis may be seen); other non-specific signs of myelopathy are a positive Hoffman’s sign (flicking the middle finger on a relaxed hand results in movement of the index finger/thumb) and a positive Babinski sign (stoking the bottom of the foot yields an upgoing toe). Myelopathy is considered a sign of “upper motor neuron disease” whereas radiculopathy is considered a sign of “lower motor neuron disease.” Classically, L4-5 and L5-S1 HNP are common causes of lumbosacral radiculopathy. Pain is usually better with standing than sitting, and radiates down a lower extremity; upon a straight leg raising, pain is felt. Treatment options are none, NSAIDs, steroids (p.o. or cortisol injections, which are both controversial), and microdiscectomy. For patients with a disc, radicular pain, and weakness/numbness which are compatible with the specific nerve root appearing compressed, the results of radicular pain relief are better than 90%.

Spinal degenerative processes – Spinal stenosis is causes by facet (usually superior) and ligamentous hypertrophy, with a positive Lhermitte’s sign (shooting pain with flexion or extension of the neck). If lumbar stenosis is present, patients may have neurgenic claudication (fatigue in the leg after walking a predictable distance, which is relieved by stooping over or sitting down), with an otherwise normal neurological exam. Stenosis symptoms (cervical or lumbar) are rarely relieved by medications, and require surgical decompression (usually laminectomy for cervical stenosis and laminectomy with medial facetectomy for lumbar stenosis).

Carpal tunnel syndrome & Ulnar neuropathy – Carpal tunnel syndrome involves compression of the median nerve by the flexor retinaculum; sensory involve pain with lateral (1st/2nd) finger numbness (start counting with the thumb as the 1st finger with thenar atrophy in advanced fingers; treatment is bracing or carpal tunnel releases (which can be done with a small incision order local anesthesia). Ulnar neuropathy involves numbness of medial fingers (3rd-5th), with finger intrinsic (interosseal) weakness; the usual site of compression is at the elbow (olecranon notch). Treatment is surgical decompression/transposition.
XV. Radiation therapy

An important treatment of neurosurgical problems can be radiation therapy. Three different types are recognized: whole brain therapy, focused external beam therapy, and stereotactic radiosurgery (gamma knife and linear accelerator). Whole brain therapy has been used for brain metastases primarily. Focused external beam therapy has been used to deliver local radiation to certain isolated areas of the brain or spine which are not amenable to surgical resection. Both whole brain and focused external beam therapy (to the brain or spine) are delivered by conjunction with radiation oncologist/physicist. Each is considered here.

Gamma Knife uses 201 Cobalt-generated gamma-rays focused to a small region of the brain. The analogy is to a magnifying glass focusing sunlight on paper – if you put you hand between the magnifying glass and paper, it will not be as hot as it is at the point of where the light is focused. Similarly, the idea is to apply maximum radiation to the tumor or AVM (focus) while sparing the surrounding brain, linear accelerators use a different method of generating high-energy particles, but essentially involve the same mechanism as gamma knife.

XVI. Syndromes

Certain neurocutaneous syndromes associated with neurosurgical problems. These include neurofibromatosis (Types I & II), Tuberous Sclerosis, and Sturge-Webber syndromes. Additionally, von Hippel-Lindau complex is noted.

Neurofibromatosis type I is neurocutaneous syndrome associated with chromosome 17, with autosomal dominant and sporadic cases noted. Café-au-lait spots are found, along with other important findings. Of neurosurgical concern, the syndrome is typically associated with schwannomas (myelin sheath tumors off nerves roots, see “Neurooncology section” above) and glial tumors involving the optic pathway. In general, symptomatic schwannomas are resected; optic pathway glomas are left alone unless symptomatic. Neurofibromatosis Type II is associated with chromosome 22 autosomal dominant inheritance, and is more often associated with acoustic neurinoma (sometimes bilateral), neurofibroma, and meningioma as neurosurgical problems.

Tuberous sclerosis is associated with chromosome 9, autosomal dominant and sporadic cases. Ash leaf spots are the cutaneous hallmark. Tubers (hamartomas) and subependymal giant cell astrocytomas can be found in these patients. Surgery is reserved for symptomatic cases.

Sturge-Weber syndrome is a sporadic greater than autosomal recessive (Chromosome 3) inherited disorder; it is associated with a V1-distribution ipsilateral portwine nervus, calcifications, cortical atrophy, and seizures. Treatment is for seizures (medications, then surgery as indicated).

Von Hippel Lindau involves chromosome 3, and is autosomal dominant as well as sporadically inherited. Associated problems include hemangioblastoms (involving the
brain, spinal cord, and eye); additionally, these patients are prone to systemic cancers such as renal cell and pancreatic/other visceral organs. Erythropoetin-like substances released by the tumor cause hematocrit increase. Treatment for hemangioblastomas is surgical resection.