Please, if you think that an answer is wrong, spread the word!

The questions which are ~~struck-through~~ are answered in the previous draft.

1. **All are signs of basal skull fracture except:**
   A. Battle sign
   B. CSF rhiorrhea
   C. Epistaxis
   D. Blindness
   E. Rakoon eyes

   ~~*<had el so2al etkarar marteen i think eno blindness el jawab>*~~

   Anterior Fossa Fracture - anosmia, epistaxis, rhinorrhea, subconjunctival hemorrhage, periorbital hemorrage (raccoon eyes, visual disturbances, altered eye movement, ptosis, loss of sensation to forehead, cornea and nare
   Middle Fossa Fracture - loss of sensation to lower face, ottorrhea, deafness, tinnitus, facial palsy, hemotympanium
   Posterior Fossa Fracture - echymosis behind the ear (battle sign), impaired gag reflex
   Catastrophic injuries can occur if there is a major disruption of the carotid artery (blood supply to middle and anterior cerebral cortex) or vertebral artery (blood supply to brainstem and posterior cerebral cortex), or if the brain stem is disrupted.
   http://www.lhsc.on.ca/Health_Professionals/CCTC/edubriefs/baseskull.htm

   Ans: Blindness

2. **7 years old boy, history of otitis media 2 weeks ago presented with headache and cerebellar signs were positive which is true:**
   A. give antibiotics
   B. Do CT scan ➔ ana 7a6eto had> ba2e el 5aearat mo mzakert-hom???

????

3. **nafs so2al 2 bs 3'aear el 5aearat la which most likely diagnosis?**
   a. i think medaloblastoma el jawab kan>

4. **The most prognostic sign in acute head injury is :**
   A. CT scan

????

5. **Methods to decrease the ICP except:**
   a. elevate the pt head
   b. hyperventilation

c. allow hypotension
d. < i cant remember el ba2e

Ans: According to Toronoto notes: For the treatment of elevated ICP ➔ "Prevent hypotension" page 6.

6. Nafs so2al 5 bs with head injery :

a. <kan 7a6 nafs el 5aearat w kolha bte3mal decreasing in the ICP bs i think eno u can't elevate the head l2no bt5af mn spine fracture>???

Ans: Correction: According to Toronoto notes, "elevate head of bed at 30-45 degrees, maintain neck in neutral position".

7. In depressed head fracture what indicate sergury:

a. the fracture have sharp edges -contaminated fracture and sth -no csf leak <i think bel contamenated coz its need depriment bs et2akad mnha>

8. eja 3an el epidural hematoma it was easy el classical characteristics

Table 6. Comparison of Epidemiology and Etiology of Intracranial Bleeds

Types of Hematoma/ Hemorrhage	Etiology	Epidemiology	Clinical Features	CT Features	Treatment	Prognosis
Epidural Hematoma	Skull fracture causing middle meningeal bleed	Male > Female (4:1)	Lucid interval before LOC	Lenticular mass	Craniotomy	Good with prompt management (Note: respiratory arrest can occur from uncal herniation)
Acute Subdural Hematoma	Ruptured subarachnoid bridging vessels	Age >50, associated with trauma	No lucid interval, hemiparesis, Pupillary changes	Crescentic mass	Craniotomy if bleed >1 cm	Poor
Chronic Subdural Hematoma	Ruptured subarachnoid bridging vessels	Age >50, EtOH abusers, anti-coagulated	Often asymptomatic Minor H/A, confusion, signs of increased ICP	Hypodense crescentic mass	Burr hole to drain; craniotomy if reoccurs	Good
Subarachnoid Hemorrhage	Trauma, spontaneous (aneurysms, idiopathic, AVM)	Age 55-60 20% cases under age 45	Sudden onset thunderclap headache, signs of increased ICP	High density blood (sensitivity decreases over time)	Conservative: NPO, IV NS, ECG, Foley, BP 120-150, vasospasm prophylaxis (nimodipine); open vs. endovascular surgery to repair if rebleed	Poor: 50% mortality 30% of survivors have moderate to severe disability
Intracerebral Hemorrhage	HTN, vascular abnormality, tumours, infections, coagulopathy	Age >55, male, drug use (cocaine, EtOH, amphetamine)	TIA-like symptoms, signs of increased ICP	High density blood	Medical: decrease BP, control ICP Surgical: Craniotomy	Poor: 44% mortality due to cerebral herniation

9. Extradural hematoma true except:

a. -the most affected artary is the middle meningeal - have lucid interval -the ttt. is evacuation -icant remember the answer

Ans: All the options above are true

~~**10.** which of the antibiotics cross the BBB without infection:~~

~~a. -penicillin - other2 -noneof the above~~

11. Sth about in meningitis except:

a. el 5aearat kanat kolha sa7 3ada eno -u don't give antibiotics until confirming the type of the bacteria < this is wrong >
   Ans: In meninigities, you start with empirical therapy until the results of C&S are back.

12. Most commen bacterea that coz shunt infections?

a. Ans: Staph species
b. If the doctor did not specify: Then staph species are the most common
c. If he wanted which species of staph. There is no consensus. However, there is more tendency toward coagulase negative staph. This book reports coagulase negative staph.
   i. http://books.google.jo/books?id=Gx8G3WUJ24sC&pg=PA1141&lpg=PA1141&dq=the+most+common+organism+shunt+infection+coagulase+negative+staph+aureus&source=bl&ots=APB3NZzDqm&sig=PUtakCTyvu1BG5vkJyWBJzwXTyQ&hl=en&sa=X&ei=riqwT5yWC86XhQer5bTzCA&redir_esc=y#v=onepage&q=the%20most%20common%20organism%20shunt%20infection%20coagulase%20negative%20staph%20aureus&f=false
   ii. The first ten google results report coagulase negative staph https://www.google.com/search?q=the+most+common+organism+shunt+infection+staph+aureus#hl=en&newwindow=1&safe=off&sclient=psy-ab&q=Coagulase-negative+staphylococci+the+most+common+cause+of+shunt+infection&oq=Coagulase-negative+staphylococci+the+most+common+cause+of+shunt+infection&aq=f&aqi=g-w1&aql=&gs_l=serp.3..33i21.248303.254098.2.254260.41.34.0.0.0.2.1024.10713.2-15j9j4j2j0j1.31.0...0.0.Gir3i3kmFcQ&psj=1&bav=on.2,or.r_gc.r_pw.r_cp.r_qf.,cf.osb&fp=e4b5fcdc68cea366&biw=1024&bih=637

الزبدة:

coagulase negative staph but not 100% consensus!...
90%

Infection of cerebrospinal fluid shunts: causative pathogens, clinical features, and outcomes. Wang KW, Chang WN, Shih TY, Huang CR, Tsai NW, Chang CS, Chuang YC, Liliang PC, Su TM, Rau CS, Tsai YD, Cheng BC, Hung PL, Chang CJ, Lu CH. Source Department of Neurosurgery, Chang Gung Memorial Hospital-Kaohsiung, Taiwan.

Abstract

This retrospective chart review describes the clinical features, pathogens, and outcomes of 46 patients with cerebrospinal fluid (CSF) shunt infections collected over 16 years. The overall CSF shunt infection rate was 2.1%, broken down into 1.7 and 9.3% in adult and pediatric groups, respectively. Fever and progressive consciousness disturbance were the most clinical features in the adult patient group, whereas disturbance of consciousness and abdominal symptoms and signs were the two most common clinical features in the pediatric patient group. **The most frequently isolated microorganisms were of the Staphylococcus spp**., including Staphylococcus aureus and coagulase negative Staphylococcus, which accounted for 47% of the episodes. Furthermore, increases in polymicrobial and Gram-negative bacilli infections were observed in our study. Due to the high proportion of oxacillin-resistant Staphylococcus spp. and polymicrobial infections, we recommend initial empirical antibiotics with both vancomycin and a third-generation cephalosporin for cases in which the causative bacteria has not been identified or for which the results of antimicrobial susceptibility tests are not available. For patients who develop smoldering fevers, progressive disturbed consciousness, seizures, or abdominal fullness after ventriculoperitoneal shunt procedures, CSF shunt infections should be suspected. Although some infections have been managed successfully with antimicrobial therapy alone, the timely use of appropriate antibiotics according to antimicrobial susceptibility testing and the removal of the shunt apparatus are essential for successful treatment.

13. The treatment of shunt infections all except:

a. -systemic antibiotics -intraventricular antibiotics -immediate removal of the shunt -replacment of the shunt -extraventicular shunting
b. < mo met2kda l2no ma a36ana margr3 nedros meno w ma shara7o>

Aggressive management of shunt infection: combined intravenous and intraventricular antibiotic therapy for twelve or less days.

James HE, Bradley JS.

Source

Lucy Gooding Pediatric Neurosurgery Center, University of Florida HSC/Jacksonville and Wolfson Children's Hospital, Jacksonville, FL 32207, USA. lgpncjax@yahoo.com

Abstract

OBJECTIVE:

This report is limited to patients with a single cerebrospinal fluid (CSF) shunt infected by a single organism, and compares two treatment protocols.

METHODS:

In the initial protocol (1975-1991), patients underwent removal of the shunt system and received intravenous and intraventricular antibiotics. Intraventricular antibiotics were administered twice daily to those with external ventricular drainage. When CSF was cultured 48 h off all antibiotics and found to be sterile at 24 h of incubation, a new shunt was inserted. Follow-up CSF cultures were obtained in all patients between 1-6 months following placement of the new shunt.

RESULTS:

There were 25 patients (ages 1 month to 16 years; mean +/- SD: 23 +/- 4.0 months). CSF obtained from the shunt yielded the following: Staphylococcus epidermidis (19), Staphylococcus aureus (2), Streptococcus species (2), Serratia marcescens (1), and Propionebacterium species (1). The duration of intravenous antibiotics was 7-12 days (mean +/- SD: 9.7 +/- 1.3 days), and intraventricular antibiotic therapy was 6.2 +/- 1.7 days. Total hospital stay was 15.2 +/- 2.3 days. The follow-up period was 7.7 +/- 3.6 years. Following the initial protocol in another 15 patients (1992-2004), the treatment regime was modified in that intraventricular antibiotics were administered once daily in patients with external ventricular drainage, and the CSF was cultured at 24 h off antibiotics, instead of 48 h. Results were similar to the initial protocol with respect to days of antibiotic therapy and hospital stay.

CONCLUSION:

Based on our retrospective nonrandomized series, we believe patients with a single shunt and noncompartmentalized hydrocephalus can be successfully treated without a

prolonged antibiotic course and lengthy hospital stay

than firm data.[20,59] First, infections can be treated nonoperatively, with systemic or intrashunt installation of antibiotics. This approach has a significant failure rate, however; in one prospective study, only 3 of 10 patients treated with intravenous and intrashunt antibiotics for 2 to 3 weeks cleared the infection.[58] Shunt tubing has been shown by scanning electron microscopy to contain irregularities to which bacteria can adhere;[60] thus, removing shunt hardware eliminates persistent contamination that can cause recurrent infection. Because of the high failure rate of nonoperative treatment, this strategy is often reserved for patients who have had multiple prior surgeries, complex shunts, or other circumstances that might increase surgical risk.

A second strategy is treatment with antibiotics, sometimes with exteriorization of the distal catheter. This maneuver, which is most often used if there are peritoneal symptoms, is usually performed at the bedside through a small skin incision over the course of the shunt along the chest wall. The distal catheter can thus be removed from the abdomen and thereby can drain the infected CSF to an external collecting system. Once the infection has cleared, as ascertained by serial cultures, the exteriorized shunt is totally removed and replaced with a new, sterile shunt.

The final strategy includes early removal of all shunt hardware, placement of a sterile temporary external ventricular drain (ventriculostomy), followed by replacement of a new shunt system when cultures remain negative. This approach obligates the patient to two surgical procedures with anesthesia, but theoretically gives the best chance of eliminating the infection by removing all contaminated hardware early in the treatment course. This may ultimately shorten hospital stay compared to less effective treatment plans.[20] In some cases, persistently positive cultures from a temporary ventriculostomy will be handled by instilling intrathecal antibiotics. In other cases the ventriculostomy will be replaced surgically to try to eliminate a potential source of reinfection if the CSF does not clear promptly. The addition of synergistic antimicrobial agents, such as rifampin in the case of persistent infections treated with vancomycin, may also be helpful in the setting of persistently positive cultures. It should be noted that the gram stain may remain positive for bacteria even when these are no longer viable; this distinction can be made because the cultures will remain negative despite persistently positive stains. The rate of repeated infections in patients treated with antibiotics plus shunt removal, in either a one-stage or a two-stage procedure, has been reported to range from approximately 5% to 20%, depending on the specific methods used for analysis.[20,58]

Apart from general agreement that removal of the shunt leads to a higher rate of clearing the infection, exactly which regimen should be undertaken under which circumstances remains a matter of physician judgement with few firm data for guidance. Available studies generally suffer from small sample sizes and other methodologic limitations.[20,58] In addition, the optimal duration of treatment is not known, and surgeons vary widely in their timing of shunt replacement after treatment of infection. In a survey of pediatric neurosurgeons regarding their practices in treating shunt infections, the duration of antibiotics for similar clinical sce-

there is no consensus! عادي شاطر وطلع إجابة...

14. In the word the pt was stable and GCS was 15/15 in the beginning of the round and in the end of it the pt was unconscious what is likely to be the coz:

a. -seizure -brain edema -meningitis -cranial vasospasim <ana 7a6et-ha seizure >

Ans: Seizure (previous years questions)

~~**15.** Ffoot drop with inversion weakness what is the affected :~~
~~-common peronial - superfical peronial - deep peronial - femoral nerve -l4 l5 radiculopathy <7a6eet el a5era>~~

~~Ans: A~~

~~**16.** All coz seizures except:~~
~~a. -ms -mass in the brain < i can't remember the choices but it was easy>~~

17. Mass effects on the brain except:
a. -headache -nerve palsy -------

Ans: Both of the above are correct!

~~**18.** Tuft hair on the back of a baby all true except:~~
~~a. -no neurological deficit usually - do ct scan - ma bazakar el ba2e~~

19. Complication of lumber disc surgery except:
a. gynecological injury -not the level of the affected disc - hit an artery

Ans: No reference of the below mentions "Gynecological injury". Mistake in the level of affected disc is mentioned in one site. So the wrong answer is "gynecological injury".

Five patients presented with acute life-threatening **iatrogenic haemorrhages from pelvic vessels**. Three patients made a complete recovery, one patient died from **acute haemorrhagic shock**, one further patient died from **sepsis** due to an associated complication — an **injury to the ureter.**
Over a period of two to ten years after primary surgery we corrected late complications such as 1 case of **posttraumatic aneurysm of the aortic bifurcation** found to have eroded the body of the fifth lumbar vertebra, and three cases of **arteriovenous fistula** between the common iliac artery and the common iliac vein.
The four cases described below are an attempt to document the vascular surgical procedures involved and to provide typical findings.
The risk of injuring the pelvic vessels intra-operatively can be explained by the close anatomical relation between the retroperitoneal vessels and the vertebral column and furthermore not only by the fact that pre-existent deficiencies but also injury to the anterior longitudinal ligament give access to the retroperitoneal space.

http://www.springerlink.com/content/j4680003683m6v35/

Other risks of surgery include spinal fluid leaks, bleeding, and infection. All of these can usually be treated, but may require a longer hospitalization or additional surgery.

The most common problem of a discectomy is that there is a chance that another fragment of disc will herniate and cause similar symptoms down the road. This is a so-called recurrent disc herniation, and the risk of this occurring is about 10-15%.
http://orthopedics.about.com/cs/herniateddisk/a/ruptureddisk_3.htm

Complications

- All types of spine surgery, including artificial discreplacement, have certain risks and benefits
- Complications from any kind of surgery can also occur from anesthesia, infection, and bleeding
- Medical complications arising from spinal surgery are rare but could include stroke, heart attack, spinal cord or spinal nerve injury, pneumonia, or possibly death

This list is not exhaustive and only includes the most common / serious potential complications.

- Thrombophlebitis
- Infection
- Nerve damage or paralysis
- Spontaneous ankylosis (fusion)
- Retrograde ejaculation (men only)
- Ongoing pain
- Problems with the implant. Long-term studies to assess the safety and effectiveness of artificial disc replacements are ongoing. So far results show a low rate of complications

http://www.singhealth.com.sg/PatientCare/ConditionsAndTreatments/Pages/Lumbar-Disc-Replacement-Surgery.aspx

- No surgery is absolutely safe and free of complications
- Some of the possible complications of lumbar disk surgery are:
    1. Untoward effects of anesthesia
    2. Bleeding or hemorrhage with the possible need for blood transfusions
    3. Nerve root injury that could result in paralysis, loss of feeling, or loss of bowel and bladder control
    4. Infection
    5. Tear in the covering of the nerves with leaking of cerebrospinal fluid
    6. Injury to blood vessels
    7. Injury to bowel or ureters
    8. The possibility of unforeseen complications

http://www.yoursurgery.com/ProcedureDetails.cfm?BR=2&Proc=34`

FREQUENCY OF THE MOST COMMON COMPLICATIONS: Postoperative mortality: < 0.1%; total morbility: 4-10%; general complications: urinary infection (after exploration) < 10% and thromboembolism < 2%; neurological complications: neurological deterioration 0-7%; lesion of the dura mater 1-5% (greater in repeated operations), CSF fistula < 1% and cauda equina syndrome 2-5%; vascular, ureteral and visceral complications: possible; mistake in the level operated on: 3%; complications in the surgical wound: haematoma (possible); infection < 10%; need for another operation: 4-11 %; other complications: possible. Complications are more common in the case of osteosynthesis. (Pareras LG, González-Feria L. Complicaciones en la cirugía de la columna (II): cirugía de la columna lumbar. Neurocirugía XXI, 2: 214-230, 1996.)

20. L4-L5 disc prolapse true except:

a. affected the nerve of that level
b. decreasing the pain when tilting to the opposite side
c. treated conservatively

Ans:

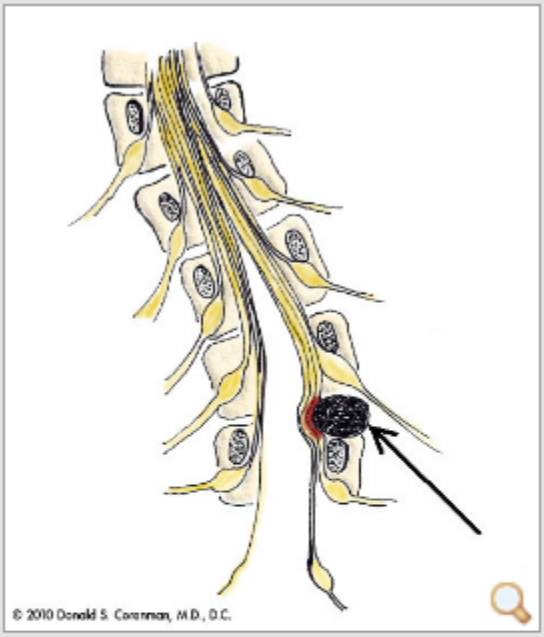
A. Tilt is to opposite side of pathology to reduce pain ➔ Correct

www.krishnaraman.com/pdf/**Lumbardisc**prolapse.pdf

The patient with a one-sided disc herniation may list or lean away from the side of the sciatica
http://mededucation.bjmu.edu.cn/reportreference/reference/Evaluation%20&%20Mgmt%20of%20Back%20Pain.htm

Leaning AWAY from the pain?

The second common disc injury is called the Postero LATERAL herniation, and you lean in the opposite direction. AWAY from the pain. It needs to be managed quite differently... but both demand a life long commitment to a daily exercise programme. Or suffer...
http://www.chiropractic-help.com/Postero-Medial-Disk-Herniation.html



This is a picture of an antalgic scoliosis of the lumbar spine. The patient unconsciously leans away from the herniated disc (arrow) to take pressure off of the nerve root. Compare this with the normal diagram of the spine with nerve roots above.

B. Treatment: Conservative (Toronoto) [Indication for surgery from toronoto notes: intractable pain despite adequate conservative treatment for > 3months, progressive neurological deficits, and cauda equina syndrome]

C. **Root involved is L5 not L4 ➔ Is this the answer?**

21. -indication for desc operation except:

Read above!

22. Kan fe cases w es2al which area is likely to be the lesion

- **Frontal lobe lesions** tend to present with personality change, gait ataxia and urinary incontinence, contralateral hemiparesis if posterior frontal and dysphasia if involving the left inferior frontal gyrus.
  - Also in the front love, center for visual field??? In middle frontal gyrus???
- **Parietal lesions** are associated with sensory inattention, dressing apraxia, astereognosis and, if on the dominant side, acalculia, agraphia, left–right disorientation and finger agnosia (Gerstmann's syndrome).
- **Temporal lobe lesions** may be associated with disturbance of memory, contralateral superior quadrantanopia or hemiparesis and, if on the dominant side, dysphasia.
- **Occipital lesions** are often associated with visual field deficits, most commonly an incomplete contralateral homonymous hemianopia.
- **A pituitary macroadenoma:** bitemporal hemianopia
- **Anterior skull base meningioma:** anosmia, ipsilateral optic atrophy and contralateral papilloedema (Foster–Kennedy syndrome)
- **Vestibular shawannoma:** ipsilateral hearing loss, tinnitus and dysequilibrium

23. So2al 3an all of are early complication in head injury except:

a. -epilepsy -brain edema - csf leak

Ans : brain edema, according to Medscape, is a type of secondary injuries. Secondray injuries, according to them as well, require hours to days to develop. Could it be the answer?

24. Causes of sec. brain injery except:

a. counter coup injury -hematoma - brain shifting -el ba2e kano sa7

Ans: Accoring to Medscape, "Coup and contrecoup" & "hematomas" are primary brain injury and edema, hydrocephalus, increased ICP & brain herniation is secondary brain injury. ????

In another website, "hematoma" is considered to be both primary and secondary. Therefore, the answer of the above question is probably "countercoup" injury which can never be secondary!

Types of Primary Injuries

Primary injuries can manifest as focal injuries (eg, skull fractures, intracranial hematomas, lacerations, contusions, penetrating wounds), or they can be diffuse (as in diffuse axonal injury).

Pathophysiology: Secondary Injury

Secondary types of traumatic brain injury (TBI) are attributable to further cellular damage from the effects of primary injuries. Secondary injuries may develop over a period of hours or days following the initial traumatic assault.

Secondary brain injury is mediated through the following neurochemical mediators[15]:

Excitatory amino acids

Excitatory amino acids (EAAs), including glutamate and aspartate, are significantly elevated after a TBI.[16]

EAAs can cause cell swelling, vacuolization, and neuronal death.

EAAs can cause an influx of chloride and sodium, leading to acute neuronal swelling.

EAAs can also cause an influx of calcium, which is linked to delayed damage. Along with N-methyl-D-aspartate receptor agonists, which also contribute to increased calcium influx, EAAs may decrease high-energy phosphate stores (adenosine 5'-triphosphate, or ATP) or increase free radical production.

EAAs can cause astrocytic swellings via volume-activated anion channels (VRACs). Tamoxifen is a potent inhibitor of VRACs and potentially could be of therapeutic value.

Endogenous opioid peptides

These may contribute to the exacerbation of neurologic damage by modulating the presynaptic release of EAA neurotransmitters.

Activation of the muscarinic cholinergic systems in the rostral pons mediates behavioral suppression, which often is observed in TBI, as well as LOC.

Heightened metabolism in the injured brain is stimulated by an increase in the circulating levels of catecholamines from TBI-induced stimulation of the sympathoadrenomedullary axis and serotonergic system (with associated depression in glucose utilization[17]), contributing to further brain injury.

Other biochemical processes leading to a greater severity of injury include an increase in extracellular potassium, leading to edema; an increase in cytokines, contributing to inflammation; and a decrease in intracellular magnesium, contributing to calcium influx.

Based on the effect on astrocytes, which are the cells that exhibit hypertrophic and hyperplastic responses to central nervous system (CNS) injury, increased production of protein kinase B/Akt with activation of P2 purinergic receptors has been implicated in neuronal survival in TBIs.[18]

Increased intracranial pressure (ICP)

The severity of a TBI tends to increase due to heightened ICP, especially if the pressure exceeds 40 mm Hg. Increased pressure also can lead to cerebral hypoxia, cerebral ischemia, cerebral edema, hydrocephalus, and brain herniation.

Cerebral edema

Edema may be caused by the effects of the above-mentioned neurochemical transmitters and by increased ICP. Disruption of the blood-brain barrier, with impairment of vasomotor autoregulation leading to dilatation of cerebral blood vessels, also contributes.

Hydrocephalus

The communicating type of hydrocephalus is more common in TBI than is the noncommunicating type. The communicating type frequently results from the presence of blood products that cause obstruction of the flow of the cerebral spinal fluid (CSF) in the subarachnoid space and the absorption of CSF through the arachnoid villi. The noncommunicating type of hydrocephalus is often caused by blood clot obstruction of

blood flow at the interventricular foramen, third ventricle, cerebral aqueduct, or fourth ventricle.

Brain herniation

Supratentorial herniation is attributable to direct mechanical compression by an accumulating mass or to increased intracranial pressure.[19] The following types of supratentorial herniation are recognized:

- Subfalcine herniation - The cingulate gyrus of the frontal lobe is pushed beneath the falx cerebri when an expanding mass lesion causes a medial shift of the ipsilateral hemisphere. This is the most common type of herniation.
- Central transtentorial herniation - This type of injury is characterized by the displacement of the basal nuclei and cerebral hemispheres downward while the diencephalon and adjacent midbrain are pushed through the tentorial notch.
- Uncal herniation - This type of injury involves the displacement of the medial edge of the uncus and the hippocampal gyrus medially and over the ipsilateral edge of the tentorium cerebelli foramen, causing compression of the midbrain; the ipsilateral or contralateral third nerve may be stretched or compressed.
- Cerebellar herniation - This injury is marked by an infratentorial herniation in which the tonsil of the cerebellum is pushed through the foramen magnum and compresses the medulla, leading to bradycardia and respiratory arrest.

http://emedicine.medscape.com/article/326510-overview#showall

Primary and Secondary Damage

Primary Damage

Damage that occurs at the time of actual impact. There are many types of Primary Damage that may occur. These include:

1. Skull Fracture- breaking of skull bone
2. Contusion/Bruise- discoloration and/or swelling at the location of actual impact or at the point or points where the force of the blow has driven the brain against the skull's bony ridges
3. Hematoma/Blood Clot- swelling or mass of blood between the skull and the brain or inside the brain itself
4. Laceration- tearing of brain tissue and/or blood vessels, caused by forceful rotation of the brain across the skull's bony ridges
5. Nerve Damage (Diffuse Axonal Injury)- shearing or tearing of white matter in connecting nerve fibers in the brain; can cause unconsciousness and/or coma

Secondary Damage

Damage that occurs over time after the actual brain injury; may include infection, hypoxia (oxygen deprivation), edema (brain swelling), elevated intracranial pressure, infraction (death of brain tissue which results in loss of blood supply to that region of the brain), and hematoma (focal area of bleeding in the skull due to tearing of blood vessels)

Many traumatic brain injuries result in multiple types of primary and secondary damage.

http://www.northeastern.edu/nutraumaticbraininjury/what-is-tbi/types-of-damage/

25. fe so2al preoperative what to give to pt. with increased ICP:

a. -diuretics -diuretics then b-blokers -diuretics then alph bloker - non < mo mezakra ba2e el 5aearat>

You give:

b. Mannitol
c. Prevent hypotension with fluid and vasopressors, dopamine, norepinephrine
d. Therefore, you should not give neither beta blockers nor alpha-blockers
e. None of the above!

Examples of vasopressors:

* * Dopamine
* Vasopressors and Inotropes * Dobutamine
* Adrenaline * Dopexamine
* Ephedrine * Salbutamol
* Methoxamine * Isophrenaline
* Metaraminol * Phosphodiesterase Inhibitors, e.g. aminophylline
* Phenylephrine * Clinical Study

~~**26.** all operation to do in hydrocephalus except~~

a. rakaz 3al shunt infections wel head injuries w kan fe as2la m3loomat 3ama