NEUROSURGERY

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INTRACRANIAL DYNAMICS

Intracranial Pressure/Volume Relationship

- ❑ intracranial volume is constant
  - $V_{brain} + V_{blood} + V_{csf} + V_{lesion} = V_{skull}$ = constant (Monro-Kellie hypothesis)
- ❑ as lesion expands, ICP does not rise initially
  - CSF, blood, some brain water displaced out of the head
  - brain tissue may shift into compartments under less pressure (herniation)
- ❑ ICP then rises exponentially

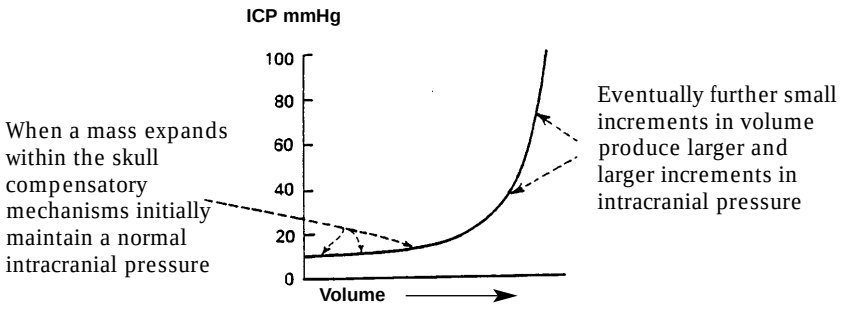

Figure 1. ICP-Volume Curve

Adapted from Lindsay KW, Bone I, Callander, R: Neurology and Neurosurgery Illustrated

- ❑ normal ICP ~ 6-15 mm Hg (80-180 mm H_2O) and varies with patient position

ICP Measurement

- lumbar puncture (contraindicated with known/suspected intracranial mass lesion)
- ventricular catheter (also permits therapeutic drainage of CSF to decrease ICP)
- intraparenchymal monitor
- subdural/subarachnoid monitor (Richmond bolt)

Cerebral Blood Flow (CBF)

- ❑ CBF depends on cerebral perfusion pressure (CPP) and cerebral vascular resistance (CVR)
- ❑ CPP = MAP (mean arterial pressure) – ICP (intracranial pressure)
- ❑ cerebral autoregulation maintains constant CBF by compensating for changes in CPP, unless
  - high ICP such that CPP < 40 mm Hg
  - MAP > 160 mm Hg or MAP < 60 mm Hg
  - brain injury: i.e. subarachnoid hemorrhage (SAH), severe trauma

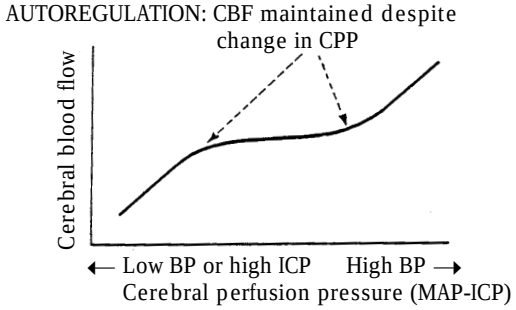

Figure 2. Cerebral Autoregulation Curve

Adapted from Lindsay et al: Neurology and Neurosurgery Illustrated

- ❑ other factors may increase ICP by increasing intracranial blood volume
  - pCO_2
    - CO_2 is a powerful vasodilator
    - CNS pathology —> respiratory compromise —> increased pCO_2 —> increased cerebral vasodilatation —> raised ICP
    - therefore ventilate/hyperventilate —> decreased pCO_2 —> vasoconstrict —> decreased ICP
  - pO_2 (< 60)
    - decreased pO_2 —> vasodilate —>raised ICP
    - therefore prevent hypoxia
  - decreased venous drainage
    - intracranial venous sinuses drain directly into superior vena cava (no intervening valves)
    - lying down, bending over, Valsalva all increase ICP
    - standing, raising head of bed both decrease ICP

HERNIATION SYNDROMES

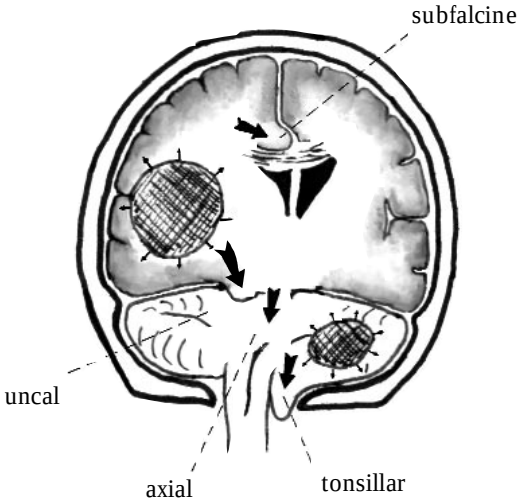

Figure 3. Herniation Types

Subfalcine (Cingulate) Herniation

- ❑ definition: cingulate gyrus herniates under falx
- ❑ cause: supratentorial lateral lesion
- ❑ presentation
  - pathological/radiological observation
  - warns of impending transtentorial herniation

Lateral Tentorial (Uncal) Herniation

- ❑ definition: uncus of temporal lobe herniates down through tentorial notch
- ❑ cause: supratentorial lateral lesion (often rapidly expanding traumatic hematoma)
- ❑ clinical presentation
  - unilateral dilated pupil, followed by extraocular muscle (EOM) paralysis (ipsilateral cranial nerve III (CNIII) compressed)
  - decreased level of consciousness (LOC) (midbrain compressed)
  - "Kernohan's notch": contralateral cerebral peduncle compressed due to shift of brain —> ipsilateral hemiplegia (a false localizing sign)

Central Tentorial (Axial) Herniation

- ❑ definition: displacement of diencephalon and midbrain through tentorial notch
- ❑ cause: supratentorial midline lesion, diffuse cerebral swelling, late uncal herniation
- ❑ clinical presentation
  - decreased LOC (midbrain compressed)
  - EOM/upward gaze impairment ("sunset eyes", pressure on superior colliculus in midbrain compresses 3rd nerve nucleus)

- brainstem hemorrhage (Duret's, secondary to shearing of basilar artery perforating vessels)
- diabetes insipidus (traction on pituitary stalk and hypothalamus) - this is an end stage sign

Tonsillar Herniation ("Coning")
❑ definition: cerebellar tonsils herniate through foramen magnum
❑ cause: infratentorial lesion, or following central tentorial herniation
❑ clinical presentation
- rapidly fatal (compression of cardiovascular and respiratory centers in medulla)
- may be precipitated by lumbar puncture (LP) in presence of space occupying lesion (particularly in the posterior fossa)

CLINICAL FEATURES

Acute Raised ICP
❑ headache
❑ nausea and vomiting (N/V)
❑ decreased LOC
❑ Glasgow Coma Scale (GCS) best index to monitor progress and predict outcome of acute intracranial process (see Neurology Notes)
❑ papilledema
- may take 24-48 hours to develop

❑ CN palsy
- CN III
  - pupillary dilatation
  - unilateral dilated pupil signifies herniation (CN III compressed)
- CN VI
  - longest intracranial course
  - causative mass may be remote from nerve root, i.e. CN VI palsy can be a false localizing sign

❑ Cushing response
- increased blood pressure (BP), decreased pulse

❑ respiratory changes
- e.g. Cheyne Stokes, apneustic, ataxic

❑ localizing neurologic signs may occur
- e.g. contralateral hemiplegia except with Kernohan's notch

❑ paralysis of upward gaze
- especially in children (obstructive hydrocephalus)

Chronic Raised ICP
❑ headache
- postural: worsened by coughing, straining, bending over (Valsalva)
- morning H/A: worse on waking in the morning

❑ visual changes
- enlarged blind spot, preserved vision (until extremely advanced, then episodic constrictions of visual fields, i.e. "gray-outs")
- long standing papilledema (not necessarily present) may produce optic atrophy and blindness

❑ differentiate from papillitis (usually unilateral with decreased visual acuity)

Imaging Features
❑ CT: key diagnostic investigation
- enlarged ventricles - hydrocephalus
- compressed ventricles with midline shift - mass lesion

❑ skull x-rays: (academic), in chronic ICP may show
- separation of sutures in infants
- digital markings in skull vault from compression of brain matter against bone ("copper beating")
- thinning of dorsum sellae

MANAGEMENT
❑ elevate head
- head of bed at 30-45 degrees —> decreases intracranial venous pressure

❑ ventilate/hyperventilate
- decreases pCO_2, increases pO_2, decreases venous pressure

- ❑ mannitol (20% IV solution preferred)
  - can give rapidly, effects in 30 minutes, (see Drugs Section)
- ❑ identify etiology
  - CT, MRI
- ❑ steroids
  - decreases edema around brain tumour
  - no proven value in head injury or stroke
  - works slowly (days)
- ❑ surgery
  - remove mass lesion
  - remove CSF by external ventricular catheter drain (if acute) or shunt
  - Note: lumbar puncture contraindicated when known/suspected intracranial mass lesion

BENIGN INTRACRANIAL HYPERTENSION (PSEUDOTUMOUR CEREBRI)

- ❑ raised intracranial pressure with no evidence of any "mass" lesion, hydrocephalus, infection or hypertensive encephalopathy

Etiology

- ❑ unknown (majority), but associated with
  - diet: obesity, hyper/hypovitaminosis A
  - endocrine: pregnancy, menarche, menstrual irregularities, Addison's disease
  - hematological: iron deficiency anemia, polycythemia vera
  - drug: oral contraceptives, steroid withdrawal, tetracycline, nalidixic acid

Clinical Features

- ❑ usually in 3rd and 4th decade (F>M)
- ❑ symptoms and signs of raised ICP
- ❑ normal radiological (CT or MRI) studies
- ❑ usually self-limited, recurrence is common, chronic in some patients
- ❑ a preventable cause of (often permanent) blindness from optic atrophy
- ❑ risk of blindness is not reliably correlated to duration of symptoms,
- ❑ papilledema, headache, visual acuity or number of recurrences

Differential Diagnoses

- ❑ true mass lesions (see Intracranial Mass Section)
- ❑ venous outflow obstruction to CSF absorption
  - sagittal sinus thrombosis, lateral sinus thrombosis (usually secondary to mastoiditis), jugular vein obstruction
  - following neck operation
  - intrathoracic mass lesion
  - superior vena cava syndrome
  - congestive heart failure
  - hyperviscosity syndromes
- ❑ infections
- ❑ inflammatory conditions: e.g. neurosarcoidosis, SLE
- ❑ vasculitis
- ❑ metabolic conditions: e.g. lead poisoning
- ❑ pseudopapilledema associated with hyperopia and drusen
- ❑ meningeal carcinomatosis
- ❑ Guillain-Barre syndrome
- ❑ following head trauma

Treatment

- ❑ treat underlying cause if known
- ❑ drugs - acetazolamide (↓CSF production)
  - thiazide diuretic
- ❑ if above fail —> lumboperitoneal shunt
- ❑ optic nerve sheath fenestration - if progressive impairment of visual acuity despite treatment

HYDROCEPHALUS (HCP)

Notes

- ❑ definition: increased CSF volume
- ❑ normal CSF volume = 100 - 150 mL (50 in ventricles, 25 around brain, 75 around spinal cord)
- ❑ CSF production is constant at 35 cc/hr (500 - 750 cc/day)

MECHANISMS

- ❑ increased production
  - e.g. choroid plexus papilloma (0.4-1% of intracranial tumours)
- ❑ decreased absorption (see below)

CLASSIFICATION

Non-Communicating (Obstructive) Hydrocephalus

- ❑ absorption is blocked within ventricular system - no escape of CSF into subarachnoid space
- ❑ causes/location of block
  - intraventricular hemorrhage
  - ventricular tumours (e.g. 3rd ventricle colloid cyst)
  - supratentorial mass causing tentorial herniation and aqueduct compression
  - infratentorial mass causing 4th ventricle obstruction or aqueduct kinking
  - congenital e.g. aqueductal stenosis (see Pediatric Neurosurgery Section)
- ❑ CT findings
  - lateral and 3rd ventricles dilated
  - normal 4th ventricle (e.g. aqueduct stenosis) or deviated/absent 4th ventricle (e.g. posterior fossa mass)

Communicating (Non-Obstructive) Hydrocephalus

- ❑ absorption is blocked at some part of extraventricular pathway, such as arachnoid granulations
- ❑ causes
  - meningitis
  - SAH
  - trauma
- ❑ CT findings
  - all ventricles dilated

Normal Pressure Hydrocephalus (NPH)

- ❑ gradual onset of classic triad
  - gait apraxia
  - incontinence
  - dementia
- ❑ CSF pressure often within clinically “normal” range
- ❑ usually communicating

Hydrocephalus Ex Vacuo

- ❑ enlargement of ventricles (and sulci) secondary to diffuse brain atrophy
- ❑ usually a function of normal aging
- ❑ not true hydrocephalus

CLINICAL FEATURES

Acute HCP

- ❑ signs and symptoms of acute raised ICP
- ❑ usually non-communicating type

Chronic HCP

- ❑ similar to NPH

INVESTIGATIONS

CT

- ❑ ventricular enlargement, may see prominent temporal horns
- ❑ periventricular lucency (CSF forced into extracellular space)
- ❑ narrow/absent sulci, +/– 4th ventricular enlargement

Ultrasound (through anterior fontanelle in infants)
- ❑ ventricular enlargement

MANAGEMENT
- ❑ spinal taps (for transient, communicating HCP)
- ❑ remove obstruction (if possible)
- ❑ choroid plexectomy (for choroid plexus papilloma)
- ❑ third ventriculostomy (for obstructive HCP)
- ❑ shunts
    - ventriculoperitoneal (VP) = ventricle to peritoneum
    - ventriculo-atrial (VA) = ventricle to right atrium
    - lumboperitoneal = lumbar spine to peritoneum (for communicating HCP)

Shunt Complications
- ❑ obstruction
    - etiology: infection, obstruction by choroid plexus, buildup of proteinaceous accretions, blood, cells (inflammatory or tumour)
    - signs and symptoms of acute HCP or increased ICP
    - radiographic evaluation: "shunt series" (plain x-rays which only show disconnection of tube system), CT
- ❑ infection (3-4%)
    - etiology: *S. epidermidis, S. aureus,* gram-negative bacilli
    - presentation: fever, nausea and vomiting, anorexia, irritability; signs and symptoms of shunt obstruction; shunt nephritis (antibodies generated against bacteria in shunt leads to kidney damage)
    - investigation: CBC, blood culture, shunt tap (LP usually NOT recommended)
- ❑ overshunting
    - slit ventricle syndrome (collapse of ventricles leading to shunt catheter occlusion by ependymal lining)
    - subdural effusion
    - secondary craniosynostosis (children)
    - low pressure headache
- ❑ seizures
- ❑ problems related to distal catheter (blockage)

INTRACRANIAL MASS

- ❑ differential diagnosis: "tumour, pus or blood"
- ❑ history important for localizing and differentiating mass lesions
- ❑ important features on CT (with and without contrast enhancement)
    - lesions (may be isodense without contrast)
    - midline shifts and herniations
    - effacement of ventricles and sulci (often ipsilateral)

TUMOUR
- ❑ primary versus metastatic
- ❑ primary tumours (benign or malignant) rarely metastasize
- ❑ presenting symptoms
    - local effects
        - dependent on site: focal deficits, lobe syndromes, seizures
    - raised ICP
        - acute or chronic depending on tumour growth rate (see Raised ICP Section)
    - sudden onset of symptoms after hemorrhage (5-10%)
- ❑ consider by
    - location (supratentorial vs. infratentorial)
    - age (adult vs. child)

Table 1. Tumour Types: Age, Location and Clinical Features

	Supratentorial	Infratentorial
children (< 15 years, primarily infratentorial - 80%)	• astrocytoma - all grades e.g. optic nerve astrocytoma • craniopharyngioma • ependymoma • other: dermoid/epidermoid, pineal tumours, primitive neuroectodermal tumors	• cerebellar astrocytoma • medulloblastoma • ependymoma • choroid plexus papilloma • brain stem astrocytoma
adult (> 15 years, primarily supratentorial - 80%)	• astrocytoma (40-50%) • metastatic (20-30%) • meningioma (15%) • pituitary ademona (5%) • oligodendroglioma (5%)	• metastatic (20-30%) • schwannoma (6%) e.g. acoustic neuroma • hemangioblastoma • medulloblastoma (5%)
signs and symptoms	raised ICP focal or lobar effects • seizures • mental status changes • personality changes • visual field deficits • endocrine disturbances (with pituitary tumour)	raised ICP local effects in posterior fossa • extremity ataxia • truncal ataxia • CN palsy - often multiple • nystagmus • LOC • long tract signs

Investigations
- ❑ CT, MRI, stereotactic biopsy (tissue diagnosis)

Management
- ❑ medical
  - steroids useful for vasogenic cerebral edema (decrease edema around tumours —> decrease mass effect/ICP)
  - pharmacological treatment for pituitary tumours (see Pituitary Adenoma Section)
- ❑ surgical
  - excisional: total, partial, decompressive, palliative
  - shunt if CSF flow is blocked
- ❑ radiotherapy - external, brachytherapy, stereotactic radiosurgery (Gamma-knife, Linear Accelerator)
- ❑ chemotherapy - alkylating agents

Metastatic Tumours
- ❑ mainly from lung, breast, GI, kidney, melanoma
- ❑ solitary tumour: surgical excision and whole brain radiation

Astrocytoma
- ❑ most common primary brain tumour (45-50%)
- ❑ low grade (grades I-II)
  - slower growing, peak age 40 years, median survival 2-4 years
- ❑ high grade (grades III-IV, glioblastoma multiforme)
  - fast growing, peak age 55 years, median survival < 2 years
- ❑ “cystic cerebellar” astrocytoma
  - pediatric population, infratentorial
  - potentially curable
- ❑ presentation: middle aged, recent onset of new, worsening H/A, with vomiting +/– other symptoms
- ❑ diagnosis: CT (see Figure 4), MRI with contrast +/– biopsy
- ❑ surgery not curative, aim to prolong “quality” life
- ❑ radiotherapy prolongs survival

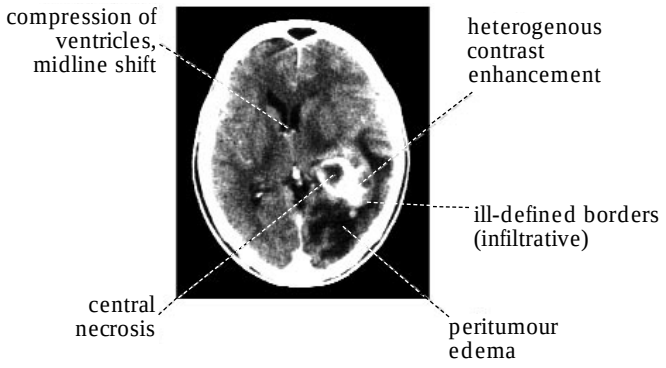

Figure 4. Malignant Astrocytoma on CT

Meningioma

- ❑ mostly benign (1% malignant), slow-growing, non-infiltrative
- ❑ common locations: parasagittal and falx convexity, sphenoid ridge
- ❑ curable if complete resection possible (5 year survival > 90%)
- ❑ presentation: middle aged, symptoms of increased ICP, focal symptoms depend on location
- ❑ diagnosis: MRI, CT with contrast (see Figure 5)

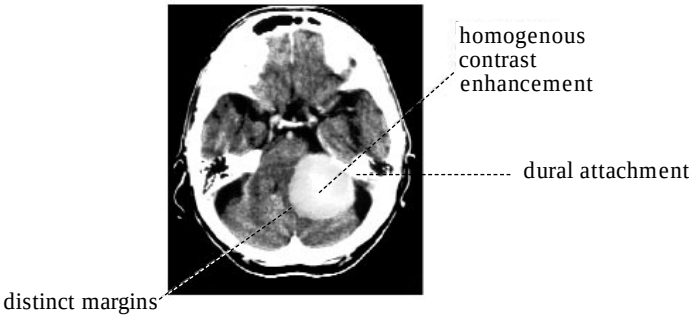

Figure 5. Meningioma on CT

Vestibular Schwannoma ("Acoustic Neuroma")

- ❑ progressive unilateral deafness = acoustic neuroma until proven otherwise
- ❑ arises from vestibular component of CN VIII at cerebello-pontine angle (CPA)
- ❑ presentation: compression of structures in CPA
  - CN V: facial numbness, loss of corneal reflex
  - CN VII: facial weakness (uncommon pre-operatively)
  - CN VIII: unilateral sensorineural deafness, tinnitus, vertigo
  - cerebellum: ataxia, nystagmus
- ❑ diagnosis
  - MRI, CT (contrast enhancing mass in CPA)
  - audiogram, caloric tests
  - if bilateral: neurofibromatosis type II
- ❑ management
  - curable by resection
  - palliative treatment: gamma-knife radiotherapy

Pituitary Adenomas

- ❑ mass effects
  - H/A
  - bitemporal hemianopsia (compression of optic chiasm)
  - CN III, IV, V1, V2, VI palsy (compression of cavernous sinus)
- ❑ endocrine effects
  - hyperprolactinemia
  - Cushing disease
  - acromegaly
  - infertility, amenorrhea, galactorrhea, impotence
  - panhypopituitarism (hypothyroidism)

- ❑ apoplexy and CSF rhinorrhea (rare presenting signs of pituitary tumour)
- ❑ diagnosis: prolactin levels, endocrine function tests; MRI
- ❑ differential: parasellar tumours (e.g. craniopharyngioma, suprasellar (tuberculum sellae meningioma), carotid aneurysm
- ❑ management
  - bromocriptine/dopamine agonists for prolactinoma
  - endocrine replacement therapy
  - somatostatin analogue (octreotide) +/– bromocriptine for acromegaly
  - surgery (+/– radiation)

PUS

Brain Abscess

- ❑ etiology
  - local spread (adjacent infection)
    - otitis media, mastoiditis, sinusitis
    - osteomyelitis
    - dental abscess
  - hematogenous spread
    - adults: lung abscess, bronchiectasis, empyema
    - children: cyanotic heart disease with R to L shunt (blood is shunted away from lungs preventing filtration of bacteria)
    - immunosuppression (AIDS - toxoplasmosis)
  - dural disruption
    - surgery, trauma
    - congenital defect, e.g. dermal sinus
  - pathogens
    - *Streptococci* (most common), often anaerobic or microaerophillic
    - *Staphylococci* (penetrating injury)
    - Gram negatives, anaerobes
- ❑ diagnosis
  - focal neurological signs and symptoms
  - mass effect, increased ICP and sequelae
  - seizures
  - +/– signs of systemic infection (mild fever, leukocytosis)
  - blood cultures rarely helpful, LP not helpful and contraindicated
  - CT scan (see Figure 6)
- ❑ management
  - multiple aspiration of abscess and/or excision, and send for C&S
  - antibiotics
    - empirically: penicillin and metronidazole (cover Streptococci and anaerobes) +/– ceftriaxone (cover Gram negatives)
    - after sensitivity results return, revise antibiotics
  - treat primary site

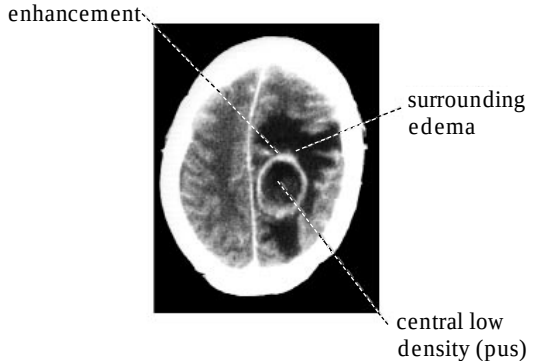

Figure 6. Brain Abscess on CT

Other Causes of Pus...

- ❑ subdural empyema (from sinusitis, mastoiditis - rare, 20% mortality)
- ❑ meningitis, encephalitis, AIDS toxoplasmosis (see Neurology Notes)
- ❑ osteomyelitis of skull (Pott's puffy tumour), usually seen with sinusitis
- ❑ granuloma (TB, sarcoid)

BLOOD

Hematoma/hemorrhage
- ❑ epidural, subdural hematoma (see Trauma Section)
- ❑ intracerebral, intraventricular hemorrhage, SAH (see Cerebrovascular Disease Section)

Vascular Abnormality
- ❑ aneurysm, AVM (see Cerebrovascular Disease Section)

CEREBROVASCULAR DISEASE

- ❑ ischemic cerebral infarction (80%)
  - embolic (heart, carotid artery, aorta) or thrombosis of intracerebral arteries (see Neurology Notes)
- ❑ intracranial hemorrhage (20%)
  - subarachnoid hemorrhage (SAH), spontaneous intracerebral hemorrhage (ICH), intraventricular hemorrhage (IVH)

SUBARACHNOID HEMORRHAGE (SAH)

Etiology
- ❑ trauma (most common)
- ❑ spontaneous
  - aneurysms (75-80%)
  - idiopathic (14-22%)
  - AVMs (5%)
  - infections e.g. mycotic aneurysms

Risk Factors
- ❑ pregnancy/parturition in patients with pre-existing AVMs
- ❑ sympathomimetic (e.g. cocaine) abuse

Clinical Features
- ❑ sudden onset severe headache: “worst headache of my life”
- ❑ vomiting, nausea (increased ICP)
- ❑ meningismus (neck stiffness, photophobia, positive Kernig’s and Brudzinski’s sign)
- ❑ decreased level of consciousness
  - transient or prolonged
- ❑ sentinel/warning leaks
  - small SAH with sudden severe H/A +/– transient focal neurological deficit
  - blood on CT or LP
  - 30-60% of patients with full blown SAH give history suggestive of a warning leak
- ❑ focal deficits: cranial nerve palsy (e.g. III, IV), hemiparesis
- ❑ ocular hemorrhage in 11-33% (due to sudden increase in ICP)
- ❑ occasionally exertional (straining, intercourse)

Clinical Course/Natural History
- ❑ 10-15% die before reaching hospital
- ❑ overall mortality 50-60% in first 30 days
- ❑ major cause of mortality is rebleeding
  - risk of rebleeding: 4% on first day, 15-20% within 2 weeks, 30% by 6 months
  - if no rebleed by 6 months chance of rebleeding decreases to same incidence of unruptured aneurysm (2%)

Diagnosis (see Figure 7)
- ❑ differential diagnosis: migraine, tension H/A, meningitis, stroke, flu
- ❑ CT without contrast (see Figure 8)
  - 90% sensitivity, 100% specificity
  - may be negative if small bleed or presentation delayed several days
  - positive history for SAH with negative CT - MUST do an LP
  - HCP, IVH, ICH, infarct or large aneurysm may be present

- ❑ Lumbar puncture (LP)
  - contraindications
    - known or suspected intracranial mass
    - non-communicating (obstructive) HCP
    - unconscious, focal deficit, papilledema
    - coagulopathy (platelets < 50, anticoagulants, etc...)
    - infection at site desired for LP (e.g. epidural abscess)
  - CSF colour: bloody initially —> xanthochromic supernatant (yellow) by 12-48 hours
  - high sensitivity
  - traumatic tap (false positive): if bloody MUST centrifuge and observe the supernatant, clear supernatant means traumatic tap and xanthochromia means SAH
- ❑ Cerebral angiography
  - demonstrates source of SAH in 80-85% of cases

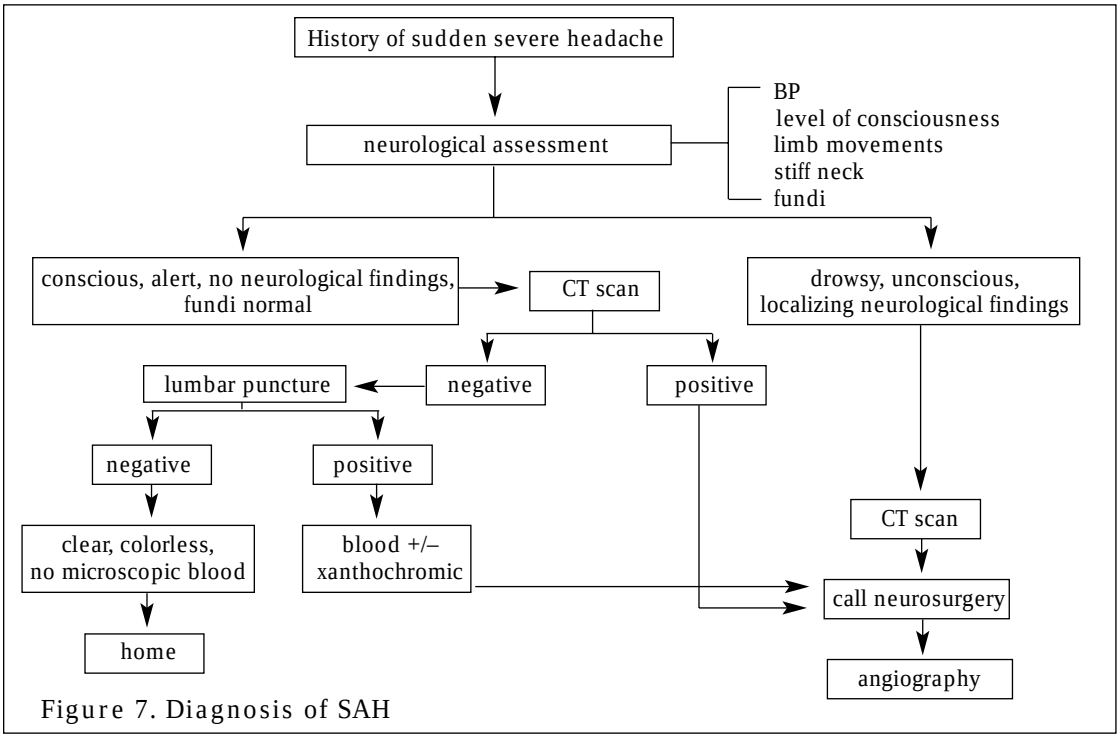

Figure 7. Diagnosis of SAH

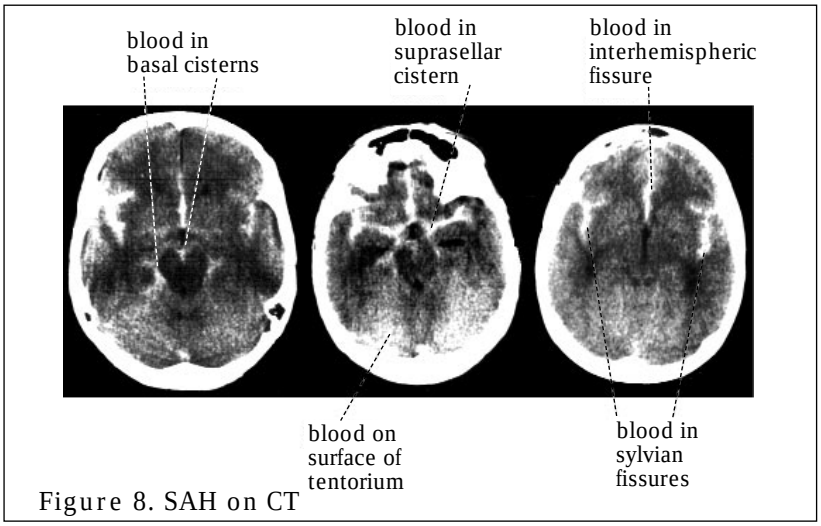

Figure 8. SAH on CT

Complications
- ❑ vasospasm
  - constriction of blood vessels in response to blood outside vessels in the subarachnoid space
  - confusion, decreased LOC, focal neurodeficit (speech or motor)
  - detect with angiogram (decreased vessel caliber) or transcranial doppler (increased blood velocity)
  - radiographic evidence seen in 30-70% of arteriograms performed 7 days following SAH (peak incidence)
  - symptomatic only in 20-30% of patients with SAH
  - onset: 4-14 days post SAH (if patient deteriorates within first 3 days, MUST look for another cause)
  - can produce permanent infarcts and death
  - a major cause of morbidity and mortality
- ❑ hydrocephalus (30%)
  - can be acute or chronic - requiring shunt or drain
- ❑ neurogenic pulmonary edema
- ❑ hyponatremia (SIADH, cerebral salt wasting)
- ❑ diabetes insipidus
- ❑ cardiac - arrhythmia, MI, CHF

Management
- ❑ bed rest, elevate head (30 degrees), minimal external stimulation
- ❑ control HTN, avoid hypotension since CBF autoregulation impaired by SAH
- ❑ prophylactic anticonvulsant: short course of Dilantin (2 weeks)
- ❑ neuroprotective agent: nimodipine
- ❑ early surgery to prevent rebleed
- ❑ intraventricular catheter if acute HCP present
- ❑ "Triple H" therapy for vasospasm: hypertension, hypervolemia, hemodilution
- ❑ angioplasty for refractory vasospasm

SPONTANEOUS INTRACEREBRAL HEMORRHAGE (ICH)

Definition
- ❑ bleeding into brain parenchyma without accompanying trauma
- ❑ can dissect into ventricular system (IVH) or through cortical surface (SAH)

Etiology - usually determines location
- ❑ hypertension (40-60%) - deep gray matter/basal ganglia, thalamus, pons, cerebellum
- ❑ aneurysm (20%)
  - berry
  - mycotic
- ❑ AVMs (5-7%) - anywhere
- ❑ coagulopathies (5-7%) - cortical and subcortical, especially cerebellum
- ❑ tumours (1%) - anywhere e.g. malignant melanoma, choriocarcinoma
- ❑ hemorrhagic infarcts - cortical and subcortical
- ❑ cavernous malformations - anywhere
- ❑ cerebral amyloid (congophilic) angiopathy - subcortical lobar (usually elderly patients)
- ❑ drugs (amphetamines, cocaine, etc...)

Clinical Features
- ❑ 30 day mortality rate is 44%, mostly due to cerebral herniation
- ❑ specific symptoms depend on location of ICH
  - basal ganglia, white matter/internal capsule, thalamus (70%)
    - rapidly progressive neurological deficit: hemiplegia, hemisensory loss, homonymous hemianopsia
    - progressively decreased LOC (versus preserved consciousness in hemispheric ischemic stroke)
    - mass effects (transtentorial herniation)
    - often headache at onset
  - cerebellum
    - sudden severe vertigo and vomiting
    - ataxia, nystagmus, dysmetria, incoordination
    - preserved consciousness until late then sudden death, "talk 'til death"

- mass effect (tonsillar herniation) —> surgical emergency
- headache (occipital)

- pons
  - quadriplegia
  - sudden decreased LOC
  - "pinpoint pontine pupils", disconjugate extraocular movements
  - respiratory abnormalities
  - rapid death
- cortical
  - focal neurological deficits
  - seizures

Diagnosis
- ❑ high density blood on CT without contrast
- ❑ MRI does not show blood immediately - not procedure of choice

Management
- ❑ medical
  - correct HTN, coagulopathy
  - control ICP (mannitol, hyperventilate, elevate head of bed)
  - anticonvulsants
- ❑ surgical
  - craniotomy with evacuation of clot under direct vision, resection of source of ICH (i.e. AVM, tumour, cavernoma), ventriculostomy to treat HCP
  - indications
    - symptomatic
    - marked mass effect, raised ICP - evacuate clot, decompress
    - rapid deterioration (especially with signs of brainstem compression)
    - favorable location, e.g. cerebellar
    - young patient (< 50)
    - if tumour, AVM, aneurysm, or cavernoma suspected (resection or clip to decrease risk of rebleed)
  - contraindications
    - small bleed: minimal symptoms, high GCS (not necessary)
    - massive hemorrhage (especially dominant lobe), low GCS/coma, brainstem lost (poor prognosis)
    - medical reasons, e.g. very elderly, severe coagulopathy, difficult location, e.g. basal ganglia, thalamus (poor surgical candidate)

INTRACRANIAL ANEURYSMS

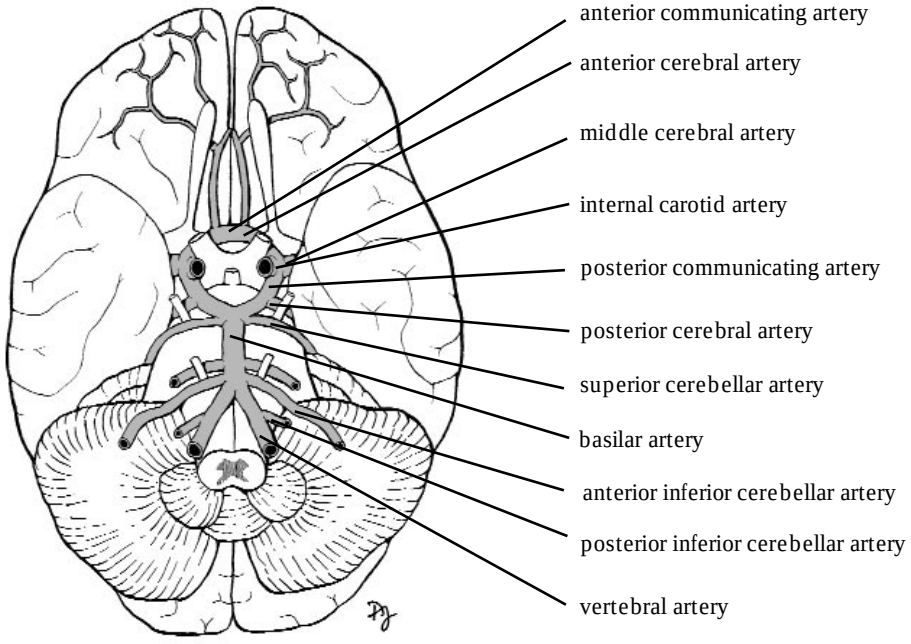

Figure 9. Circle of Willis

Drawing by Andree Jenks

Epidemiology
- ❑ prevalence of 5%
- ❑ female > male
- ❑ 20% multiple aneurysms
- ❑ age 35-65 years

Types
- ❑ saccular (berry)
  - most common type of aneurysm
  - located at branch points of major cerebral arteries (Circle of Willis)
  - common locations: anterior communicating artery/anterior cerebral artery (30%), posterior communicating artery (25%), middle cerebral artery (20%)
- ❑ fusiform
  - atherosclerotic
  - more common in vertebrobasilar system
  - rarely rupture
- ❑ mycotic
  - secondary to vessel wall infection (e.g. SBE)

Clinical Presentation
- ❑ rupture (SAH, ICH, IVH, subdural blood)
- ❑ mass effect (giant aneurysms)
- ❑ small infarcts due to distal embolization
- ❑ seizures
- ❑ headache without hemorrhage
- ❑ incidental CT or angiography finding (asymptomatic)

Management
- ❑ imaging: CT, angiogram
- ❑ ruptured aneurysms
  - initial management of SAH/ICH
  - overall trend towards better outcome with early surgery
  - surgical clipping is the optimal treatment
  - other treatment options: trapping, thrombosing (endovascular technique), balloon embolisation, wrapping, proximal ligation
- ❑ unruptured aneurysms
  - 1-3% annual risk of rupture
  - risk dependent on size of aneurysm
  - no clear evidence on when to operate
  - consider operating at 10 mm by angiography – risk of rupture 41%
  - need to weigh life expectancy risk of hemorrhage and mortality/morbidity of SAH vs. that of aneurysm surgery (age, medical risk, etc...)
  - follow smaller aneurysms with serial angiography

VASCULAR MALFORMATIONS OF THE NERVOUS SYSTEM

Types
- ❑ arteriovenous malformations (AVMs)
- ❑ cavernous malformations (cavernoma, cavernous hemangioma, angiographically occult vascular malformation)
- ❑ venous malformations
- ❑ capillary telangiectasias

Clinical Significance
- ❑ principally AVMs and cavernous malformations produce intracranial hemorrhages and seizure

ARTERIOVENOUS MALFORMATIONS (AVMs)

Etiology
- ❑ tangle of abnormal vessels, arteriovenous shunts
- ❑ congenital, tends to enlarge with age
- ❑ male:female = 2:1
- ❑ present in younger age group than aneurysms (peak age 10-30)

Presentation
- ❑ ICH (40-60%)
  - risk of major bleed: 4% per year
  - 10% mortality (versus 50-60% for aneurysmal SAH) per bleed
  - 50% morbidity (serious neurological deficit) per bleed

- ❑ seizures (50%)
- ❑ mass effect (e.g. Tic Douloureux 2º to CPA AVM)
- ❑ focal neurological signs secondary to ischemia (high flow —> "steal phenomena")
- ❑ localized headache (infrequent; resembles migraine)
- ❑ bruit (especially with dural AVMs)
- ❑ may be silent

Diagnosis
- ❑ MRI (flow void)
- ❑ angiography

Management
- ❑ decreases risk of future hemorrhage and seizure
  - surgical excision
  - endovascular embolisation (glue, balloon)
  - stereotactic radiotherapy (for small AVMs)
- ❑ conservative (seizure control if necessary)

CAVERNOUS MALFORMATIONS
- ❑ venous malformation
- ❑ symptoms: H/A, seizure, neurological deficit, ICH
- ❑ prevalence: 0.3-0.5%
- ❑ hemorrhage risk may be up to 3.6% per year
- ❑ bleeding less severe than from AVM or aneurysm
- ❑ posterior fossa cavernous malformations have much worse clinical picture
- ❑ diagnosis: MRI or contrast enhanced CT
- ❑ treatment: surgical excision

SPINE

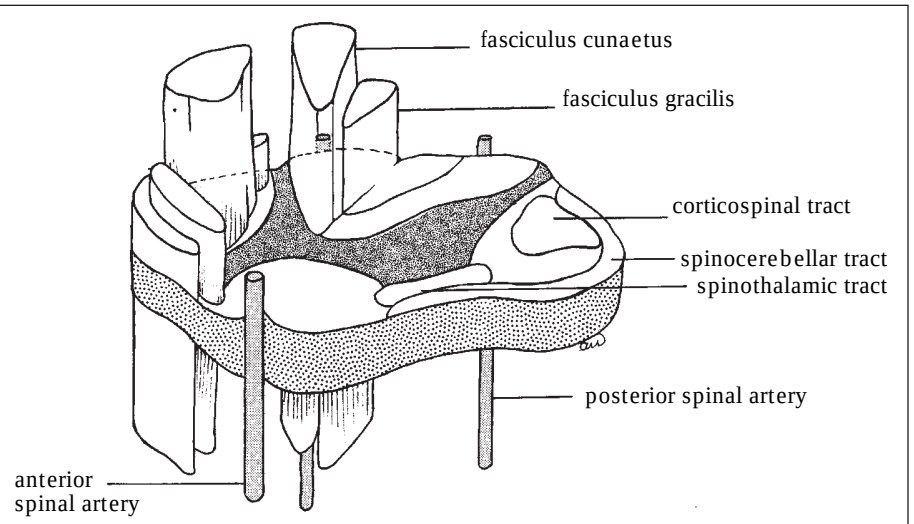

Figure 10. Anatomy of the Spinal Cord

Figure drawn by Aimée Warrell

CORD AND ROOT COMPRESSION

Etiology
- ❑ herniated nucleus pulposus of disc
- ❑ spondylosis or spinal stenosis
  - degenerative process of the spine, may result in stenosis of spinal canal or spinal foramen
- ❑ spondylolisthesis
  - anterior subluxation of one vertebral body on another
- ❑ abscess

- ❑ AVM (rare)
- ❑ tumours
    - extradural (lymphoma or metastases from prostate, lung, breast, kidney)
    - intradural
        - extramedullary (schwannoma, meningioma)
        - intramedullary (ependymoma, astrocytoma, hemangioblastoma)

Clinical Features

- ❑ local pain at site of lesion
- ❑ pain in nerve root distribution, "radiculopathy" (see Figure 10)
- ❑ nerve root dysfunction (sensory loss, lower motor neuron findings)
- ❑ varying degrees of weakness or sensory loss at or below the lesion
    - partial lateral compression (see Spinal Cord Syndromes Section)
    - complete compression
        - bilateral decreased sensation (all modalities) and upper motor neuron lesion findings, bladder retention/incontinence
- ❑ sphincter disturbance (bowel and bladder)

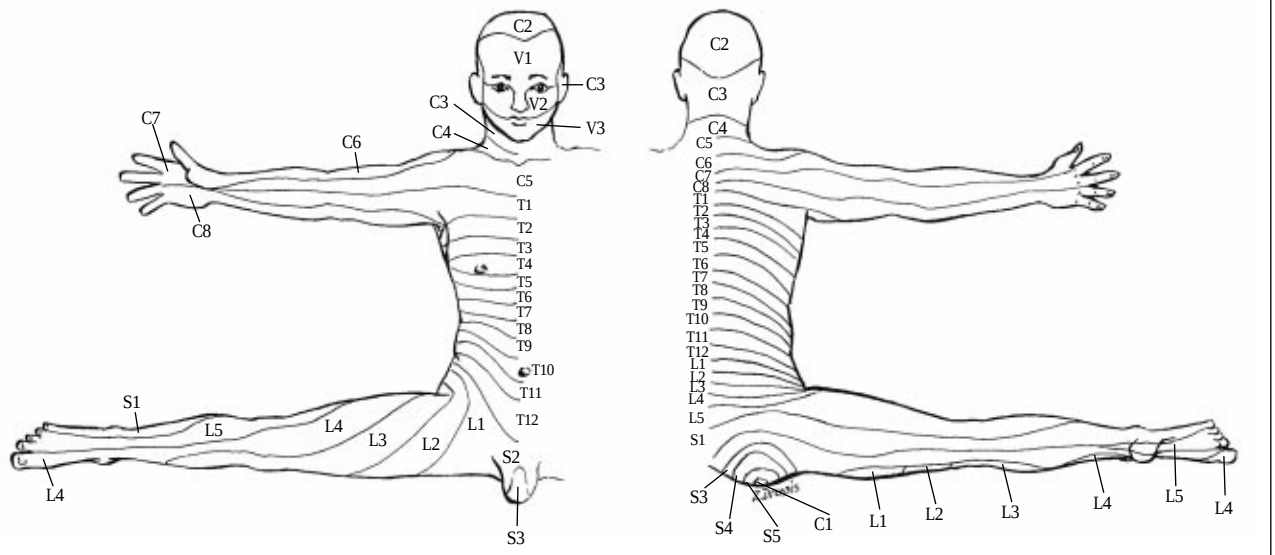

Figure 11. Sensory/Dermatomal Distribution

Drawing by Roula Drossis

Investigations

- ❑ plain x-ray of spine
- ❑ myelogram
- ❑ CT, MRI
- ❑ electromyography (EMG), nerve conduction studies

Management

- ❑ for disc herniation see Lumbar Disc Syndrome
- ❑ radiotherapy (primary/adjuvant therapy for AVM/tumour, palliative for tumour)

SPINAL CORD SYNDROMES (see Neurology Notes)

Brown-Sequard's Syndrome (Hemicord)

- ❑ causes
    - penetrating trauma
    - extrinsic compression
- ❑ clinical features
    - contralateral pain and temperature sensory deficits (deficits are 1 to 2 levels below injury)
    - ipsilateral position/light touch sensory deficits
    - light touch preserved
    - ipsilateral weakness (UMN lesion)
- ❑ best prognosis of cord injuries (90% independently ambulate and have good sphincter control)

Central Cord Syndrome
- ❑ most common incomplete spinal cord injury syndrome
- ❑ cause: spinal flexion-extension injury
- ❑ clinical features
  - dissociated sensory loss
    - "vest" or bilateral suspended pain and temperature deficit with sacral sparing
    - spared touch, joint position and vibration sensation
  - weakness upper (LMN lesion) > lower (UMN lesion) extremities
  - sphincter dysfunction (usually urinary retention)
- ❑ 50% recover enough LE function to ambulate

Anterior Cord Syndrome
- ❑ causes
  - anterior cord obstruction and compression
- ❑ clinical features
  - dissociated sensory loss
    - bilateral pain and temperature deficit
    - spared touch, joint position and vibration sensation
  - bilateral paraplegia (UMN below and LMN at level of the lesion)
  - sphincter dysfunction
- ❑ worst prognosis, only 10-20% recover functional motor control

Posterior Cord Syndrome
- ❑ causes
  - trauma
  - posterior spinal artery infarct
- ❑ clinical features
  - joint position and vibration sensation loss
  - pain and paresthesias in neck, back, or trunk
  - mild paresis of upper extremities

SYRINGOMYELIA
- ❑ "syrinx", cavitation of spinal cord substance

Etiology
- ❑ idiopathic
- ❑ post traumatic
- ❑ associated with
  - craniovertebral anomalies (congenital) e.g. Arnold-Chiari
  - intramedullary tumours
  - arachnoiditis (traumatic)

Presentation
- ❑ suspended, dissociated sensory loss
  - pain and temperature loss in a cape-like distribution
  - preserved light touch and other modalities
- ❑ wasted hand muscles
- ❑ may have spastic weakness of legs
- ❑ may have hydrocephalus, often asymptomatic

Investigations
- ❑ MRI is best method
- ❑ myelogram with delayed CT

Management
- ❑ conservative if NOT progressing
- ❑ shunt (syringosubarachnoid or syringopleural)
- ❑ if associated with Arnold-Chiari malformation
  - first decompress posterior fossa, if not successful then shunt

CERVICAL DISC SYNDROME

Etiology
- ❑ most common levels
  - C5-6 (C6 root), C6-7 (C7 root)
- ❑ less common, but important with respect to activities of daily living
  - C4-5 (C5 root), C7-T1 (C8 root)

Clinical Features
- ❑ lateral disc protrusion compresses nerve root
  - pain down arm in nerve root distribution, worse with neck extension
  - referred parascapular pain
  - +/– nerve conduction velocity abnormalities
- ❑ central cervical disc protrusion compresses spinal cord as well as nerve roots

Table 2. Lateral Cervical Disc Syndrome

	C4-5	C5-6	C6-7	C7-T1
root involved	C5	C6	C7	C8
motor	deltoid supraspinatus biceps	biceps	triceps	digital flexors intrinsics
reflex	supinator	biceps	triceps	finger jerk
sensory	shoulder,	thumb	middle finger	ring finger, little finger

Differential Diagnosis
- ❑ shoulder lesion
- ❑ thoracic outlet syndrome (including Pancoast tumour)
- ❑ cervical spine tumour
- ❑ peripheral nerve lesion (e.g. carpal tunnel)
- ❑ acute brachial neuritis

Investigations
- ❑ C-spine x-ray
- ❑ CT, MRI
- ❑ EMG, nerve conduction studies

Management
- ❑ conservative
  - NSAIDs, collar, traction may help
  - most patients get better spontaneously in 4 to 8 weeks
- ❑ surgical indications
  - intractable pain despite adequate conservative treatment for > 3 months
  - progressive neurological deficit
  - anterior cervical discectomy is usual surgical choice

LUMBAR DISC SYNDROME

Etiology
- ❑ protrusion/herniation of nucleus pulposus
  - laterally: compressing nerve root
  - centrally: compressing cauda equina
- ❑ common: L4-5 (L5 root), L5-S1 (S1 root)
- ❑ uncommon: L3-4 (L4 root)

Clinical Features
- ❑ leg pain > back pain
- ❑ limited back movement
- ❑ limited straight leg raising (L5, S1 roots) or femoral stretch (L4 root)
  - if there is true limitation in straight leg raising, patient should also be unable to sit up and extend knee fully
- ❑ nerve root irritation
  - pain in distribution of root (e.g. sciatica)
  - aggravated by movement, Valsalva maneuvers (increases CSF pressure leading to mechanical stimulation of inflamed nerve root)
  - relieved by rest
- ❑ impaired nerve root conductivity (may or may not be present)
  - motor deficit (lower motor neuron weakness)
  - sensory deficit (numbness in dermatome)
  - loss of reflex
  - sphincter paralysis (S2-S4)
- ❑ functional scoliosis (paravertebral muscle spasm)

Table 3. Lateral Lumbar Disk Syndromes			
	L3-4	L4-5	L5-S1
root involved	L4	L5	S1
pain	femoral pattern	sciatic pattern	sciatic pattern
motor	quads (knee extension)	tibialis anterior (dorsiflexion), EHL (hallux extension)	gastrocnemius, soleus (plantar flexion)
reflex	knee jerk	medial hamstrings	ankle jerk
sensory	medial leg	dorsal foot to hallux	lateral foot
relative incidence	< 10%	45%	45%

Differential Diagnosis
- ❑ spinal: stenosis, tumour, spondylolisthesis or ankylosing spondylitis
- ❑ leg: spinal stenosis, arthritic hip, sciatic nerve lesion (e.g. tumour)
- ❑ pelvic bones: tumour
- ❑ functional /nonorganic

Investigations
- ❑ x-ray spine (only to rule out other lesions)
- ❑ CT
- ❑ myelogram and post-myelogram CT (if surgery contemplated and plain CT not conclusive)
- ❑ MRI

Management
- ❑ conservative
  - bedrest, braces, physiotherapy and NSAIDs
  - 95% improve spontaneously within 4 to 8 weeks
- ❑ surgical indications
  - intractable leg pain despite adequate conservative treatment for > 3 months
  - disabling neurological deficit
  - progressive neurological deficit
  - bowel or bladder paralysis is a surgical emergency (cauda equina syndrome)

CAUDA EQUINA SYNDROME

Etiology
- ❑ secondary to compression of lumbosacral nerve roots below conus medullaris
- ❑ extrinsic tumour, carcinomatous meningitis, arachnoiditis, spinal stenosis

Clinical Features
- ❑ multiple dermatome, bilateral sensory loss
- ❑ weakness and paraparesis in multiple roots and depression of reflexes
- ❑ saddle anesthesia (perianal, genital areas)
- ❑ bowel and bladder dysfunction (incontinence or retention)
- ❑ loss of sexual sensation and function

Management
- ❑ requires urgent investigation and decompression to preserve bowel and bladder function

HEAD INJURY

SPECIFIC INJURIES

Scalp Injury
- ❑ rich blood supply
- ❑ considerable blood loss (vessels contract poorly when ruptured)
- ❑ minimal risk of infection due to rich vascularity

Skull Fractures
- ❑ depressed fractures —> double density on skull x-ray
  - simple fractures
  - compound fractures —> increased risk of infection
- ❑ internal fractures into sinus —> meningitis, pneumocranium
- ❑ basal skull fractures —> not readily seen on x-ray, rely on clinical signs
  - retroauricular ecchymoses (Battle's sign)
  - periorbital ecchymoses (raccoon eyes)
  - hemotympanum
  - CSF rhinorrhea, otorrhea
  - suspect with Lefort II or III midface fracture

Cranial Nerve Injury
- ❑ most commonly olfactory

Arterial Injury
- ❑ e.g. carotid-cavernous (C-C) fistula

Extradural ("Epidural") Hematoma (see Figure 12)
- ❑ young adult, male > female
- ❑ temporal-parietal skull fracture —> ruptured middle meningeal artery
- ❑ brain often not damaged
- ❑ symptoms delayed: classic = "lucid interval" between concussion and coma (not present in children)
- ❑ prognosis: good with optimal prompt management
- ❑ CT: high density biconvex mass against skull, usually with uniform density and sharp margins "lens-shaped"
- ❑ management: evacuation with small craniotomy flap

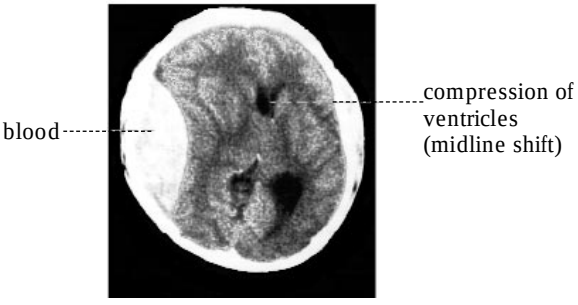

Figure 12. Extradural Hematoma on CT

Subdural Hematoma (see Figure 13)
- ❑ acute
  - torn cortical artery, large vein, or venous sinus due to violent trauma
  - often with associated brain injury, making prognosis poor overall
  - CT: increased density concave mass usually less uniform, less dense and more diffuse than extradural hematoma
  - management: craniotomy flap
- ❑ chronic
  - often minor injuries or no history of injury
  - several weeks after injury
  - "the great imitator" (of dementia, tumours, etc...)
  - risk factors: older, alcoholic, patients with CSF shunts, anticoagulants
  - expands due to repeated bleeding
  - brain usually undamaged
  - CT: low density concave mass
  - management: burr hole drainage

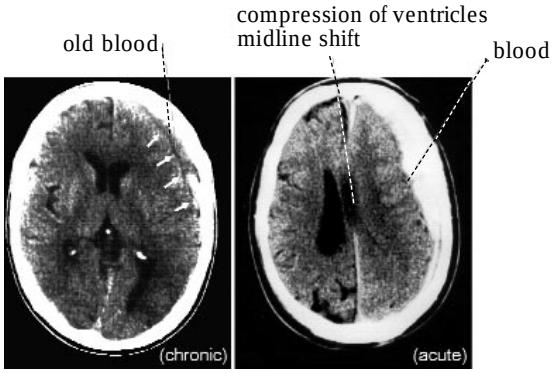

Figure 13. Subdural Hematoma on CT

Traumatic Intracerebral Hemorrhage
- ❑ any size, any part of brain, may be multiple
- ❑ immediate or delayed
- ❑ frontal and temporal lobes prominent (by coup/contre-coup mechanism)

BRAIN INJURY

Primary Impact Injury
- ❑ mechanism of injury determines pathology: i.e. with penetrating injuries, gun shot wounds
  - low velocity —> local damage
  - high velocity —> wave of compression, can get distant damage
- ❑ concussion
  - brief (< 6 hours) loss of consciousness
  - no parenchymal abnormalities on CT
- ❑ coup (damage at site of blow)
- ❑ contre-coup (damage at opposite site of blow)
  - acute decompression causes cavitation
  - followed by a wave of acute compression
- ❑ contusion (hemorrhagic)
  - high density areas on CT with little mass effect
  - commonly occurs with brain impact on bony prominences (falx, sphenoid wing, floor of frontal and temporal fossae)
- ❑ diffuse axonal injury (diffuse axonal shearing)
  - may tear blood vessels->hemorrhagic foci
  - wide variety of damage results
  - all brain injury causes shear
  - often the cause of decreased LOC if no space occupying lesion on CT

Secondary Pathologic Processes
- ❑ 1/3 who die in hospital after head injury were able to talk after the injury
- ❑ delayed and progressive
  - edema
  - intracranial hemorrhages
  - ischemia/infarction
  - raised ICP

Extracranial Conditions
- ❑ hypoxemia
  - trauma: chest, upper airway, brainstem
  - exceptionally damaging to traumatized brain cells
  - leads to ischemia, raised ICP
- ❑ hypercarbia
  - —> raised ICP
- ❑ systemic hypotension
  - caused by blood loss, not by head injury (e.g. ruptured spleen)
  - cerebral autoregulation lost in trauma
  - —> decreased CPP, ischemia
- ❑ hyperpyrexia
  - —> increased brain metabolic demands

- ❑ fluid and electrolyte imbalance
  - causes
    - iatrogenic (most common)
    - SIADH (from head injury)
  - diabetes insipidus (from head injury)
  - cerebral edema —> raised ICP
- ❑ fat embolism
  - multiple trauma
  - long bone fractures
  - petechiae and edema
  - hypoxia a key feature due to pulmonary effects
  - decreased LOC, seizures
- ❑ coagulopathy

Intracranial Conditions
- ❑ raised ICP due to
  - traumatic cerebral edema OR traumatic intracranial hemorrhage
- ❑ raised ICP results in
  - decreased cerebral perfusion (CPP = MAP – ICP)
  - +/– herniation

LATE COMPLICATIONS OF HEAD INJURY

Seizures
- ❑ 5% of head injured patients develop seizures
- ❑ incidence related to severity of injury, higher with local brain damage or intracranial hemorrhage
- ❑ with early (within first week) post traumatic seizure, incidence of later seizures rises to 25%

Meningitis
- ❑ associated with CSF leak from nose or ear

Hydrocephalus
- ❑ acute HCP or delayed NPH

SPINE INJURY

Vertebral Column (bone, discs, ligaments)
- ❑ commonly damaged
- ❑ compression fracture (stable)
- ❑ burst fracture (unstable)
- ❑ dislocation (unstable)
- ❑ fracture-dislocation (unstable)
- ❑ “special” fractures, e.g. odontoid (unstable)

Spinal Cord
- ❑ cord injury with initial bony or ligamentous trauma or after moving an unstable vertebral column
- ❑ complete
  - no preservation of sensory/motor function below lesion
  - no recovery
- ❑ incomplete lesions (see Spinal Cord Syndromes Section)

Nerve roots
- ❑ avulsion, e.g. brachial plexus in motorcycle accident

TRAUMA MANAGEMENT

Aims
- ❑ recovery from primary injury
- ❑ prevent further damage from secondary pathologic processes

Initial Management
- ❑ ABC’s of trauma management take priority

  A - airway with C-spine immobilization to prevent further spinal cord injury immobilize (collar, sandbags, fracture board, skull tong or halo traction)

B - breathing, ensure adequate oxygen supply
- oxygen (100%), ventilate if necessary

C - circulation
- differentiate hypotensive shock (BP low, HR high) from neurogenic shock (BP low, HR low)
- ensure adequate perfusion of spinal cord and manage neurogenic shock (dopamine, IV fluids, MAST)

❑ suspect spinal cord injury with weakness, numbness, spine pain, head injury, high energy injury or multisystem injuries
❑ rule out spinal fracture (cervical, thoracic, lumbar)
❑ 5-10% of patients with spinal injuries have injuries at other levels

Neurological Assessment
❑ mini history
- period of LOC
- post traumatic amnesia
- loss of sensation/function

❑ neurological exam
- head and neck (lacerations, bruises, basal skull fracture signs, facial fractures, foreign bodies)
- spine (palpable deformity, midline pain/tenderness)
- Glasgow Coma Scale
- eyes (pupillary size and reactivity)
- brainstem (breathing pattern, CN palsies)
- motor exam, sensory exam (only if GCS is 15), reflexes
- cranial nerve exam
- sphincter tone
- record and repeat neurological exam at regular intervals

Initial Investigations
❑ CT head and upper C-spine
❑ ABG, CBC, drug screen (especially alcohol)
❑ C,T,L-spine x-rays
- AP, lateral, odontoid views for C-spine
- must see C1 to C7 and C7-T1 interspace (swimmer's view if necessary)
- ABCS - Alignment, Bone, Cartilage, Soft tissues (see Orthopedics Notes)

❑ chest and abdomen x-ray as indicated

Late Management
❑ treatment for minor head injury
- observation over 24-48 hours
  - wake every hour
  - no sedatives or pain killers during this monitoring period

❑ treatment for severe head injury
- clear airway and ensure breathing (intubate if necessary)
- secure C-spine
- maintain adequate BP
- monitor to detect complications (Glasgow Coma Scale, CT, ICP)
- manage increased ICP if present
  - elevation of head
  - hyperventilation (target PCO2 32-35 torr)
  - mannitol (temporary preoperative measure)
- remove hematoma if present

❑ treatment of spinal injury
- reduce dislocation if present by traction or surgery
- stabilize spine if unstable (halo vest, fusion, etc...)
- further investigations (CT, tomogram, myelogram, MRI) to rule out cord compression
- emergent surgical decompression and/or fusion if necessary
- more likely to be beneficial in incomplete cord injury
- emergent surgery contraindicated for: complete spinal cord injury > 24 hours, medically unstable patient, and central cord syndrome
- steroids (methylprednisone) is proven to be of benefit if given within 8 hours of injury

❑ which patients should be admitted to hospital?
- skull fracture
- indirect signs of basal skull fracture

- confusion, impaired consciousness
- focal neurological signs
- extreme headache, vomiting
- seizures
- concussion with > 5 minutes amnesia
- social (i.e. no friend/relative to monitor for next 24 hours)
- unstable spine
- if there is any doubt, especially with children, or alcohol clouding consciousness

❑ which patients need CT head or transfer to a neurosurgical center?
- remains unconscious after resuscitation
- focal neurological signs
- deteriorating

KEY POINTS
❑ never do lumbar puncture in head injury
❑ all patients with head injury have C-spine injury until proven otherwise
❑ don't blame coma on alcohol - there may also be a hematoma
❑ low BP after head injury means injury elsewhere
❑ must clear spine both radiologically AND clinically

PERIPHERAL NERVES

INJURY

Classification and Clinical Course
❑ neuropraxia: intact nerve, recovery within hours to days
❑ axontomesis: axon disrupted but nerve sheath intact —> Wallerian degeneration —> recovery 1 mm/day
❑ neurotmesis: nerve completely severed, need surgical repair for recovery

Management
❑ electrophysiological studies (EMG, nerve conduction velocities) may be helpful in assessing nerve integrity
❑ surgical repair unless nerve is known to be intact
❑ delay surgical repair for a few weeks (unless first 2 conditions met) to allow
- clean wound
- optimal surgical facilities
- optimal cell metabolism
- possible spontaneous recovery/regeneration

❑ microsurgery: suture nerve sheaths +/– nerve graft

ENTRAPMENT

General
❑ nerve compressed by nearby anatomic structures
❑ often secondary to localized, repetitive mechanical trauma with additional vascular injury to nerve
❑ consider systemic causes
- rheumatoid arthritis
- diabetes mellitus
- hypothyroid
- acromegaly
- vasculitis
- amyloidosis
- pregnancy

❑ symptoms
- pain distal (occasional proximal) to lesion
- burning paresthesia/dysesthesia
- sensory loss in nerve distribution
- muscle weakness/wasting (advanced cases)

Carpal Tunnel Syndrome (CTS)
❑ etiology
- most common entrapment neuropathy
- median nerve entrapment at wrist, usually bilateral
- female:male = 4:1

- ❑ presentation
  - classically: patient awakened at night with numb/painful hand, relieved by shaking/dangling/rubbing
  - distribution: radial 3.5 fingers
  - decreased light touch, 2 point discrimination, especially finger tips
  - job/hobby related repetitive trauma, especially forced wrist flexion
  - advanced cases: wasting/weakness of thenar muscles, especially abductor pollicis brevis
- ❑ diagnosis
  - history, physical
  - +/– Tinel's sign (tingling sensation on percussion of nerve)
  - +/– Phalen's sign
  - confirm with NCV, EMG pre-operatively
- ❑ management
  - conservative
    - neutral wrist splints (bedtime)
    - NSAIDs
    - steroid injection
  - surgical
    - release of flexor retinaculum
    - indications for surgery: refractory pain, +++ sensory loss, muscle atrophy

Ulnar Nerve Entrapment at Elbow

- ❑ second most common entrapment neuropathy
- ❑ may be entrapped at several locations
  - behind medial epicondyle
  - at medial intermuscular septum
  - distal to elbow at cubital tunnel
- ❑ presentation
  - sensory: pain, numbness in ulnar 1.5 fingers
  - wasting of interossei (especially first dorsal interosseous —> thumb web space)
  - weakness (especially abduction of index finger)
- ❑ diagnosis
  - history, physical
  - NCV: conduction delay across elbow
- ❑ management
  - conservative: prevent repeated minor trauma (e.g. leaning on elbow or sleeping with hand under head), elbow pads, NSAIDs
  - surgical: nerve decompression and transposition to front of elbow

Less Common Entrapments

- ❑ common peroneal nerve
  - superficial and fixed behind fibular head
  - sensitive to trauma (e.g. fracture of fibular head)
  - motor: decreased foot and toe extension ("drop foot"), decreased ankle eversion
  - sensory: decreased lateral foot and dorsum (less common)
  - distinguish from L5 radiculopathy
- ❑ lateral cutaneous nerve of the thigh ("meralgia paraesthetica")
- ❑ posterior tibial nerve ("Tarsal Tunnel")
- ❑ radial nerve ("Saturday Night Palsy")
- ❑ motor branch of ulnar nerve at wrist (Guyon's canal)
- ❑ thoracic outlet syndrome (compression of inferior trunk or C8-T1 nerve roots of brachial plexus by cervical rib, fascial bands, etc... hard to diagnose)

PHYSIOLOGY OF PAIN

- ❑ peripheral sensors: free nerve endings
- ❑ neurotransmitters: substance p, endorphins
- ❑ gate control theory:
  - summation of inhibitory and excitatory afferent input at the synapse to the second order neuron of the spinothalamic tract determines amount of pain felt
  - segmental and higher center influence
- ❑ perception:
  - thalamus to cerebral cortex —> awareness
  - personality and mood —> intensity
  - spinothalamic tract, reticular formation and limbic system —> unpleasant, emotional aspect

MEDICAL TREATMENT

- ❑ acute pain (< 2-3 weeks duration): analgesics +/– tranquilizers
- ❑ benign chronic pain: antidepressants, anticonvulsants, topical (capsicin), NOT narcotics or sedatives
- ❑ malignant chronic pain: strong narcotics in frequent, small doses

SURGICAL TREATMENT

Central

- ❑ stereotactic thalamotomy
  - remove spinoreticular relay
  - indication: malignancy of head, neck or brachial plexus
- ❑ deep brain stimulation
  - stimulation of electrodes placed in periventricular gray matter, sensory relay nucleus of thalamus or internal capsule +/– radiocontrolled stimulator subcutaneously
- ❑ hypophysectomy (chemical: uses alcohol)
  - unknown mechanism
  - indication: metastatic disease
- ❑ dorsal root entry zone lesions
  - indication: deafferentation pain (brachial plexus avulsion, postherpetic neuralgia)
  - major complication: ipsilateral leg weakness
- ❑ percutaneous anterolateral cordotomy
  - lesion of spinothalamic tract giving pain relief contralaterally
  - 90% patients respond
  - complications: respiratory difficulties and ipsilateral limb weakness
- ❑ myelotomy
  - division of decussating pain fibers for temporary pain relief
  - indication: terminal malignancy
- ❑ dorsal column stimulation
  - percutaneous electrodes in epidural space
  - indication: intractable chronic pain

Peripheral

- ❑ nerve blocks
  - dermatomal pain relief, loss of motor and sympathetic function
  - permanent: neurolytics (phenol, alcohol)
  - temporary: local anesthetics
  - paravertebral or peripheral: NOT neurolytics —> painful neuritis
- ❑ transcutaneous electrical nerve stimulation (TENS)
  - prolonged stimulation of large diameter fibers inhibiting ascending pain fibers or via higher centers
- ❑ dorsal rhizotomy
  - dorsal root division
  - infrequently done: high failure rate and short effect
- ❑ denervation of facet joints
  - cut posterior ramus of spinal nerves
  - temporary: relief until nerve regrows

TIC DOLOUREUX

Clinical Features

- ❑ older age
- ❑ location: V2 > V3 > V1 (combinations occur), mostly unilateral

- ❑ short, sharp jabs in series, last a few seconds to a few minutes
- ❑ violent, terrible, "lightning", "electrical", "lancinating" pain
- ❑ may be weeks or months of remission
- ❑ neurological examination commonly normal
- ❑ triggers: areas on face (especially around mouth), wind, eating, drinking, talking

Diagnosis
- ❑ history
- ❑ rule out structural lesion affecting trigeminal nerve (tumour, aneurysm) - CT, MRI
- ❑ may be due to multiple sclerosis (especially in younger patients)

Management
- ❑ pharmacologic
  - drug of choice is carbamazepine 200 mg tabs, 3-5 per day
  - phenytoin is second choice
  - baclofen (potentiates carbamazepine effect)
  - response to medication is almost diagnostic
  - eventually becomes refractory
- ❑ procedures
  - percutaneous thermocoagulation of CN V
  - glycerol injection into Meckel's cave (trigeminal cistern)
  - division/avulsion of branches of CN V in face
  - microvascular decompression of CN V at pons

CAUSALGIA (REFLEX SYMPATHETIC DYSTROPHY)

Etiology
- ❑ incomplete peripheral nerve injury in nerve with sympathetic fibers

Clinical Features
- ❑ intense, continuous, burning pain
- ❑ touch worsens pain
- ❑ red, warm, dry and swollen skin initially (sympathetic overactivity)
- ❑ cool, clammy, glossy and atrophic skin in advanced stages

Treatment
- ❑ sympathetic nerve blockade: medical or surgical

POSTHERPETIC NEURALGIA

Etiology
- ❑ reactivation of latent varicella zoster virus that lay dormant in dorsal root or gasserian ganglion

Clinical Features
- ❑ burning, constant pain
- ❑ severe, sharp paroxysmal twinges over area of affected sensory neurons
- ❑ touch worsens pain

Treatment
- ❑ no specific treatment and is difficult to treat
- ❑ medical: antidepressants, carbamazepine, ethychloride spray (temporary relief), topical (capsicin, promising new treatment that works by blocking substance P), steroid injection or topical in eye during acute eruptive phase (decreases severity of pain and decreases corneal scar)
- ❑ surgical: percutaneous cordotomy, possible dorsal root entry zone lesion

THALAMIC PAIN

Clinical Features
- ❑ begins with hemianesthesia (due to thalamic infarction or hemorrhage)
- ❑ becomes diffuse, burning pain contralateral to lesion
- ❑ worse with light touch (e.g. clothing)
- ❑ may have prior history of thalamic stereotactic procedure for movement disorder

Treatment
- ❑ medical: poor response to medication
- ❑ surgical: stereotactic thalamic stimulation but may increase sensory deficit

PHANTOM LIMB PAIN

Etiology
- ❑ complication of 10 % amputation patients
- ❑ neuroma formation in stump

Clinical Features
- ❑ continuous burning
- ❑ pain from some point on missing limb

Treatment (see Postherpetic Neuralgia Section)

PEDIATRIC NEUROSURGERY

SPINA BIFIDA OCCULTA

Definition
- ❑ neural defect
- ❑ usually CNS (cauda and PNS not involved)
- ❑ radiological diagnosis (not associated with neurologic defect, only bony deficit)

Epidemiology
- ❑ 20-30% of the general population

Etiology
- ❑ failure of fusion of the posterior arch

Clinical Features
- ❑ no obvious external markings
- ❑ no obvious clinical signs
- ❑ presence of skin dimple or hair tuft should increase suspicion of an underlying anomaly (occult spinal disraphysm)

Investigations
- ❑ plain film: absence of the spinous process along with minor amounts of the neural arch
- ❑ most common at L5 or S1

Treatment and Results
- ❑ requires no treatment

MENINGOCELE

Definition
- ❑ a defect consisting of a herniation of meningeal tissue and CSF through a defect in the spine

Etiology
- ❑ 2 theories
  - primary failure of neural tube closure
  - rupture of a previously closed neural tube due to overdistension (Gardner; unpopular theory)

Clinical Features
- usually no disability
- low incidence of associated anomalies and hydrocephalus

Investigations
- ❑ plain films, CT, MRI, ultrasound, cardiac echo, GU investigations

Treatment and Results
- ❑ surgical excision (excellent results)

MYELOMENINGOCELE

Definition
- ❑ a defect consisting of a herniation of meningeal tissue and CNS tissue through a defect in the spine

Etiology - same as meningocele

Clinical Features
- ❑ sensory and motor changes distal to anatomic level producing varying degrees of weakness, anesthesia, urine and fecal incontinence

Investigations
- ❑ plain films, CT, MRI, ultrasound, cardiac echo, GU investigations

Surgical Indications
- ❑ preserve intellectual, sensory and motor functions
- ❑ prevent CNS infections

Results
- ❑ operative mortality close to 0%
- ❑ 95% 2 year survival
- ❑ 80% have IQ in > 80 (but most are 80-95)
- ❑ 40-85% ambulatory
- ❑ associated with hydrocephalus (80%)
- ❑ complications: ventriculitis, ICH

INTRAVENTRICULAR HEMORRHAGE

Definition
- ❑ a disease of the premature, low-birth weight infant
- ❑ consists of hemorrhage into the germinal matrix of the developing brain

Classification (based on ultrasound or CT)
- ❑ Grade I: germinal matrix hemorrhage only
- ❑ Grade II: blood filling lateral ventricles without distention
- ❑ Grade III: blood filling and distending lateral ventricles
- ❑ Grade IV: hemorrhage with parenchymal involvement (ICH)

Epidemiology
- ❑ occurs in 45% of infants born with a birth weight of 1500 g or less
- ❑ uncommon after 32 weeks of gestation
- ❑ essentially nonexistent in full-term infants

Predisposing Factors
- ❑ low gestational age
- ❑ low APGAR score
    - vaginal delivery
    - birth asphyxia, resuscitation
    - respiratory distress syndrome
    - rapid volume re-expansion
    - hypoxemia, hypercarbia, acidosis
    - seizure, pneumothorax

Investigations
- ❑ ultrasound is the method of choice to screen for ICH/IVH
- ❑ should be done routinely to screen preterm babies < 24 weeks gestation or < 1500 gm
- ❑ CT scan will also show ICH and IVH as described above

Treatment
- ❑ best to withhold tapping ventricles, ventriculostomies, and shunting until blood has cleared
- ❑ if progressive hydrocephalus develops, then
  - serial LP
  - acetazolamide (25-100 mg/kg/day) and Lasix (2 mg/kg/day)
  - ventriculostomy
  - shunt (low pressure)

Results
- ❑ grade I-III hemorrhages can do as well as children without hemorrhages
- ❑ grade IV: only 50% chance of attaining normal life status
- ❑ prognosis more dependent on the degree of asphyxia than on hydrocephalus

HYDROCEPHALUS IN PEDIATRICS

Etiology
- ❑ congenital
  - aqueductal anomalies
    - primary aqueductal stenosis in infancy
    - secondary gliosis due to intrauterine viral infections (mumps, varicella, TORCH) or germinal plate hemorrhage
  - Dandy Walker (2-4%)
  - Chiari malformation, especially Type II
  - myelomeningocele
- ❑ acquired
  - post meningitis
  - post hemorrhage (SAH, IVH)
  - masses (vascular malformation, neoplastic)

Clinical Features
- ❑ symptoms and signs of hydrocephalus are age related in pediatrics
- ❑ increased head circumference
- ❑ irritability, lethargy, poor feeding and vomiting
- ❑ bulging anterior fontanelle
- ❑ widened cranial sutures
- ❑ ”cracked pot” sound on cranial percussion
- ❑ scalp vein dilation (increased collateral venous drainage)
- ❑ sunset sign - forced downward deviation of eyes
- ❑ episodic bradycardia and apnea

Management
- ❑ similar to adults (see Hydrocephalus Section)

DANDY-WALKER MALFORMATION

Definition
- ❑ atresia of foramina of Magendie and Luschka, resulting in
  - complete or incomplete agenesis of the cerebellar vermis
  - posterior fossa cyst
  - dilatation of 4th ventricle
  - enlarged posterior fossa
- ❑ associated anomalies
  - hydrocephalus (90%)
  - agenesis of corpus callosum (17%)
  - occipital encephalocele (7%)

Epidemiology
- ❑ 2-4% of pediatric hydrocephalus

Clinical Features
- ❑ 20% are asymptomatic
- ❑ only 50% have normal IQ
- ❑ symptoms and signs of hydrocephalus combined with a prominent occiput in infancy
- ❑ ataxia, spasticity, poor fine motor control common in childhood
- ❑ seizures occur 15%

Treatment
- ❑ asymptomatic patients require no treatment
- ❑ associated hydrocephalus requires surgical treatment

CHIARI MALFORMATIONS

Definition
- ❑ malformations at the medullary-spinal junction

Clinical Features
- ❑ Type I (cerebellar ectopia): cerebellar tonsils lie below the level of the foramen magnum
  - average age at presentation 41 years
  - brain compression: suboccipital headache, nystagmus, ataxia, spastic quadraparesis
    - foramen magnum compression syndrome (22%)
    - central cord syndrome (65%)
    - cerebellar syndrome (11%)
  - hydrocephalus
  - syringomyelia
- ❑ Type II: part of cerebellar vermis, medulla and 4th ventricle extend through the foramen magnum often to midcervical region
  - present in infancy
  - findings due to brain stem and lower cranial nerve dysfunction: swallowing difficulties, apneic spells, stridor, aspiration, arm weakness
  - syringomyelia, hydrocephalus in > 80%
- ❑ Type III: displacement of posterior fossa structures with cerebellum herniated through foramen magnum into cervical canal (rare, usually incompatible with life)
- ❑ Type IV: cerebellar hypoplasia without cerebellar herniation

Investigations
- ❑ MRI or CT myelography

Treatment
- ❑ surgical decompression - indications
  - Type I: symptomatic patients (early surgery recommended)
  - Type II: neurogenic dysphagia, stridor, apneic spells

CRANIOSYNOSTOSIS

Definition
- ❑ premature closure of the cranial suture(s)

Classification
- ❑ saggital - most common
- ❑ coronal
- ❑ lambdoid - least common
- ❑ metopic (forehead)
- ❑ multiple suture synostosis or pansynostosis

Epidemiology
- ❑ 0.4/1000 live births
- ❑ most cases are sporadic
- ❑ familial incidence is 2% of saggital and 8% of coronal synostosis

Clinical Features
- ❑ skull deformity
- ❑ raised ICP
- ❑ ophthalmologic problems
  - due to increased ICP or bony abnormalities of the orbit
  - strabismus most common
- ❑ hydrocephalus may accompany multiple craniosynostoses

Investigations
- ❑ plain radiographs, CT scan (3D)
- ❑ bone scan: increased activity during active phase of union, decreased once union has occurred

Management
- ❑ parental counseling about
  - nature of deformity
  - difficulty growing up as "cone head"
  - associated neurological symptoms
- ❑ surgery for cosmetic purposes, except in cases of elevated ICP

PEDIATRIC BRAIN TUMOURS
- ❑ 20% of all pediatric cancers (second only to leukemia)
- ❑ 80% of pediatric tumours are infratentorial (see Intracranial Mass Section)
- ❑ most common manifestations
  - vomiting
  - arrest or regression of developmental milestones
  - macrocrania
  - poor feeding, failure to thrive
  - hydrocephalus

DRUGS

- ❑ the following are guidelines ONLY; follow clinical judgment and up-to-date prescription recommendations in practice; dosages refer to adults unless otherwise specified

Carbamazepine (Tegretol)
- ❑ Tic Douloureux
  - 100 mg PO bid, increase by 200 mg/day up to the usual dose 200 mg tid (maximum 1200 mg/day divided tid)
- ❑ seizures
  - 600-2 000 mg/day, start low and increment slowly (inpatient: every 3 days; outpatient: every week)
  - monitor CBC (potential hematological toxicity)

Dexamethasone (Decadron)
- ❑ ICP (e.g. secondary to tumour)
  - loading: 10-20 mg IV
  - maintenance: 4-6 mg IV q6h

Lorazepam (Ativan)
- ❑ status epilepticus
  - 4 mg IV over 2 minutes, q5 minutes
  - start phenytoin loading simultaneously

Mannitol
- ❑ raised ICP
  - bolus 1 gm/kg IV rapid infusion (350 mL of 20% solution)
  - followed by 0.25 g/kg IV q6h
  - effect occurs in 1-5 minutes, maximal at 20-60 minutes
  - often alternated with furosemide (Lasix) 10-20 mg IV q6h

Methylprednisolone (Solumedrol)
- ❑ spinal cord injuries
  - within 8 hours of injury
  - provided as solution of 62.5 mg/mL
  - bolus 30 mg/kg over 15 minutes, then 45 minute pause, then 5.4 mg/kg/hour continuous infusion x 23 hours (maintained during any necessary surgery if possible)

Nimodipine (Nimotop)

- ❑ SAH
  - vasodilation
  - only Ca channel blocker to cross BBB
  - 60 mg PO/NG q4h x 21 days, started within 96 hours of SAH
  - half dose for liver failure
  - monitor BP

Phenytoin (Dilantin)

- ❑ seizures
  - IV: loading dose 18 mg/kg, maintenance of 200-500 mg/day
  - oral: 300-600 mg/day divided bid/tid, loading dose 300 mg PO q4h until 17 mg/kg given
  - average maintenance dose: 300 mg/day po
  - important to give over time to prevent causing a cardiac arrest
- ❑ status epilepticus
  - not on phenytoin: 1200 mg IV over 30 minutes (~ 20 mg/kg)
  - already on phenytoin: 500 mg IV over 10 minutes