Acute Care SINS:

Surgical Insights for the Non-surgeon

Chapter 12: Brain Surgery SINS

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Summary

“Surgical Insights for the Non-surgeon,” or SINS, is composed of several short chapters intended to cover fundamental surgical knowledge for non-surgeons. The authors focus on surgical pearls, operative insights, and applied anatomy. In Chapter 12 of this series, the authors address the brain and neurosurgery Part One.

Résumé

L’ouvrage « Surgical Insights for the Non-surgeon » (aperçu de la chirurgie à l’intention du non-chirurgien) se compose de courts chapitres couvrant les connaissances fondamentales en chirurgie. Les auteurs se concentrent sur des enseignements tirés de leur expérience, des aspects opératoires et l’anatomie appliquée. Dans le chapitre 12 de cet ouvrage, les auteurs abordent le cerveau et la première partie de neurochirurgie.

*“I would like to see the day when somebody would be appointed surgeon who had no hands,
for the operative part is the least part of the work.”*
—Harvey Cushing, Father of Neurosurgery

Anatomy

First, the disclaimer: brain anatomy can be daunting, and what takes a moment to describe can take a career to master. Moreover, there are variations in our “brain wiring,” such that cerebral insults (whether strokes, trauma, or tumours) affect people differently. However, a basic knowledge of neuroanatomy means that pinpointing the injury usually explains the deficit, and informs the prognosis.

The cerebrum (Figure 1) has four primary lobes: frontal, temporal, parietal, and occipital. It contains folds that i) increase its surface area (two-thirds of the brain is hidden in the folds), and ii) increase its ability to grow (a smooth brain's growth would be limited). These folds, in turn, are called i) sulci: depressions or grooves, the singular of which is sulcus; ii) gyri: the intervening up-swelling, the singular of which is gyrus, and iii) fissures: larger grooves separating the brain into primary lobes and into two hemispheres. The lateral fissure (a.k.a. the lateral sulcus or the Sylvian fissure) separates the frontal and parietal lobes (above) from the temporal lobe (below). The midline interhemispheric fissure separates the brain into left and right hemispheres. The central sulcus/fissure (a.k.a. the Rolandic fissure) divides the frontal and parietal lobes.

The pre-central gyrus (i.e. the frontal lobe area in front of the central fissure) is responsible for motor output, and the post-central gyrus (i.e. the parietal lobe area behind the central fissure) is responsible for sensory input from the body. Regarding speech, the left hemisphere is “dominant” (meaning it mediates speech) in > 95% of right-handed individuals and approximately 85% of left-handed individuals. This means that approximately 1 in 7 people will control speech from their non-dominant right hemisphere or have significant bilateral speech representation. Within the frontal lobe, Broca's area is responsible for speech expression. Within the posterior temporal lobe, Wernicke's area is responsible for speech comprehension. Damage will cause, respectively, an expressive

aphasia (and the inability to utter expressions such as, “no ifs, ands, or buts”), versus a receptive aphasia (fluent but non-comprehensible speech).

The thalamus (from the Greek for “chamber”) is a bilateral, midline symmetrical structure, located between the cortex and the midbrain. It relays sensory and motor signals and helps regulate consciousness, sleep, and alertness. Laterally, the basal ganglia comprise the striatum (caudate nucleus and putamen), globus pallidus, substantia nigra, nucleus accumbens, and subthalamic nucleus. The basal ganglia help mediate such diverse behaviours as voluntary motor movements, procedural learning, eye movements, and cognition. These diverse functions help explain why midline (or deep cortical) damage is so serious.

The brain's ventricles contain cerebrospinal fluid (CSF) (Figures 2a and 2b). The two lateral ventricles are composed of frontal (or anterior) horns, a body, an atrium, temporal (or inferior) horns, and occipital (or posterior) horns. CSF is produced within those lateral ventricles by the choroid plexus. Benign calcifications within the choroid plexus are common and are visible on CT as small hyperdensities—these are not small intraventricular bleeds! CSF drains into a single third ventricle via the interventricular foramen of Monro, then via the cerebral aqueduct of Sylvius to the fourth ventricle. Thereafter, it leaves the ventricular system via the foramen of Luschka laterally and via the midline foramen of Magendie (“lateral Luschka, midline Magendie”). CSF enters the subarachnoid space and is reabsorbed into the superior sagittal sinus through arachnoid granulations. We produce 500 mLs of CSF daily, but the ventricles have capacity for only 150 mLs. This means that CSF is produced and continually reabsorbed. Inability to drain CSF causes hydrocephalus, increased intracranial pressure (ICP), and tissue damage.

Brain herniation (Figure 3) is frequently deadly and occurs when part of the brain squeezes outside of its usual confines. The two categories of herniation are i) supratentorial

Figure 1. The cerebral lobes and fissures.

Figure 2A. The ventricles of the brain.

Figure 2B. Mid-sagittal illustration of the brain identifying areas of cerebrospinal fluid (CSF) production, flow and reabsorption.

(structures above the tentorial notch) and ii) infratentorial (below the tentorial notch). “Tentorial” refers to the tentorium cerebelli, which is the dura mater separating the cerebellum from occipital lobes. Supratentorial herniation can be subdivided into uncus (transtentorial), central, cingulate (subfalcine), and transcalvarial. Infratentorial herniation is of the cerebellum and can be upward (a.k.a. upward cerebellar or upward transtentorial) or downward (tonsillar herniation). Now, on to more clinical matters . . .

Head Trauma

General Comments

- Predictors of good outcome:
 - Better neurologic status on arrival
 - i.e. Higher Glasgow Coma Score (GCS) (see Trauma SINS chapter)

Figure 3. Illustration of supratentorial herniation (divided into transcalvarial, subfalcine, transtentorial, or central); and infratentorial herniation (upward or downward tonsillar herniation).

- Younger age
- Prompt surgical treatment of mass lesions
- Predictors of bad outcome:
 - Hypoxia
 - Hypotension
 - Unreactive pupil
- Following major head trauma
 - Approximately one-third require urgent surgical evacuation

Epidural (a.k.a. Extradural) Hematoma (EDH)

- Typically follows trauma to the middle meningeal artery (MMA)
 - The MMA runs in a groove on the inner surface of the thin temporal bone
 - Therefore, it can be torn when the skull is fractured

- Might be the explanation for how David beat Goliath with just a slingshot!
- Bleeding causes the dura mater to separate from the skull's inner table, filling the epidural space
- Classic clinical presentation
 - Brief loss of consciousness (LOC)
 - Followed by a 'lucid interval' which is usually brief but can last hours
 - Lucid interval followed by obtundation (due to symptoms of mass effect)
 - Mass effect on ipsilateral motor strip causes contralateral hemiparesis
 - Further mass effect causes uncal herniation (see above)
 - Which causes ipsilateral pupillary dilatation (a.k.a. "a blown pupil")
 - Due to pressure on the ipsilateral third cranial nerve
- Evaluation
 - CT head:
 - Hyperdensity due to acute blood
 - 'Biconvex' or lens-shaped hematoma in the epidural space
 - Epidural blood may not spread past bony suture lines
 - This is because the dura mater adheres to the suture lines
 - This contrasts with subdural bleeds (SDH) (see below)
 - Following an EDH, also look for skull fracture on the CT
- Medical management
 - Small volume EDH can be followed (albeit frequently)
 - If no neurological deterioration/focal signs, then no surgery
 - Admit to hospital for observation
 - OPERATE EARLY if patient deteriorates (i.e. if GCS drops)
- Surgical management
 - For symptomatic patients
 - Craniotomy for hematoma evacuation
 - Cauterize any bleeding arteries
 - Simple burr hole is ineffective for an acute epidural hematoma
 - Because acutely clotted blood is too thick to aspirate
- Outcomes
 - Mortality is reduced substantially with surgery
 - Earlier intervention yields better outcomes
 - 'Time is brain'
- Bridging veins more likely to tear because of enlarged subdural space
 - i.e. veins are under tension
- SDH often results from severe acceleration-deceleration head movement
 - This acceleration/ deceleration motion can mean significant injury to the underlying brain
- Clinical presentation
 - Patients often more obtunded than those with EDH
 - Also no lucid interval following SDH (versus EDH)
- Evaluation
 - CT head shows crescent-shaped hematoma
 - Compared to lens-shaped for EDH
 - Usually over the convexity of the entire hemisphere
 - Compared to blood restricted by suture lines after an EDH
 - Appearance of blood on CT (density) changes over time
 - Acute (first few days)
 - Hyperdense to the brain (white blood; grey brain)
 - Subacute (week to month)
 - Isodense to the brain (grey blood; grey brain)
 - Chronic (greater than 1 month)
 - Hypodense to the brain (black 'blood'; grey brain)
- Treatment
 - If a small bleed (<1 cm, 5 mm midline shift) and a stable/asymptomatic patient,
 - Frequent observation but no automatic need for surgery
 - If large clots: mass effect
 - Typically need a craniotomy for evacuation
 - Bleeding vessels are rarely sought out or found, so rarely need to cauterize
 - In contrast to EDH
- Outcomes
 - All surgical decisions should be individualized
 - The decision to operate depends on premorbid state, extent of disease, and patient's wishes
 - However, mortality following SDH increases with age
 - Therefore, some surgeons are reluctant to operate on those aged over 65 years
 - Therefore, more surgeons are reluctant to *re-operate* on those aged over 65 years

Subdural Hematoma (SDH)

Acute Subdural Hematoma

- Etiology
 - Most commonly from tearing of the bridging veins
 - These extend between the cortex and the dural sinuses
 - More common in an atrophied brain (alcohol; advanced age)

Chronic Subdural Hematoma

- Typically occurs in elderly patients
- Ask about minor head trauma in the preceding weeks
 - Though only recalled in 50% of patients
- Also likely due to tearing of bridging veins
 - But requires less and less head motion as the brain ages
 - Due to the increased stretch on these veins in an atrophic brain
- Often present with several weeks of headache
- Can be confused with meningitis/alcoholism, etc.

- Undiagnosed, this can progress to confusion and focal neurologic deficits
- Most of the blood is old and liquefied
 - Consistency is like 'motor-oil' when removed
- In symptomatic patients
 - Evacuate via a burr hole
 - Wash out the blood
 - Plus/minus insert a subdural drain
- Majority of symptomatic patients improve within hours of surgery

Traumatic Subarachnoid Hemorrhage (SAH)

- Blood in the SA space
 - From trauma to small cortical vessels (usually veins)
- More common (and different) than aneurysmal SAH (see below)
 - Less clinically concerning than aneurysmal SAH
- Traumatic SAH has minimal clinical significance in isolation, but serves as marker of a more significant head injury
 - Does not require specific treatment, other than observation
 - Treatment focuses on managing any other associated head injury
- No surgical treatment required
 - Neurologically intact patients managed with observation
 - Repeat imaging, based on GCS
 - Patients may have concussion symptoms during the recovery phase

Intraparenchymal Contusion and Hematoma

- CT appearance is mixed-density following contusion
- CT appearance is high-density following hematoma
- Small contusions in asymptomatic patients can be observed
 - Repeat imaging only if clinical deterioration
- When symptomatic from mass effect, contusions and hematoma may require surgery
 - Via craniotomy
 - Remove blood and dead brain to minimize swelling
 - Via craniectomy
 - Leave bone flap off to give extra room for cerebral edema
- In patients *without* trauma
 - Intraparenchymal hematomas are usually hypertensive hemorrhagic strokes
 - Typically managed by the stroke neurology team, rather than neurosurgeons
 - Hydrocephalus from posterior fossa blood warrants a neurosurgical opinion

Diffuse Axonal Injury

- Diffuse damage to the brain (as opposed to focal damage from bleed, etc.)
- Shearing of axons

- At the junction of grey and white matter and in the deep white matter
- Patient's injury may be neurologically devastating, despite CT scan looking relatively innocuous
- MRI can help determine the extent of injury
- Surgical management is limited
 - After all, there are no focal lesions to evacuate
 - Rare decompressive craniectomy (see below) for elevated ICP if medical management fails
- Long-term outcome may be acceptable, even if low GCS initially
 - So, life support may be maintained for longer than following other head injuries

Management of Increased Intracranial Pressure (ICP)

Pathophysiology

- Monro-Kellie doctrine
 - Intracranial volume is constant
 - Therefore, any increase in any one of its contents (brain, CSF, blood, space-occupying lesion) must be offset by an equal decrease in one of the others
 - If not, then ICP increases
 - Increased brain pressure → increased brain swelling → increased brain injury
 - Higher pressure means CSF is displaced through the foramen magnum and venous blood is shunted extracranially
 - Further increased brain pressure obstructs arterial flow
 - Mean arterial pressure (MAP) must increase to maintain status quo
 - If unable to compensate:
 - Decreased oxygenation of cerebral tissue occurs
 - Neuronal hypoxia, anoxia, and death
- Cerebral perfusion pressure (CPP)—and therefore cerebral blood flow (CBF)—depends on MAP (forward flow) minus ICP (obstruction to forward flow)
 - $CPP = MAP - ICP$
 - $CBF = CPP / Resistance$
 - BUT resistance cannot be easily measured, so we target CPP
- Normal adult ICP in adults is <10–15 mmHg; sustained >20 mmHg is pathologic
- Cushing's response
 - Increased ICP causes increased pressure on the brain stem
 - This causes irregular respirations, increased systolic blood pressure, and bradycardia
 - A "Cushing's response" indicates the body is trying to compensate for higher ICP
 - By increasing systolic blood pressure (SBP) via the sympathetics in order to increase CPP
 - This increased SBP causes reflex bradycardia
 - Cushing's response indicates that 'the brain is not happy'

(i.e. its CBF is inadequate)

- It indicates that brainstem herniation via the foramen magnum (a.k.a. “coning”) is imminent without intervention

Medical Management of High ICP

- Raise head off bed to 30 degrees to enhance venous outflow
 - Beware, however, because raising the head also decreases cerebral arterial perfusion
 - Therefore, compensate by placing your arterial transducer at the tragus (level with the ear/circle of Willis)
 - Rather than usual phlebostatic axis (level of the heart)
 - Because you want to know the perfusion pressure in the brain
 - Not in the major systemic vessels
 - Or do not move transducer, but instead target a higher CPP
 - i.e. 70 rather than 65 mmHg, to compensate for the raised head
- Mannitol
 - Causes an osmotic diuresis: flow of fluid out of a swollen brain
 - Decreases intravascular volume and raises serum sodium
 - This (hopefully) decreases brain edema, improves blood rheology, and increases CBF
 - Mannitol comes only in 20% solution; 250 mL and 500 mL bags
 - Dose is 0.5–1.0 g / kg; given as an intravenous (IV) bolus
 - Give approximately one large bag for a large person (i.e. 100 g)
 - Give approximately one small bag for a small person (i.e. 50 g)
- Hypertonic saline – same osmotic mechanism as mannitol
 - Currently an alternative to mannitol
 - May lead to less volume depletion, compared to mannitol
 - Dose for 3% hypertonic saline is 150–250 mLs IV
- Hyperventilation – decrease arterial CO_2
 - End Tidal CO_2 can be used to estimate PaCO_2
 - End Tidal CO_2 is usually 5 mmHg less than arterial PCO_2
 - Lower PaCO_2 leads to cerebrovascular vasoconstriction (CVV)
 - CVV leads to decreased intracranial volume
 - HOWEVER, PaCO_2 reduction is a temporary measure
 - New equilibrium will be reached and vessels return to normal calibre
 - Will also decrease brain oxygenation (bad in an injured brain!)
 - Therefore, do not target low PaCO_2 until you need to
 - Also, only hyperventilate to temporize
 - While coordinating definitive management, such as decompression of clot or craniectomy

- Osmotic agents and hyperventilation are used in emergency situations, such as:
 - Acutely elevated ICP
 - Patient arriving in the Emergency Room with un-reactive dilated pupils
 - En route to the OR for emergency surgery
- Sedation/paralysis/analgesics/anesthetics
 - Given to reduce sympathetic tone
 - Given to reduce ICP effect of voluntary muscle movements
 - Given to reduce cerebral metabolic demands
- Therapeutic hypothermia
 - Not universally applied, as little evidence of benefit
 - Benefit may just be from avoidance of hyperthermia
 - Theory is that it reduces cerebral metabolic demands
 - Which protects against injury from increased ICP
 - Some believe the lack of benefit is because hypothermia is started too late
- Other
 - Prompt treatment of seizures
 - Avoid hyperglycemia and hypoglycemia

Surgical Management of High ICP

- An intraparenchymal pressure monitor
 - Inserted through the skull and rests in superficial brain tissue
 - Measures ICP, but does not drain CSF
 - For obtunded patients
 - Where serial examinations won't help determine increased ICP
 - Used for monitoring only (and to direct other treatments)
 - For ventricles that are too small to access with external ventricular drain (EVD)
 - For patients with normal ICP but at risk of increased ICP
 - Potentially for coagulopathic patients
 - If ICP rises despite medical management, definitive management required
 - EVD or surgical decompression
- Use of an EVD (see Surgical Pearls, one through three)
 - Inserted through the skull and rests in the lateral ventricle
 - Monitors ICP and also removes CSF
 - Requires a CSF drainage unit
- Source control
 - Craniotomy for evacuation of space-occupying lesion/hematoma
- Decompressive craniectomy
 - Remove a large area of cranium and open the dura to allow intracranial volume expansion
 - Bone flap is left out and re-implanted weeks later
 - When cerebral volume has returned to normal
 - Studies have not conclusively shown a benefit
 - The alternative is death without decompression
 - But almost certain significant disability with

decompression

- Therefore, decompression is not automatic
 - Debate rages on
 - If it is done, it is better to do it early

Aneurysmal Subarachnoid Hemorrhage

Presentation

- Classic sign is the “thunderclap” headache
 - Acute onset of the worst headache (10/10) of a patient’s life
- May be accompanied by meningism
 - Irritation of the meninges from blood, manifesting as neck pain and stiffness
- May be accompanied by obtundation
 - Increased ICP, widespread damage from ischemia/hemorrhage, seizure
- Death before even reaching medical care as high as 10%
 - Overall case fatality is 40% (in a recent series)

Evaluation

- Unenhanced CT head is the initial test of choice
 - Will see subarachnoid blood (white/hyperdense) in most cases
 - If done using a third-generation scanner within 6 hours of the ictus
 - Sensitivity declines significantly after 12–24 hours
 - Distribution of blood depends on the location of aneurysm
 - Can also get intraparenchymal or intraventricular blood
- If > than 6 hours from presentation, and CT head negative, perform a lumbar puncture if safe to do so
 - Test is to detect CSF blood
 - Usually > 100,000 RBC/mm³ in SAH
 - If lower count/less discolouration in sequential tubes
 - Suggests traumatic tap rather than SAH
 - Look for xanthochromia
 - Yellow discoloration of CSF following centrifugation of cellular components
 - Due to breakdown products of hemoglobin
 - Decreased sensitivity (70% sensitivity) at 3 weeks
- If SAH is confirmed, or if CT scan/lumbar puncture negative but clinical suspicion is high
 - Perform a CT-angiogram (CTA)
 - To determine the presence and location of aneurysm
 - 97% sensitive in diagnosing an aneurysm
- Cerebral catheter angiogram is still the gold standard for ruling in, or ruling out, an aneurysm
 - Indicated if aneurysmal suspicion high and CTA negative
 - Also used to characterize the morphology of the aneurysm in planning treatment

Management Considerations

- Following a ruptured cerebral aneurysm, the goals are to prevent and treat three major causes of morbidity and

mortality:

1. Aneurysm re-rupture
 - Re-rupture most likely in the first 24 hours following initial rupture
 - Decreases but remains a concern for several months
 - Mortality is higher with re-ruptured aneurysms
 2. Hydrocephalus and increased ICP
 - May be acute (over hours/days) or chronic (over weeks/months)
 3. Vasospasm
 - Major cause of post-rupture brain ischemia/infarction
 - Onset day 3; may last for 3 weeks
- Also, there is the risk of seizures
 - Also, sodium abnormalities
 - Diabetes insipidus: high sodium
 - Syndrome of inappropriate anti-diuretic hormone (SIADH): low sodium

Medical Management of Unsecured Aneurysms

- Blood pressure management
 - Treating headache will often help in managing blood pressure
 - Target systolic blood pressure (SBP) <140 mmHg in patients with normal GCS or treated ICP (ventricular catheter)
 - Use labetalol/hydralazine
 - 5–20 mg IV q30 mins prn
 - Exercise caution in the obtunded patient
 - They may have elevated ICP
 - Therefore, aggressive BP lowering can compromise cerebral perfusion
- Sedation
 - Can prevent re-rupture by decreasing spikes in SBP
 - Can also be helpful in managing ICP
- ICP management
 - As outlined above
- Nimodipine for treatment of vasospasm
 - A calcium channel blocker shown to improve outcome
 - But no significant reduction in rates of radiographic vasospasm
 - Also, does not cure vasospasm once it has occurred
 - Patient should remain on this medication for 21 days
 - Or until discharge, whichever is sooner

Surgical/Endovascular Management (i.e. Securing an Aneurysm)

- If symptomatic hydrocephalus is present
 - Place an EVD for ICP control
- Surgical clipping
 - Involves craniotomy and surgical clipping
 - The clip is placed across the base of the aneurysm

- An open procedure, with associated surgical risk
- If successful, the risk of recurrence/re-bleed is effectively removed
- Endovascular treatment (“coiling”)
 - Via a catheter placed through the femoral artery
 - Metal coils then placed into the aneurysm under fluoroscopy
 - To occlude the lumen from the inside
 - Lower risk than open procedure
 - But higher risk of aneurysm recurrence
 - And may need long-term monitoring

Vasospasm

Definition/Presentation

- Delayed onset of focal neurological deficits due to narrowed cerebral vessels
 - Due to ischemia and often distal to location of spasm

Pathophysiology

- Spasm risk is proportional to the amount of blood in the SA space
 - An EVD does not remove blood from the SA space
 - Therefore, an EVD is to drain CSF and prevent hydrocephalus, not spasm
- Amount of blood is expressed by the Fisher Grade (FG)
 - FG1: no hemorrhage
 - FG2: <1 mm thickness in layering of blood on CT
 - FG3: >1 mm thickness in layering of blood on CT
 - FG4: intra-parenchymal blood/intra-ventricular extension
- Highest spasm risk is from post-rupture day 3 to day 14
 - However, spasm can occur (and reoccur) up to day 21
 - Beware: some patients have delayed presentation
 - Therefore, post-rupture day not always the same as post-admission day
- Clinical examination
 - Suspicion means the need for frequent neurological examination
 - Physical exam testing of the major arterial supplies
 - Face/arm weakness for middle cerebral artery
 - Leg weakness for anterior cerebral artery
 - Level of consciousness/cerebellar testing for posterior circulation
- Screening and confirmatory testing
 - Formal (dye) angiography remains the gold standard, but has increased risks
 - Risk of stroke (albeit low)
 - Also not practical for repeat testing
 - Transcranial Doppler (TCD)
 - Finicky (but safe) procedure: learn to love your TCD technician
 - Ultrasound performed via acoustic windows in the skull
 - Requires volume-based competence
 - Measures mean flow velocity (FV) (and also resistance)

- The trend in FV is more important than the absolute number
- ‘Normal’ <120 cm/s; ‘moderate’ spasm 120–200; ‘severe’ >200
- Abnormal TCDs numbers are confirmed by CTA, CT perfusion, angiography

Vasospasm Management

- Nimodipine is started on admission to *prevent* (not treat) vasospasm
 - Goal is to relax the smooth muscle of the cerebral vasculature
- Hyperdynamic (“Triple-H”) therapy previously used to *prevent and treat* vasospasm
 - “Triple H” refers to hypertension, hypervolemia, hemodilution
 - Only hypertension (i.e. with a noradrenaline infusion) shown to effectively treat vasospasm
 - Therefore, hypervolemia and hemodilution no longer widely recommended
- Intra-arterial drug injections to *treat* vasospasm
 - Vasodilators act locally at site of spasm to induce vessel wall relaxation
 - Papaverine, verapamil, milrinone
- Mechanical arterial dilatation to *treat* vasospasm
 - Cerebral balloon angioplasty
 - Performed in angiography suite with anesthesia back-up
 - Risky procedure:
 - Concerns of arterial occlusion or rupture
 - Families should provide informed consent

Intracranial Tumours

Basic Nomenclature

- “Intra-cranial” denotes anywhere within the bony cranium
- “Intra-axial” denotes within the brain parenchyma
- “Extra-axial” denotes outside the brain parenchyma
- “Intra-ventricular” denotes (you guessed it!) within the ventricular system

Common Tumours

- Most common *intracranial* tumour is the pituitary adenoma
 - Divided into microadenoma (< 1cm) or macroadenoma (> 1cm)
 - Also classified as functioning (hormone-producing) or non-functioning
 - Non-functioning microadenomas common but rarely require surgery
 - Usually just followed
 - Non-functioning macroadenomas
 - Require surgery if enlarging or causing neurologic symptoms

- Most common neurologic symptom is bi-temporal hemianopsia (no lateral vision on either side)
 - Due to pressure on the optic chiasm
- Functional adenomas require treatment according to the hormone they secrete
 - Options include medical therapy, radiation, and surgical excision
 - Prolactinomas are, by far, the most common and respond very well to medical therapy
 - Most commonly a dopamine agonist (bromocriptine, cabergoline)
 - Even large prolactinomas can be treated without surgery
- Next most common is the meningioma
 - Homogenous enhancing dural-based extra-axial tumour leading to mass effect
 - In everyday English: grows under the dura, then presses on the brain
 - 20% of all intracranial tumours
 - Present in 3% of autopsies in patients aged over > 60 years
 - Typically benign
 - Therefore, can become large before symptoms of mass effect noted
 - Good prognosis with complete excision
- Most common intra-axial tumour is a metastatic tumour
 - Often multiple lesions present on imaging
 - Common sites of primary lesion (in order of prevalence):
 - Lung, breast, melanoma, GI tract, renal
 - Tumour of origin predicts how aggressive/treatable a cerebral metastasis is
 - Oncology (not neurosurgery) manages the majority of workup and treatment
- Most common primary *intra-axial* tumour is an astrocytoma, the most aggressive form of which is glioblastoma multiforme (GBM)
 - An aggressive glial tumour arising most commonly from astrocytes
 - Therefore, also known as a Grade IV Astrocytoma
 - Appearance can be similar to metastatic lesion, though rarely more than one present
 - Typically requires surgical resection and adjunct chemo/radiation therapy
 - Prognosis is not good
 - About 40% die within one year
 - About 80% die within three years

Surgical Pearl 1: Troubleshooting a Blocked EVD

- Ensure all stopcocks are open to the collection bag
- Ensure no disconnections or breaks have occurred
- Drop the collection system to the floor
 - To see if the drain is patent and rule out that flow stopped because of low ICP
 - If patent, then CSF drains when the bag is lowered
- Working from most proximal (patient) port, you flush distally (towards the bag)
 - Flush particulate matter through the tubing toward collection bag
 - Use sterile saline
 - Observe for flow after each flush
- If all distal flushes fail
 - Call neurosurgery to consider a proximal flush
 - With the stopcock open to the patient
 - Neurosurgery may attempt to aspirate
 - They may gently inject a few mLs sterile saline through the proximal port
 - Then, once again, check for flow
- If no flow after any of the above and EVD still necessary
 - Consider removing drain/inserting a new catheter and repeating the CT
 - Neurosurgery definitely should be involved by this point

Surgical Pearl 2: Removing Cranial Catheters

- Ensure all stopcocks are closed to the patient and that CSF is not flowing
- Apply sterile gloves and clean/drape area
- Infiltrate the exit site with lidocaine
 - This step is not always necessary, particularly if only one stitch to be placed
- Cut the sutures that hold the drain in place
 - Then remove the catheter
- Close the incision/exit site with 1 or 2 interrupted sutures
- If patient is awake, have them do a Valsalva and observe for any CSF leakage from exit site
 - Apply another stitch if any leakage observed

Surgical Pearl 3: Inserting an EVD (a right frontal approach is usual to minimize damage)

(Requires experience and precise positioning: usually reserved for neurosurgery)

Equipment: Personal protective equipment, sterile towels, EVD tray and drill, hair clippers, ICP transducer, sterile occlusive dressing (Tegaderm®), suture, IV pole.

Preparation: Head is elevated to 30 degrees, immobilized, and in a neutral position. Hair is clipped and scalp is infiltrated with lidocaine plus epinephrine.

To determine the site at which to drill: Draw a backwards line 11 cm from the nasion in the mid-line (nasion = top of the nasal bone, just under the brow ridge). Then draw a lateral line 3 cm from midline and make a mark. This is Kocher's point (KP): just anterior to the coronal suture and in the midpupillary line.

To determine where to aim the catheter: Draw a line from KP to the ipsilateral medial canthus. Draw a second line from KP to 1 cm anterior to the ipsilateral tragus.

Insertion technique:

- 2-cm scalpel incision at KP and down to the skull
- Clear the periosteum and insert a small retractor

- Use a manual twist drill aimed perpendicular to the skull (to penetrate the skull)
- A stop guard prevents plunging into the brain parenchyma
- Introduce a probe/18-gauge needle to ensure the drill penetrated the bone
- Use probe/18-gauge to score and puncture the dural surface
- Direct catheter toward the ipsilateral medial canthus (in sagittal plane) AND 1 cm anterior to the tragus (in the coronal plane)
- Catheter should be advanced 5 cm below the dura (6–7 cm below the skull surface)
- This will place the tip of catheter close to the foramen of Monro
- A "pop" is felt at about 3–4 cm, indicating entry into the ventricle
- Egress of CSF also confirms entry and the height of the column approximates ICP

Set up the drain:

- Distal end is tunnelled under the scalp posteriorly and laterally (to decrease infection)
- This is secured with 2-0 nylon suture, connected to the EVD drainage system
- Then the EVD is zeroed at the tragus, and (usually) left open to 10 cm (to allow drainage, but not over drainage)

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