Cranial Trauma Procedures

Last updated: December 19, 2020

GENERAL PRINCIPLES

INDICATIONS FOR SURGERY

See p. TrH1 >>

PREOPERATIVELY

- correct ***coagulopathy*** - fresh frozen plasma (for prolonged PT/aPTT), cryoprecipitate (for fibrinogen < 1.5 g/L), thrombocyte transfusions (for platelets < 100,000).
- add **antiepileptic** (AED)
- **antibiotic** prophylaxis.
- **tetanus immunization** status should be checked and updated (esp. lacerations, contaminated wounds).
- if suspect vascular lesion (e.g. young person with deep bleed):
    1) order **CTA** preop
    2) plan craniotomy so will have *proximal control* (e.g. by dissecting Sylvian fissure).
    3) start evacuating blood clot ***farthest from suspicious area*** and may leave small clots.

ANESTHESIA

- even if patient is in coma it is unwise to begin without full anesthetic support (only exception is patient thought likely to expire during time taken to organize these precautions, which can usually be done while theater is prepared).
- if head is rotated for surgery:
    - make sure neck veins are not twisted.
    - head above heart level (if BP permits).
    - *3-point head fixation device* is used if unstable C-spine fracture is present but careful with skull fractures.
- ***drape*** to allow extension of surgical incision beyond actual confines of wound, for EVD ad drain exit, or to allow possible scalp rotation procedures.
- patients with high ICP are very vulnerable to incidents of respiratory obstruction, hypercarbia, systemic hypotension.

    N.B. once intracranial hematoma begins to be removed blood pressure may fall precipitously (esp. if multiple injury has produced hypovolemia, masked by effects of raised ICP).
- ICP monitor (or ventricular drain) usually is placed intraoperatively in patients with GCS ≤ 8.

TECHNIQUE

- whole head is shaved (e.g. for placement of ICP monitor on contralateral side).
- never make small trauma craniotomy (brain may swell and may need to leave bone flap out); big trauma flap is never wrong choice (postoperative brain swelling may strangulate brain against tight craniotomy bone edges).
- Dr. Mathern likes ***dural tuck-ups*** placed before opening dura (if time permits) – place stitch under bone – helps with hemostasis (during the case) and dural closure!
- after evacuating mass lesion, **leave ICP monitor in** (even if removed bone flap).
- send selected removed blood **clots for pathology** (may find tumor, vascular lesion).
- **malignant cerebral edema** – *see below >>*

POSTOPERATIVE

- CT is obtained ≤ 24 hours postoperatively.

COMPLICATIONS

Infection (cranial osteomyelitis, subdural empyema, meningitis, brain abscess).
- risk factors – open scalp fractures, violated paranasal sinuses (esp. with CSF leak).

- prophylaxis – place vancomycin powder into surgical bed at the end of the case.

SCALP WOUNDS

SCALP ABRASIONS

- often contaminated with pieces of dirt - should be thoroughly cleaned and inspected for puncture wounds to ensure removal of unsuspected foreign bodies.

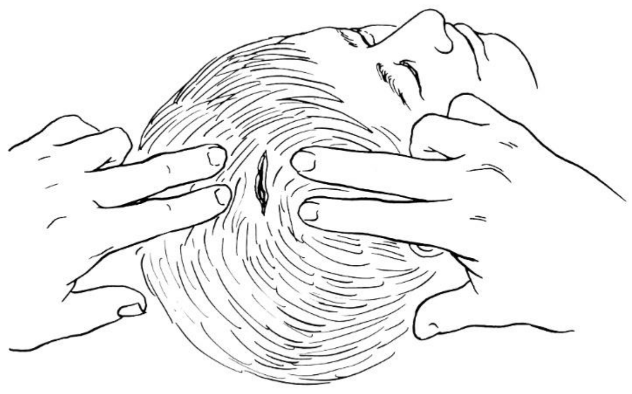
SCALP LACERATIONS

Because of scalp's ***rich vascular supply***:

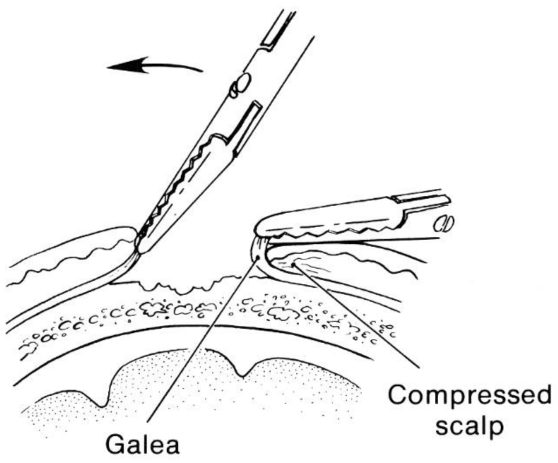
1) scalp lacerations may be source of significant bleeding.
2) most uncomplicated lacerations can be closed (after cleansing and debridement) and antibiotics are usually not needed.
3) even very large scalp avulsions can survive:
   - if avulsion remains attached to rest of scalp by *tissue bridge*, it should be reattached to surrounding tissue.
   - if avulsion is *completely detached* from scalp it should be treated as any other amputated part and reimplanted ASAP.
   - *small scalp deficit* is repaired by rotating portion of scalp.
   - *large scalp deficit* requires skin graft or vascularized free flap.

HEMOSTASIS methods:

1) direct *digital compression* of bleeding vessel against skull; skalpo kraujagyslės išsidėstę tarp dviejų tamprių sluoksnių - odos ir aponeurozės (galea):
   a) *pažeista tik oda* - kraujavimą lengva sustabdyti spaudžiamuoju tvarsčiu.
   b) *pažeista oda ir galea* - žaizda plačiai žiojėja, o iš jos kraštų profūziškai kraujuoja (kartais nepastebimai iki didelio kiekio į uždėtą storą tvarstį) - kraujavimą galima sustabdyti tik stipriai prispaudžiant žaizdos kraštus prie kaulo; to naudoti negalima, jei yra nestabilus (nelinijinis) lūžimas ties žaizda - tokiu atveju prispaudžiama prie kaulo *a. temporalis* (virš arcus zygomaticus).



2) *infiltration of wound edges* with LIDOCAINE + EPINEPHRINE.
3) *ligation* of identified bleeding vessels (likely futile maneuver except when very large vessels are found to be injured).
4) if *galea* is lacerated, it can be pulled up with clamp and its *edges folded over* lacerated skin edges to tamponade bleeding vessels:

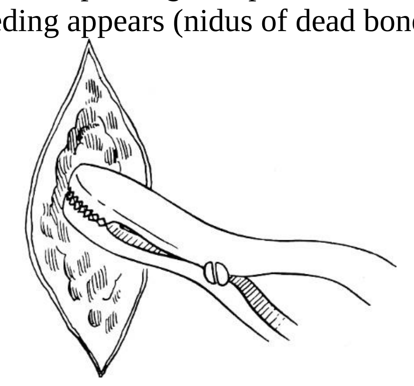


5) ***Raney scalp clips*** (plastic - do not interfere with CT) - easy to apply rapidly to wound edges - useful in unstable patients.
6) definitive method - *wound closure* after proper debridement and irrigation.

Once hemostasis is obtained, wound is copiously irrigated:

- **blood clots & debris** should be removed (rinsed away).
- careful digital exploration of scalp wound, palpate galea and underlying cranium to detect any remaining debris, disruptions, or bony step offs - shear injuries may deposit contaminants at sites distant from apparent injury.
- subgaleal emissary vessels drain directly into diploe veins → venous sinuses → potential to intracranial infections.

  N.B. stellate lacerations are particularly prone to infection.
- ***base of laceration must be directly visualized*** (easy to confuse galea disruption or periosteum tear with skull fracture) - by clipping away small area of hair parallel to edges of wound (alternatively, antibiotic ointment can be applied to hair immediately surrounding wound and used to plaster hair away from injury site).
- if there has been *prolonged exposure to skull*, exposed portion of bone should be rongeured until active bleeding appears (nidus of dead bone is source of osteomyelitis):



Closure

- *galea disruption results in wound gaping* - large galea lacerations must be closed (interrupted absorbable 2-0 sutures) - to prevent edges of wound from pulling apart as muscles within galea contract.

  Būtina susiūti sužalotą aponeurozę!
- other scalp layers do not require separate suturing - dermis & skin are repaired in ***single layer*** with *interrupted* or *vertical mattress* sutures of 3-0 nylon or polypropylene.

  N.B. sutures must be **tied more tightly** than elsewhere, so that hemorrhage is controlled (wound hematoma is particularly undesirable complication in scalp!).
- if galea is not involved, scalp lacerations can be repaired with staples.
- if laceration begins on forehead and extends upward beyond hair line, surrounding hair should not be removed (this would obliterate useful landmark for cosmetic closure → malalignment of two laceration edges).
- when repairing ***bald scalp***, use *intradermal* suture.
- accidentally *embedded hair* within repaired laceration *delays healing* - by interfering with proper granulation (by producing inflammatory reaction or by serving as nidus of infection).

- compression dressing over large lacerations (collodion dressing to small ones).
  - when compression dressing is applied, ears must be padded with cotton to prevent pressure necrosis of auricular cartilage!

SKULL FRACTURES

DEPRESSED SKULL FRACTURES

Used sources:
Sekhar "Atlas of Neurosurgical Techniques - Brain" (2006), ch. 78 (pages 909-910)

- indications – see p. TrH5 >>
- lazy "S" or horseshoe-shaped incision over depression; bicoronal incision is preferred for forehead depressions; in open fractures, scalp wound is debrided and incorporated into incision.
- ***burr hole*** near fracture (and over intact dura to prevent brain injury) → bony fragments are elevated → soaked in antibiotic and isotonic saline solution (if wound seems clean and occurred in < 48 hours).
  - cultures of wound and bone and devitalized tissue should be sent for later tailoring of antibiotic coverage should an infection develop.
- ***dural tears are repaired*** (thus, entire lacerated dura must be exposed).
- ***bony fragments are reassembled**** (larger pieces may be wired together); *titanium mesh* is applied to cover larger skull defects; *methyl methacrylate* can be used as substitute for bone fragments (avoid in children; H: *absorbable bone plates and screws*).

  *no difference in infection rate if bone fragments are replaced (soon after trauma) vs. removed (→ need for cranioplasty surgery):

  1. *Jennett B, Miller J: Infection after depressed fracture of skull. Implications for management of nonmissile injuries. J Neurosurg 36:333–339, 1972*
  2. *Kriss F, Taren J, Kahn E: Primary repair of compound skull fractures by replacement of bone fragments. J Neurosurg 30:698–702, 1969*
  3. *Braakman R: Depressed skull fracture: Data, treatment, and follow-up in 225 consecutive cases. J Neurol Neurosurg Psychiatry 35:395–402, 1972*
  4. *Adeloye A, Shokunbi MT: Immediate bone replacement in compound depressed skull fractures. Cent Afr J Med 39:70–73, 1993*
  5. *Blankenship JB, Chadduck WM, Boop FA: Repair of compound-depressed skull fractures in children with replacement of bone fragments. Pediatr Neurosurg 16:297–300, 1990*

  Blankenship et al. demonstrated a 0% infection rate in 31 children with compound depressed cranial fractures treated with primary bone fragment replacement, regardless of the degree of contamination of the wound at the time of surgery.

PING PONG FRACTURE

- there are reports of using obstetric vacuum extractor at bedside to elevate fracture - works for prematures.

- make small incision at and perpendicular to the fracture edge.
- small bur hole (with M-8 bit) on the normal bone just next to fracture line.
- insert **Joker periosteal elevator** and pop fracture up:

- if that fails – extend incision across entire fracture; make circular craniotomy along fracture line; flip bone flap and secure to skull with small plates.
- postop - no need to restrict anything (likely kid won't lay on that side anyway because of pain).

LEPTOMENINGEAL CYST (S. GROWING FRACTURE)

CLINICAL PART → see p. TrH5 >>

- treatment: **cyst excision** → **dural closure** → **cranioplasty**. See p. Op320 >>
  - occasionally, shunt surgery is performed to decompress cyst and treat localized dilatation of ventricles.

From Goodrich (2008)
Growing fracture is a rare complication of skull fracture occurring in infancy and early childhood. This late compli- cation of skull fracture is also known as a leptomeningeal cyst. "Growing" fracture is somewhat of a misnomer, but it is characterized by progressive diastatic enlargement of the fracture line. Although skull fracture is a common occurrence in the pediatric age groups, the incidence of growing fracture is only 0.05 to 1% among skull fractures in childhood.

Patient Selection
The usual presentation of the growing fracture is a progres- sive, often pulsatile, lump on the head. Neurological symp- toms such as seizure, hemiparesis, and mental retardation are less frequent. Often these patients are perfectly asymp- tomatic, and a palpable mass or widening of the fracture line is the sole sign of neurological sequalae noted inci- dentally by the parents. Usually a growing fracture devel- ops within a few months following the initial skull frac- ture, but it may not be recognized for many years. Growing skull fractures usually occur during the fi rst 3 years of life (most often during infancy), and almost never occur after 8 years of age. Although fractures may form in any part of the skull, the most common site for growing fracture is over the skull vault in the parietal region. Dural laceration is always present along the fracture line, and it is an essential factor for the development of a

growing fracture. The dural laceration enlarges with the growing fracture. Computed tomography (CT) or magnetic resonance imaging (MRI) of- ten demonstrates a focal dilatation of the lateral ventricle near the growing fracture. Lack of resistance of both dura and skull leads to focal amplifi cation of the pulse wave of the intracranial pressure, causing herniation of the brain or subarachnoid space through the fracture line and the dural defect. The "growth" of the fracture line is caused by bone resorption due to continuous pulsatile pressure at the edge of the fracture line. A rapidly developing infantile brain and associated pathological conditions such as brain edema or hydrocephalus also contribute an outward driving force to cause brain herniation through the dural and skull defect. This pulsatile force of the brain during the period of its rapid growth produces the brain herniation through the dural lac- eration and fracture line, causing the enlargement of the fracture line of the thin skull. One of the risk factors for the development of a growing fracture is the severity of head trauma. A linear skull fracture with underlying hemorrhagic contusion of the brain suggests a severe injury, significant enough to cause a dural laceration. Initial CT scans for the evaluation of head trauma in patients who ultimately develop a growing fracture usually reveal significant hemorrhage or contusion subjacent to the skull fracture. When a growing fracture is inspected at the time of surgical repair, the herniated brain is seen to be developing a cerebromeningeal cicatrix. In some cases, loculated sub- arachnoid cerebrospinal fluid (CSF) cyst(s) may be noted with underlying gliotic, atrophic brain. Although the loculated sub- arachnoid apace may become cystic (leptomeningeal cyst), true leptomeningeal cysts are rare. The cystic changes in the growing fracture usually represent cystic encephalomalacia. Depressed fractures usually do not cause growing fractures, but a linear fracture extending from the depressed fracture can lead to a growing fracture. The child who on initial x-ray films of the skull has diastasis of the fracture >4 mm is considered to be at risk for future development of a growing fracture. Diastasis of a cranial suture, however, is an unusual site for a growing fracture. A growing fracture at the skull base can occur in an older age group, especially where the bone is thin such as in the orbital roof, if a linear fracture is accompanied by a dural laceration. Growing fracture and a meningoencephalocele can develop with a similar mechanism as those occurring in the skull vault of the young patient.

Radiological Studies

X-ray films of the skull show wide diastases of the fracture line. If initial skull films are available, one can compare the films to confirm "growth" of the fracture line during the interval. When multiple fractures are noted in the same patient, healing of the fracture in one area may be noted as opposed to a growing fracture in another area. The fracture line can cross the coronal or lambdoid sutures but is usually limited to one parietal bone. Neuroimagings such as CT and MRI provide information regarding the sequelae within the growing fracture and any intracranial pathological changes. Furthermore, if they are available from the time of initial trauma, it should be possible to demonstrate progressive changes. It is not unusual that the initial neuroimagings show hemorrhagic contusion, or subarachnoid or extraparenchymal hemorrhage. At the time of discovery of the growing fracture, neuroimag ings demonstrate the diastasis of the fracture line and of- ten cystic lesions near the fracture site. These cystic lesions represent encephalomalacia, a loculated arachnoidal cyst, or cortical atrophy. The ipsilateral ventricle tends to show focal porencephalic dilatation with ipsilateral shift of the midline structure. This phenomenon may be due not only to lack of dural resistance but also to cerebral atrophy.

Management

Surgical intervention is indicated with a growing fracture line, seizure disorder, or progressive neurologic deficits. A progressive cystic degeneration in the brain that has herniated through the dural and cranial defects can occur; there- fore, surgical correction is recommended in young children even when seizures or other neurological symptoms or signs are absent. However, incidental, asymptomatic, and stable fractures in late childhood or adulthood probably do not require surgery. The goal of surgery for growing skull fractures is to repair the dural laceration and cranial defect, and to resect seizure foci. Growth of the growing fracture may arrest after CSF diversion shunting by a decrease of the CSF pulse pressure, but this does not correct a seizure disorder. Placing a shunt for primary treatment of these patients is not advised unless hydrocephalus is present. Shunting for nonhydrocephalic patients creates undesirable shunt de- pendency.

Operative Procedure

The scalp incision should be large enough to expose the entire length of the skull defect. An S-shaped or semicircular skin in- cision is made, and the scalp fl ap is turned subgaleally, leaving the underlying periosteal tissue intact (Fig. 7–1A). By palpa- tion, the entire length of the cranial defect covered by pericra- nium is exposed in surgical view. The site of the cranial defect is often bulging and may be accompanied by blush appearance due to an underlying subarachnoid cyst. As the cranial defect is dissected by incising the pericranium along the edge of the bony defect (Fig. 7–1B), soft tissues adherent to the edge of the cranium defect are scraped off by a sharp dissector. The surgeon should remember that the dural edge is invar- iably larger than the cranial defect, and that the pericranium is directly adherent to the underlying cerebral tissue at the cranial defect. An effort to expose the dural edge by removing the cranial edge should not be undertaken, as this procedure is often complicated by removing the dura simultaneously with the skull bone due to the adhesive nature of the du- ral edge. To identify the dura, several bur holes are made away from the skull defect with a distance of at least 50% of the width of the cranial defect. At this time, a large enough amount of pericranium is removed from the neighboring skull to use it for repair of the dural defect. Once the dura is identifi ed at each bur hole site, the dura is separated from the inner table of the skull toward the defect (Fig. 7–1C). A crani- otomy is made around the skull defect by connecting the bur holes with a craniotomy. Two pieces of the craniotomy fl ap are obtained, one from each side of the growing fracture. After the craniotomy is completed (Fig. 7–2A), reactive pe- riosteal tissue and the cerebromeningeal cicatrix are identi- fi ed in the dural defect. Under magnifi ed vision by means of surgical loupes, the cicatrix including the periosteal tissue is lifted, and all abnormal tissue is separated and transected us- ing a bipolar cautery until normal white matter is exposed (Fig. 7–2B). The edge of the dura is separated from the cer- ebral tissue, carefully avoiding trauma to the cerebral blood vessels. In this region, abnormal tissue such as cystic changes or xanthochromic discoloration due to previous hemorrhage is often noted. After adequate debridement of the cicatrix at the growing fracture and freeing of the intact dural edge from the corti- cal surface, the dural defect is closed using the periosteal graft (Fig. 7–2C). Autologous pericranium is preferable to cadaver dura. A watertight closure of the dura is important to avoid a recurrence of the growing fracture or postoperative CSF leakage. Each of the obtained craniotomy fl aps is split at the diploic space with an osteotome, separating it into inner and outer tables (Fig. 7–2D). The cranial defect is then repaired by laying in the split autologous skull grafts. Usually four pieces are laid next to each other side by side to fill the cranial defect. These flaps are secured to each other with either nylon sutures or stainless steel wires through drill holes (Fig. 7–2E). These fl aps are further secured to the craniotomy edge. If the defect of the skull is too large or the skull is too thin to separate into inner and outer tables, one may consider autologous rib grafts. These autologous bone grafts are well incorporated, and healing is excellent. Foreign materials such as methyl methacrylate should be avoided for cranioplasty in the growing skull.

Figure 7–1 (A) The scalp fl ap is turned subperiosteally. The cranial defect is usually covered by the pericranium. (B) The pericranium is in- cised along the edge of the cranial defect. Then, the edge of the cranial defect is exposed by scraping off the soft tissues adherent to it. (C) The pericranium is removed from the surrounding skull surface and pre- served for dural repair. Four bur holes are made in the surrounding skull for a craniotomy. After the confi rmation of intact dura matter under the bur hole, the dura is separated from the bur hole toward the cranial defect. The surgeon should not attempt to identify the dura by remov- ing the bone from the edge of the cranial defect. The craniotomy is performed on both sides of the growing fracture. The two bone fl aps are removed and preserved for autologous bone cranioplasty.

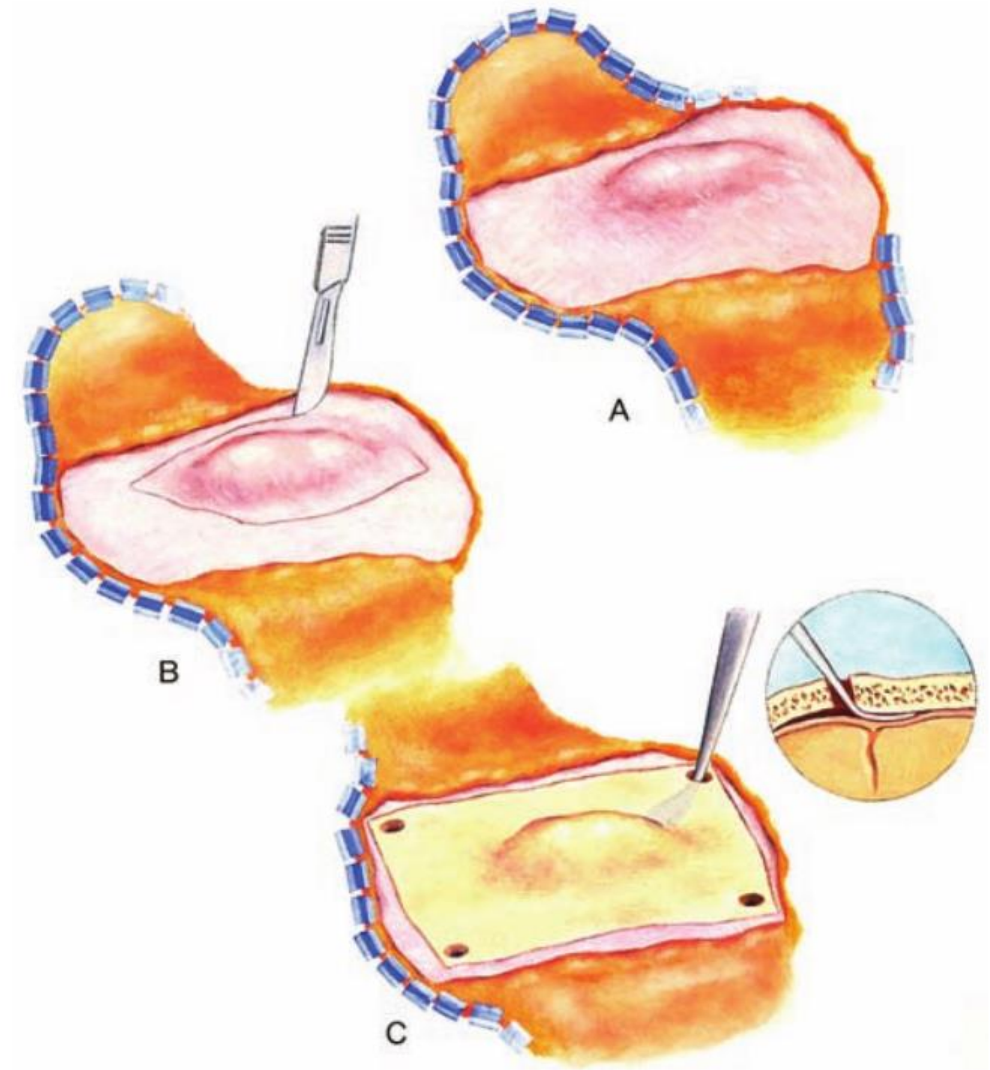
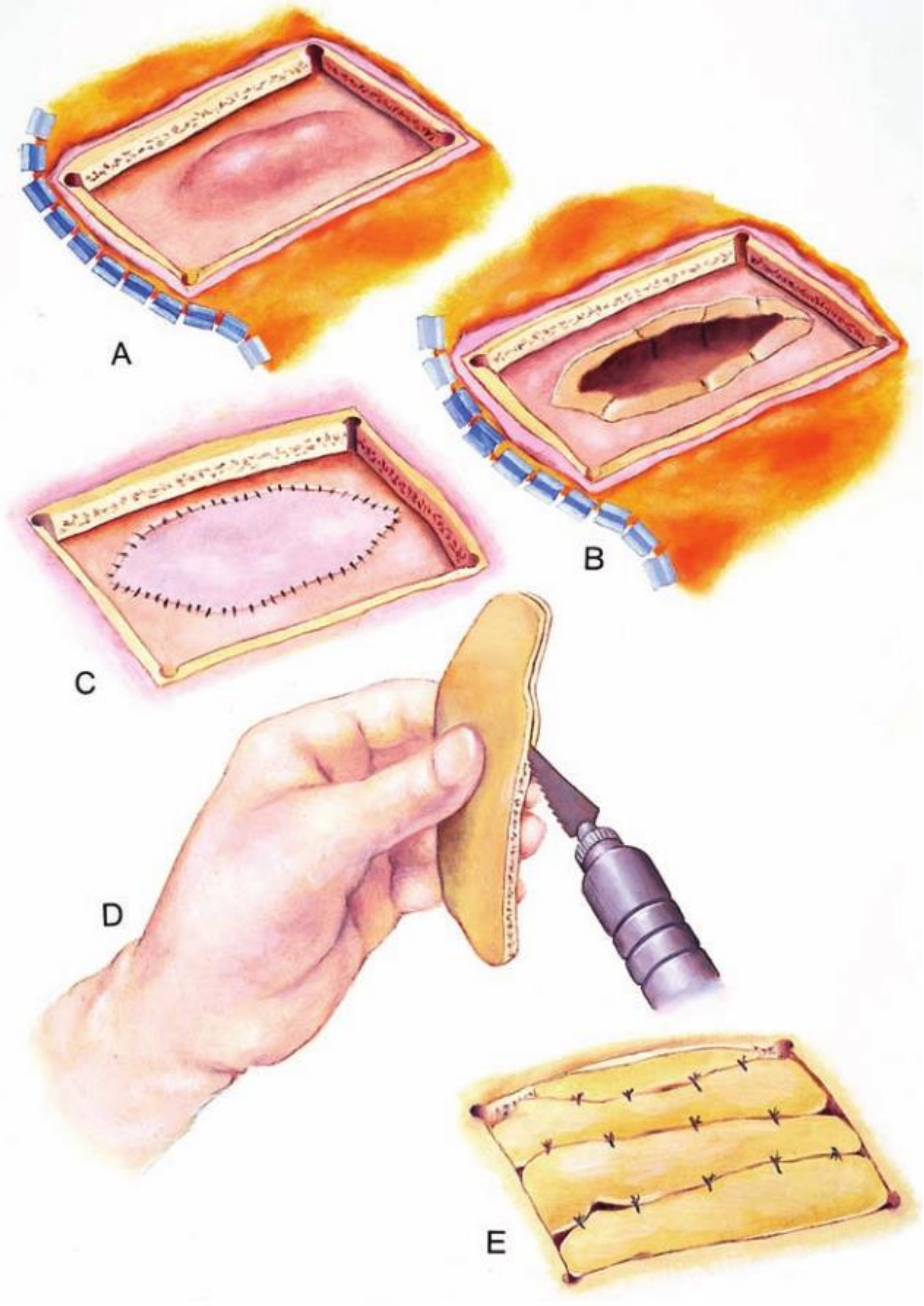


Figure 7–2 (A) After the craniotomy, the intact dura mater is exposed around the dural defect, which is covered by the periosteum. Underneath the overgrowing periosteum is a cerebromenin- geal cicatrix that is removed using bipolar cautery and sharp dissection until healthy white matter is exposed. (B) After all pathological tissues have been removed, the edge of the surrounding dura is separated from the intact cortical surface. (C) The previously removed periosteum is used to repair the dural defect. A watertight closure is achieved with 4–0 sutures. (D) The bone grafts are split at the diploic space between the inner and outer ta- bles by means of an osteotome. (E) The obtained split bone fl aps are used to repair the cranial de- fect. The bone fl aps are secured to each other and to the edge of the cranial defect with nylon sutures or stainless steel wires.



Specific Considerations

The growing fracture may extend toward a dural venous sinus such as the superior sagittal or lateral sinus. Although these venous sinuses were spared from direct injury at the initial trauma, direct exposure of them is not advised or necessary. When the fracture line extends perpendicularly to these sinuses, the closest end to the sinus does not need dural repair. However, if the growing fracture is parallel and near to the sinus, dural repair may be difficult due to the lack of enough dural edge next to the sinus. In these cases, one may repair the dural defect with a periosteal graft sutured to the periosteum of the skull above the sinus.

Postoperative Management

Including Possible Complications CSF diversion shunting has been recommended for persist- ent postoperative CSF leakage from the craniotomy wound. It is justified if coexisting hydrocephalus is evident, or if CSF leakage occurs despite adequate repair of the growing fracture. A lumboperitoneal shunt or temporary lumbar CSF drainage is to be considered under these circumstances.

TWIST DRILL CRANIOSTOMY (FOR CHRONIC SUBDURAL HEMATOMA)

- hole is drilled at 45° angle to skull over thickest part of hematoma (unless this lies over motor strip); possible under local anesthesia at bedside.
- twist drill is used to perforate dura and to release subdural hematoma.
- thin rubber catheter is gently guided* into subdural space, tunneled under scalp, and brought out through stab incision (connect to closed drainage system without suction for 24-72 hours).

  *e.g. on stylet bent like hockey stick
- postop – see below >>

BURR HOLE WASHOUT (BHWO), s. CRANIOSTOMY (FOR CHRONIC SUBDURAL HEMATOMA)

Principles of chronic SDH treatment (incl. indications, surgery types) → see p. TrH13 >>

INDICATIONS

- subacute / chronic SDH – BHWO is the procedure of choice. see p. TrH13 >>
- sometimes even acute SDH can be evacuated through burr holes (e.g. unstable patient with low Hb – nonclotting hematoma); but usually, if patient is minimally symptomatic (e.g. headaches), – may wait until hematoma liquefies (usually 10-14 days).

PROCEDURE

Position

- supine position ± towel roll under ipsilateral shoulder.
- head on gel donut / horseshoe / subdural head holder slightly rotated to the contralateral side.

  Dr. Graham likes subdural head holder – keeps head straight vertical (helps with drainage during procedure) and permits access to posterior region.

Anesthesia

a) local anesthesia at **bedside**
b) monitored local anesthesia or general anesthesia in **OR** (best results!)

Procedure

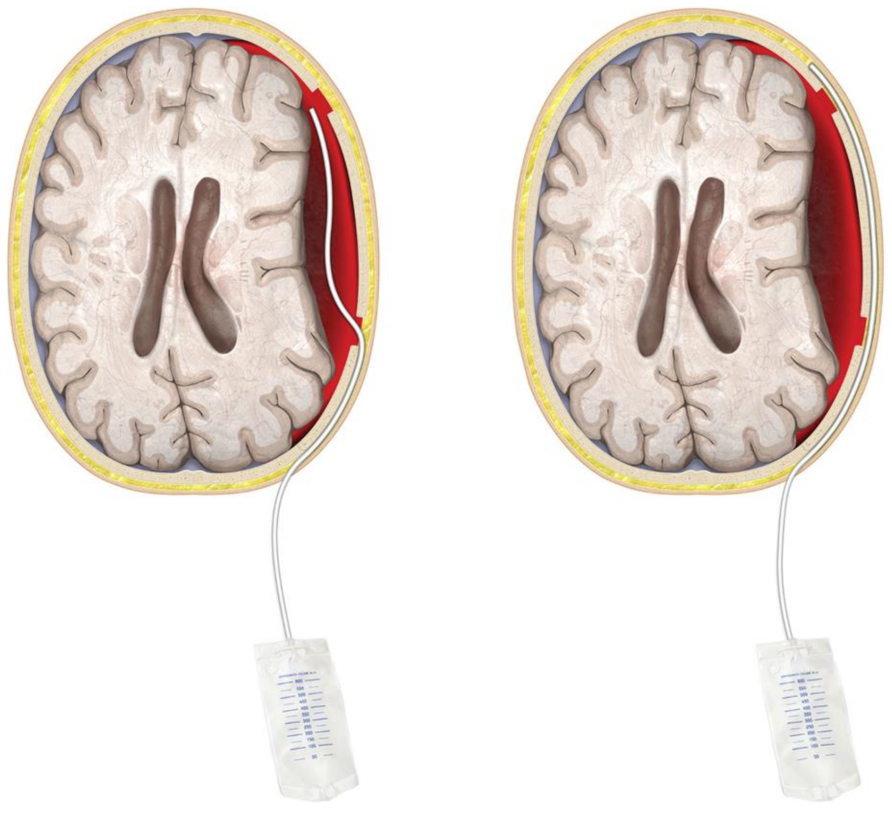
- burr holes are placed over thickest aspects of hematoma; ideally, at superior temporal line (just above temporalis muscle; if needed, OK to go through muscle)
- make 2 (1-3) burr holes.
  – e.g. frontal and parietal burr holes - if needed, can be incorporated into craniotomy;
  – studies show that patients do well with just **1 burr hole** but difficult to irrigate through 1 hole!
  – if holes are placed too near extremes of hematoma, drainage may be blocked by expanding brain (H: depress surface of brain with patty or spatula).
- coagulate dura with bipolar cautery
- incise dura in cruciform manner* with a #15 or #11 blade (*do not cut to the edge of bone – leave some dura – easier to stop bleeding if it happens) – watch for motor oil color thin fluid.
- cut with Bovie to completely open up burr holes.
  – if clearly see blood through dura, may open dura with Bovie (avoiding bipolar & blade step)
- if thick dark clot is found → thorough copious washout; attempt to scrape (mobilize) clot with Penfield #3, #4, hockey-stick
  – may need additional bur holes or even mini craniotomy; alternative – if significant part of clot was evacuated and brain reexpanded, - may finish and follow with CT (repeat BHWO if needed – less morbidity than craniotomy)
- attempt to fenestrate (with bipolar) visible pathologic membranes (but they may ooze blood nonstop!)
- irrigate with sterile saline via Becker catheter inserted subdurally in all directions (except superiorly towards sagittal suture – not to disrupt bridging veins)
  – irrigate until returning fluid is clear and free of old clot fragments.
- see for brain re-expansion; often, brain does not re-expand right away → drain

Drain reduces 2-3-fold subdural hematoma recurrence (8-9% vs. 20-24% without drain) and mortality:

	Drain	No drain	Statistical significance
Recurrence	9.3%	24%	$p = 0.003\%$
Mortality at 6 months	8.6%	18.1%	$p = 0.042\%$

Santarius T. Use of drains versus no drains after burr-hole evacuation of chronic subdural haematoma: a randomised controlled trial. Lancet 2009; 374 : 1067 – 1073

Drain location



A. Subdural drain
  – ventriculostomy catheter (Drs. Graham, Holloway use round 7F JP drain – it is more traumatic than EVD catheter).
  – insert through posterior (parietal)* or anterior (frontal)* bur hole and advance anteriorly (i.e. *direct drain towards frontal pole* - when brain reexpands postop, last

fluid to disappear is frontal; if drain sits posteriorly, it becomes blocked early by reexpanding brain)

*studies show no difference

- do not force drain (stop with slightest resistance encounter – drain goes to parenchyma very easily!); if brain reexpanded, do not place subdural drain, rather place drain under galea and running over bur holes.
- drain complications: puncturing cortex*, causing hematoma, subdural empyema.

*H: Becker catheter is used after the stylet is bent in shape of hockey stick – so that catheter slides not into the brain!

B. Subgaleal drain – not positioned in direct contact to cortical structures, bridging veins, or hematoma membranes
- use flat 10F JP drain; make sure holes are over bur holes (sometimes difficult as flat 10F JP drain has long tapering part that has no holes; alternative – use fluted / channeled 10F drain).
- tunnel drain under galea from anterior bur hole to posterior and the further posterior.

N.B. there is no difference in recurrence rates (7.7-9.1%)* and outcomes with **subdural** and **subgaleal** drains but **subgaleal** drains have reduced risk of infection, intracranial hemorrhage, brain injury, empyema, and epilepsy.

Laurence Johann Glancz et al. Does Drain Position and Duration Influence Outcomes in Patients Undergoing Burr-Hole Evacuation of Chronic Subdural Hematoma? Lessons from a UK Multicenter Prospective Cohort Study. Neurosurgery 85:486–493, Oct 2019

Jehuda Soleman et al. Subperiosteal vs Subdural Drain After Burr-Hole Drainage of Chronic Subdural Hematoma: A Randomized Clinical Trial (cSDH-Drain-Trial). Neurosurgery 0:1–10, Oct 2019

*there are studies that show even lower recurrence rate (8.3% vs. 12%) with **subgaleal** than **subdural** drains.

Jehuda Soleman et al. Subperiosteal vs Subdural Drain After Burr-Hole Drainage of Chronic Subdural Hematoma: A Randomized Clinical Trial (cSDH-Drain-Trial). Neurosurgery 0:1–10, Oct 2019

CLOSURE

- both burr holes are covered with cranial plates and screws; do not seal off bur holes – allows subdural excess fluid to get absorbed in subgaleal space.
- tunnel drain under skin as far as you can posteriorly.
- galea is approximated with 2-0 Vicryl in interrupted fashion except small gap in upper (anterior) incision → sterile saline is gently injected through the drain allowing intracranial air to be expelled through galeal gap (→ table is brought into Trendelenburg position, anesthesia applies Valsalva maneuver) → galea closed completely.
- drain is connected to ***closed drainage system*** ["transfer pack" or "bile bag"] put on gravity drainage without suction (or JP bulb with thumbprint gentle suction – Dr. Graham; suction creates negative intracranial pressure – may cause bradycardia, lightheadedness).
- simple running 3-0 Monocryl, bacitracin ointment.

POSTOPERATIVELY

- postoperative seizures are reported in 3-10% patients (many surgeons use prophylactic anticonvulsants for 7-30 days after operation).
- **hCT** next day
  - CT often (≈ 92%) shows *residual subdural collection* - should be left alone (unless it continues to exert significant mass effect); thus, recommendation is not to do postop head CT until ≥ 3 days postop (unless patient deteriorates)
- **flat bed** regimen as long as subdural drain is present (to prevent sucking air intracranially)* + adequate **IV hydration** (to promote brain re-expansion).

  *alternatively, subdural drain may be connected to JP bulb (with minimal suction – "dimple on bulb") – patient may sit up to 30 degrees - much better tolerated.
- keep drain 1-3* days; studies show that patients do best if drain is left for ≥ 72 hours and patient remains flat all that time; Dr. Graham keeps longer (with daily stripping of JP drain) if on CT brain is not reexpanding.

  *N.B. there is no difference in recurrence rates (6.4-8.4%) or outcomes with drainage for 1 or 2 days

  Laurence Johann Glancz et al. Does Drain Position and Duration Influence Outcomes in Patients Undergoing Burr-Hole Evacuation of Chronic Subdural Hematoma? Lessons from a UK Multicenter Prospective Cohort Study. Neurosurgery 85:486–493, Oct 2019
- if drainage is sub-optimal (< 50 cc) TPA can be instilled through drain.

  Neils D, Singanallur P, Huaping W, et al. Recurrence-Free Chronic Subdural hematomas: A Retrospective Analysis of the Instillation of Tissue Plasminogen Activator in Addition to Twist Drill or Burr Hole Drainage in the Treatment of Chronic Subdural Hematomas

  In this study intrathecal tPA lead to zero cSDH recurrences, zero complications

SPECIAL SITUATIONS

IF SDH RECURS AFTER BURR HOLE DRAINAGE

Rate of 2-37%.

Risk factors:
1. **Patient factors**: alcoholism, seizure disorders, coagulopathy, ventriculoperitoneal shunt.
2. **Radiologic factors**: poor brain reexpansion postoperatively, significant subdural air, greater midline shift, heterogeneous hematomas (layered or multi-loculated), and higher-density hematomas.
3. **Surgical factors**: lack of or poor postoperative drainage.

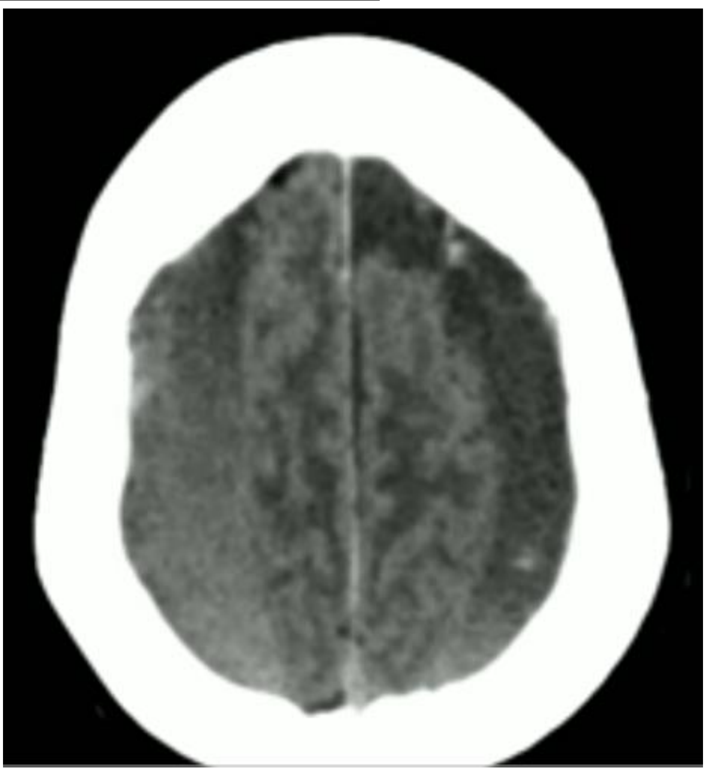
Further workup:
1) MRI, CTA of brain – for ***vascular abnormalities***
2) MRI of spine, myelogram – for ***occult CSF leak*** (Dr. Holloway would do blood patch even if spine looks unremarkable)

Treatment:
1) correct causes
2) repeat bur hole washout.
3) medications (TXA, APROTININ, DEX, etc) → see p. TrH13 >>
4) craniotomy with partial removal of enveloping membranes (membranectomy*) or shunting of subdural cavity into pleural or peritoneal cavity.

REOPERATION RATES (for hematoma reaccumulation) ≈ 12-22%.

*if you see clearly brain surface, leave that "membrane" alone – do not violate arachnoid (→ CSF leak)

BILATERAL CHRONIC SDH



- many experts do **bilateral BHWO**.
- modern Japanese (and now also UPMC) – do **MRI preop with intention to BHWO on one side only**; if hematoma on the "conservative" side looks iso- or hypo-dense on T1, there is 25-fold increased chance that the second side will also need BHWO.

ACUTE EPIDURAL HEMATOMA EVACUATION

IMMEDIATE EXPLORATORY BUR HOLES

INDICATION

- conservatively uncontrollable ICP↑ with rapid patient deterioration in ED (consciousness↓ + asymmetric findings) → think of **brain compression*** → **emergency BURR HOLES**.

*if ***skull fracture*** is present – most commonly **epidural hematoma**.

It is life-saving procedure - unless burr hole is done patient will die or be damaged: you and patient have nothing to lose and everything to gain - inelegant burr hole now will do much more good than elegant operation one hour or more later.

STRATEGY

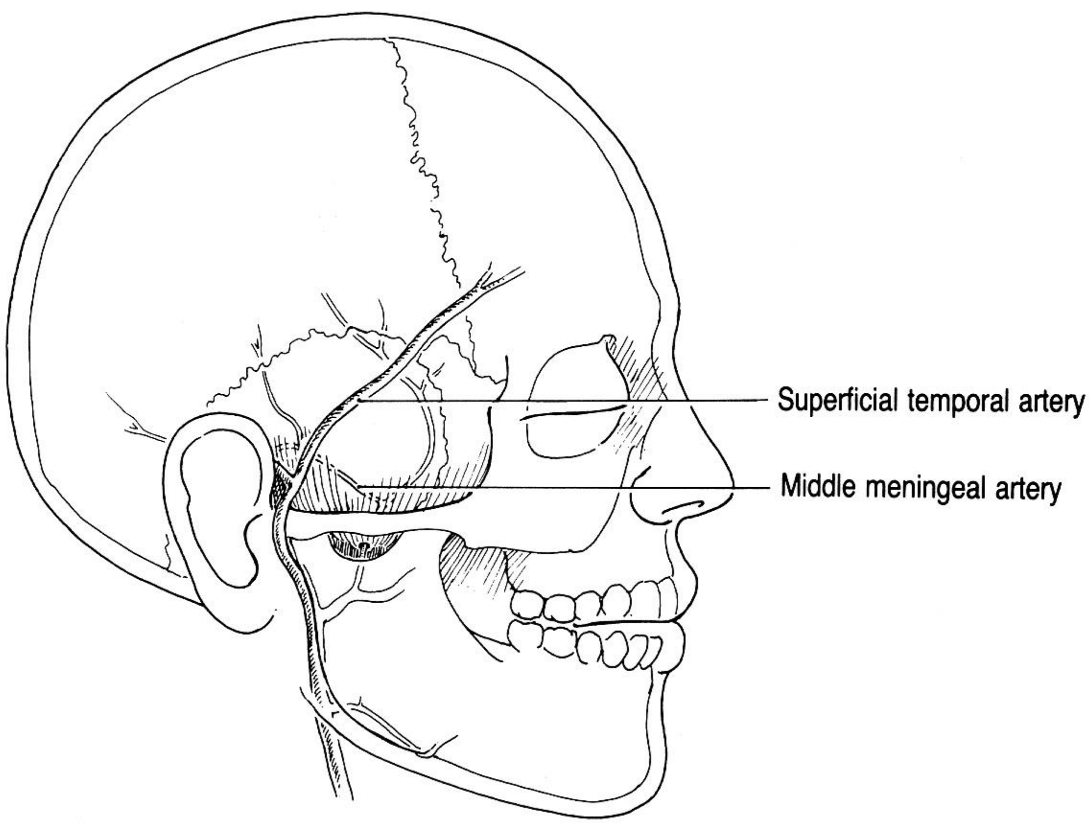
If CT is unavailable – side of burr hole is chosen:

Feature	Hematoma Side
First (or only) dilated pupil	Ipsilateral 94%
Most abnormal motor response	Contralateral 82%
Skull fracture	Ipsilateral 66%

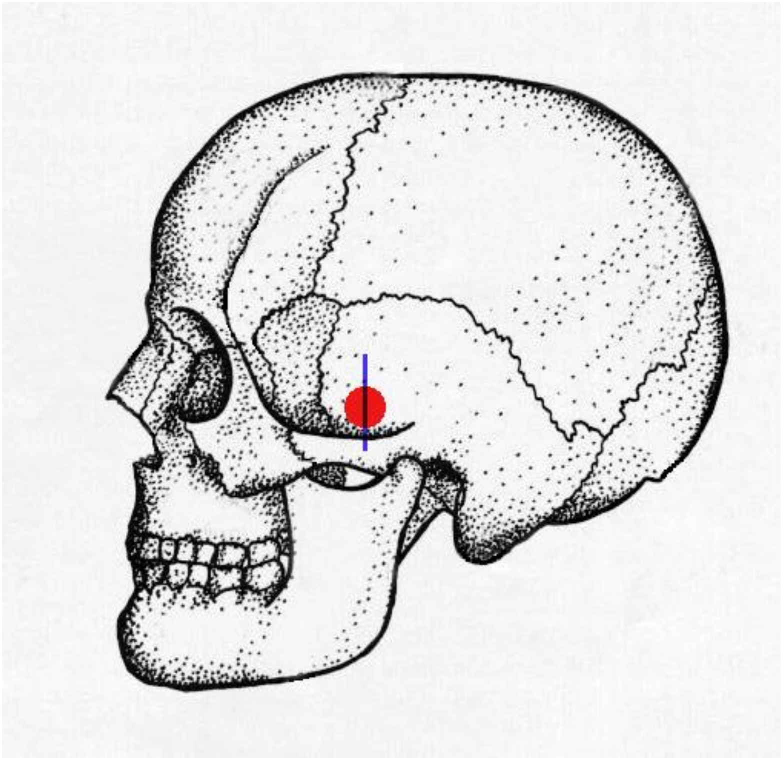
  – also can be helpful - scalp wound side, M-echo signal dislocation, pineal gland dislocation (on X-ray).

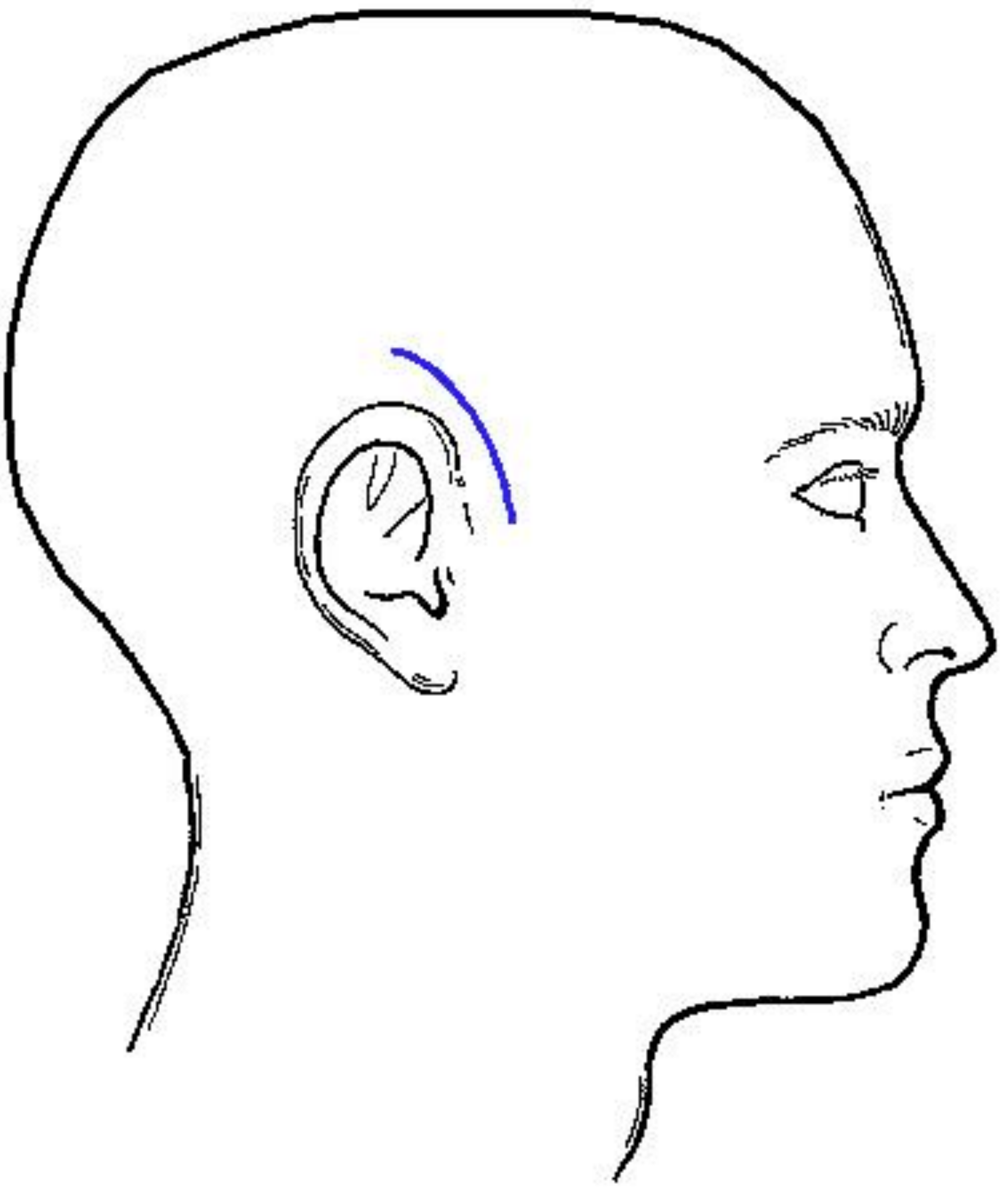
- position patient supine – allows approach to both skull sides.

1. *First burr hole* - low in temporal region, just above zygoma (one finger's breadth anterior to tragus and one finger's breadth above zygomatic process).
   – skin incision is started perpendicular to zygomatic process and curved so it can be incorporated into scalp flap.
   – inexperienced surgeons often make this burr hole too high, near temporal crest (this may fail to disclose hematoma in temporal region and is also difficult to incorporate into suitable bone flap).
   – superficial temporal artery will likely be transected (cauterize or pick up with hemostat & tie).



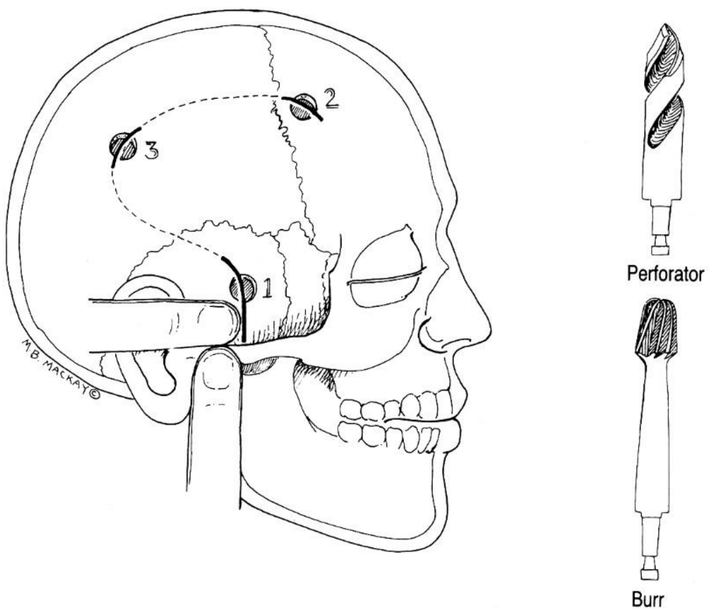
Source of picture: Paul W. Roberts "Useful Procedures in Medical Practice" (1986); Lea & Febiger; ISBN-13: 978-0812109856 >>





2. *If initial temporal burr hole is negative*, other openings are made:
   a) close to **fracture-line**
   b) in region of any **scalp wound** (in absence of fracture)
   c) **frontal** (1 cm anterior to coronal suture [still behind hairline] and 3 cm from midline; skin incision is parallel to midline; periosteum here is directly beneath galea) and **parietal** (at parietal boss – most prominent point in parietal bone).

   N.B. incision & burr holes should, if possible, be made in such sites that can be incorporated into "question mark" flap; burr hole must be adjacent to, but not over, skull fracture.



Source of picture: Paul W. Roberts "Useful Procedures in Medical Practice" (1986); Lea & Febiger; ISBN-13: 978-0812109856 >>

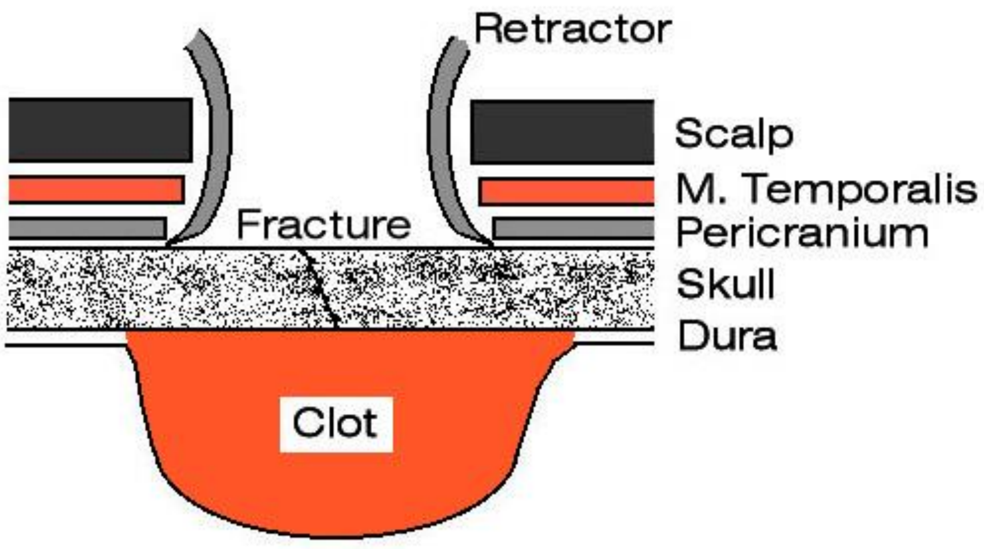
3. *If no hematoma is found on one side* → proceed in analogous order on other skull side.

4. *If no hematoma is found on supratentorially* ± occipital fracture → bilateral occipital burr holes.

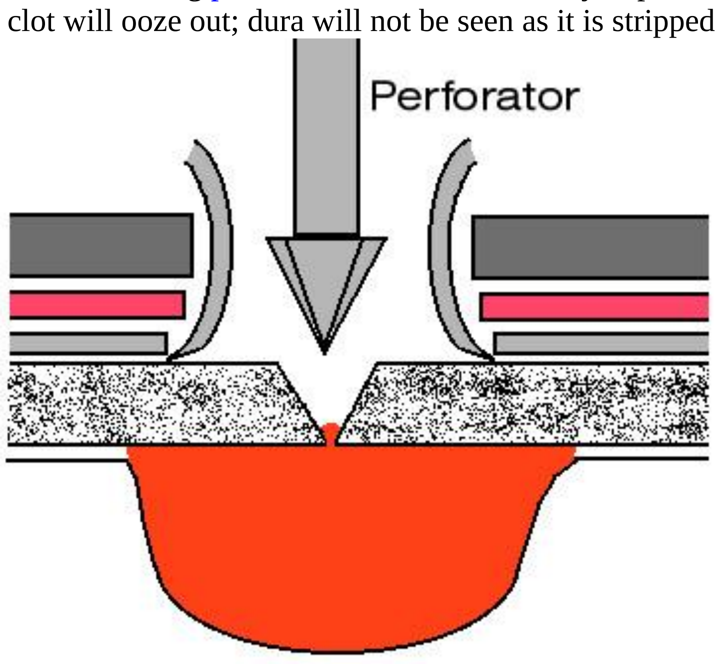
- if **epidural hematoma** is not found – consider **subdural hematoma**. see p. TrH13 >>

TECHNIQUE

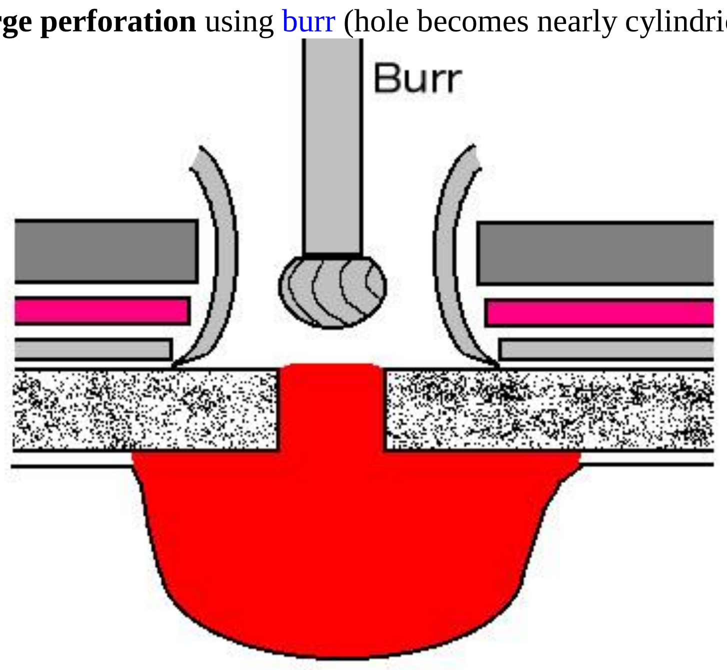
- on occasion, procedure may be performed in ED (under sterile conditions!)
- shave entire scalp (if time permits).
- usually *no local anaesthetic* is necessary.
- 3-4 cm **scalp incision**; incise right down to bone (if available, cutting cautery is used to incise galea and temporalis muscle); do not stop to stop scalp bleeding.
- **scrape back pericranium (periosteum)** using periosteal elevator (or similar instrument, e.g. handle of scalpel) to expose skull.
- **insert** self-retaining retractor - this will stop all bleeding.
  - persisting bleeding (from skin or muscle) must be controlled before proceeding.



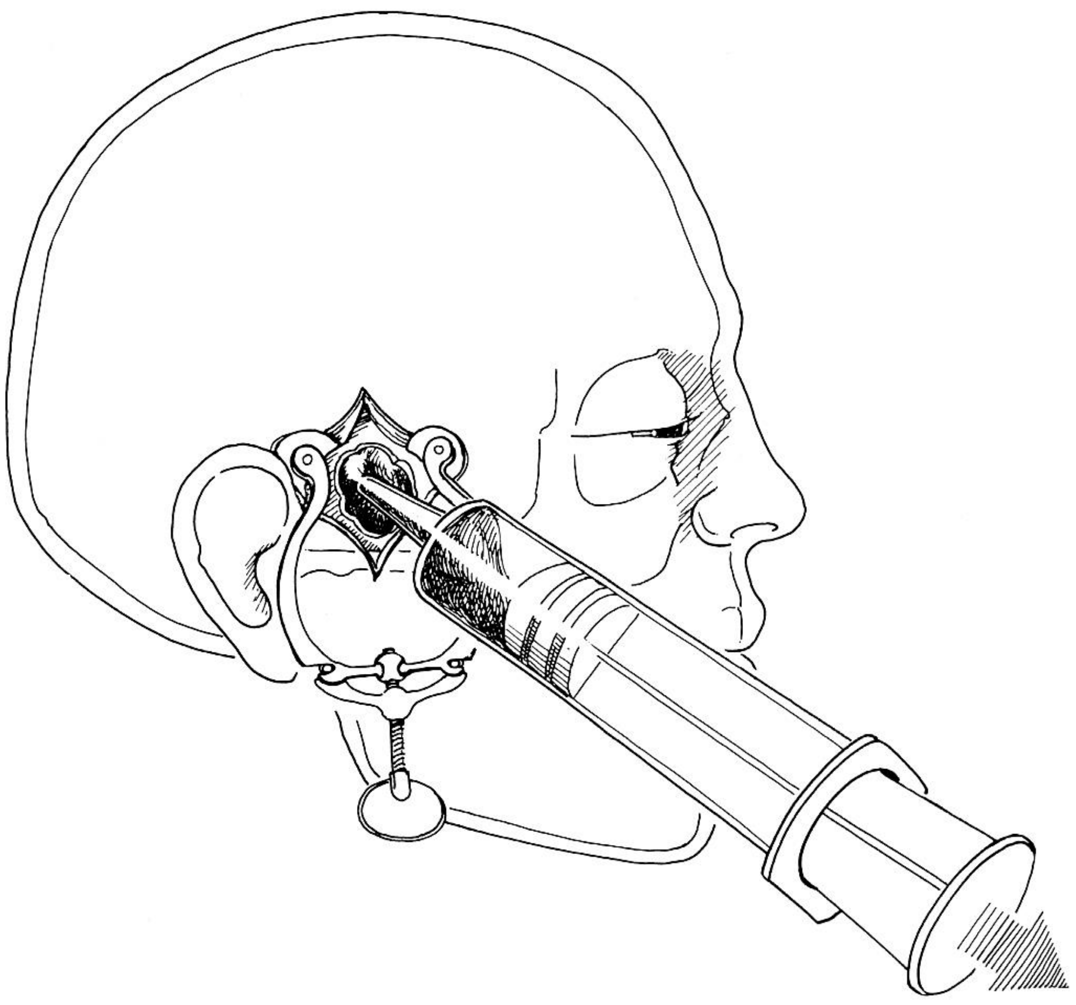
- **perforate bone** using perforator - do no more than just perforate skull - this will create conical hole - dark clot will ooze out; dura will not be seen as it is stripped away by clot:



- **enlarge perforation** using burr (hole becomes nearly cylindrical); clot will immediately ooze out:



- **suck clot away** by applying sucker to burr hole:
  N.B. do not insert sucker into cavity - that will cause more bleeding and might damage brain.



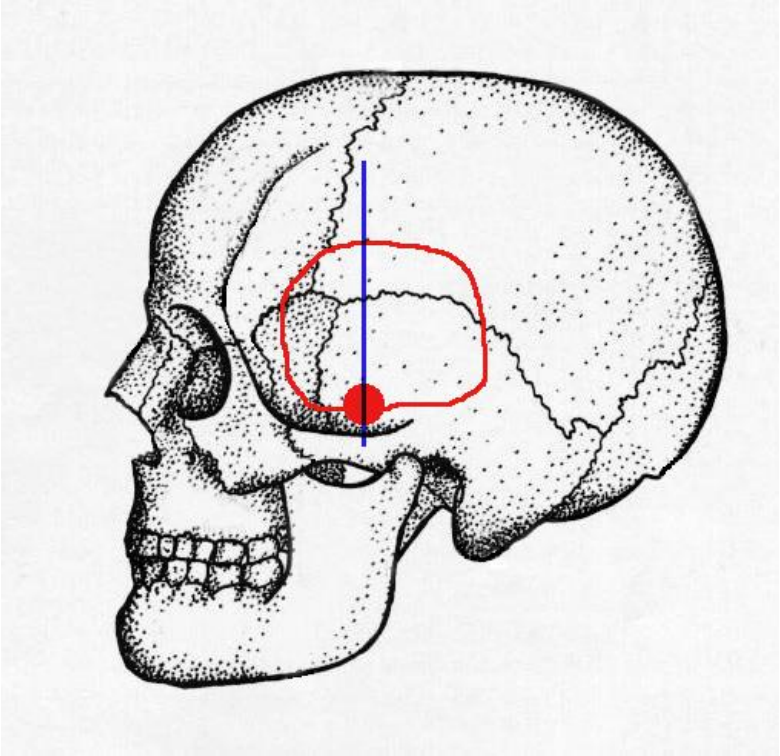
Source of picture: Paul W. Roberts "Useful Procedures in Medical Practice" (1986); Lea & Febiger; ISBN-13: 978-0812109856 >>

- to remove clot adequately, it may be necessary to enlarge hole with rongeur (i.e. to do **craniectomy**).
- be ready to cope with BP drop (as Cushing reflex is relieved).
- middle meningeal artery (if identified to be torn) is cauterized, waxed into bone, or secured in some manner.
- bone edges are waxed.
- Gelfoam is placed in opening to prevent blood from subgaleal space from entering cranial cavity.
- it is now safe to **transfer patient** for definite neurosurgical help.
  - leave *SCALP RETRACTOR* in with voluminous sterile dressing (but some surgeons suture scalp before transfer).
  - leave in endotracheal tube and leave drip up.
  - send CT scans and any blood that has been cross matched with patient.

CRANIECTOMY

- unusual (method best suited for non-neurosurgeons)

CUSHING incision and craniectomy:



CRANIOTOMY

INDICATIONS

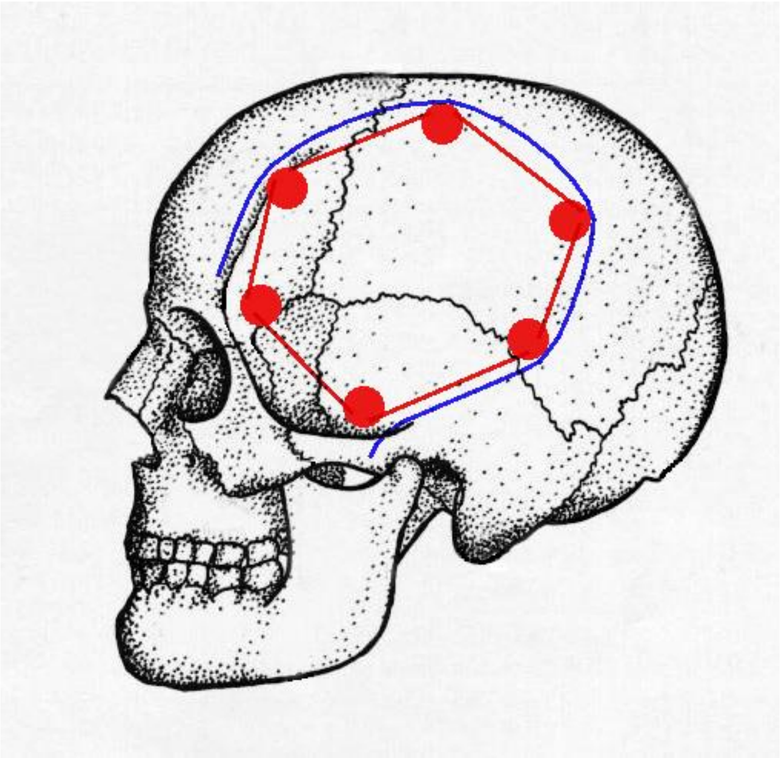
see p. TrH11 >>

APPROACH

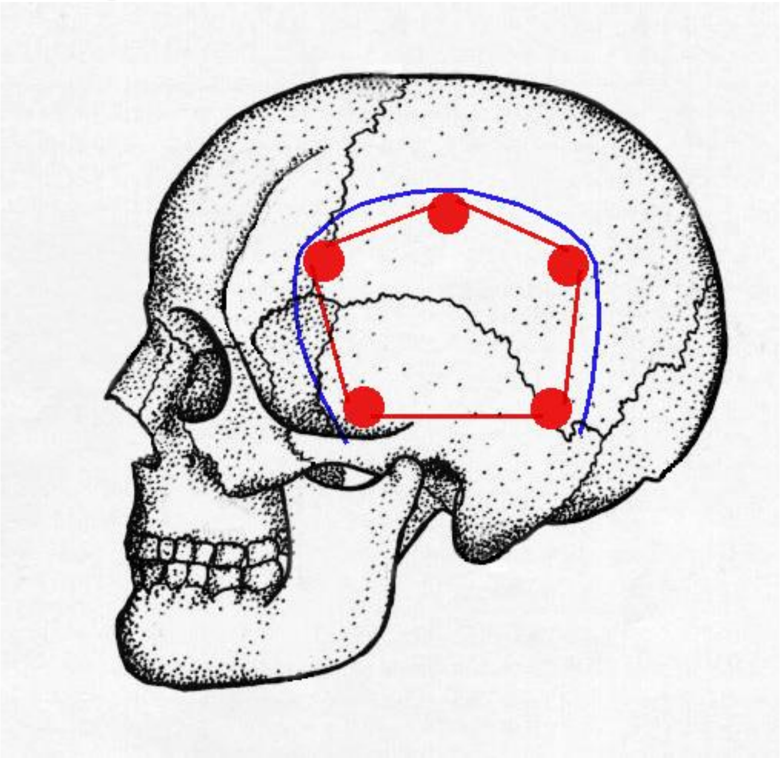
- body is supine, head is placed on donut.
- careful with head ***head clamps*** - may propagate existing skull fractures.
  - occipital / posterior fossa EDH requires positioning in lateral, semiprone, or prone position (three-point head clamps are used for stable head fixation).
- calvarium is opened over site of hemorrhage;
  - if patient presents with *clinical signs of herniation*, rapid BURR HOLE is made over hematoma → hematoma is partially evacuated (pressure relief until entire epidural blood clot can be evacuated).

"Question Mark" skin flap (frontotemporoparietal craniotomy) - best for classical anterior temporal hematoma:

- incision is started at zygoma, 1 cm anterior to auricle; continued parietally upward and backward ("question mark"); finished frontally not far from midline at hairline.
- initial burr hole is made over zygoma:



"Horse Shoe" skin flap - for more posterior hematoma:



PROCEDURE

- hematoma evacuation.
- inspection of dura → bleeding control:
  - **arterial sites** - coagulation/ligation/clipping; it may be necessary to follow middle meningeal artery to foramen spinosum, where it can be controlled by plugging foramen with cotton, bone wax, or swab stick.
  - **dural venous sinus** - tamponade with muscle + head-of-the-bed elevation;
  - **diploic vein** bleeding is controlled with bone waxing.
- if dark subdural hematoma shines through dura (or preoperative CT demonstrated contusions beneath EDH) → open dura to explore subdural space.
- epidural **tack-up sutures** are placed from dura to craniotomy bone edge ± to center of craniotomy flap - *to tamponade epidural bleeding* from areas beyond craniotomy edges and *to prevent recurrence*.
- occasionally, ***epidural Jackson-Pratt drains*** are employed for as long as 24 hours.

POSTOPERATIVELY

- unusual to require ***ICP monitoring*** & ***treatment***.
- follow-up CT scans (in ≈ 7% cases EDH recurs).

ACUTE SUBDURAL HEMATOMA EVACUATION

Indications for surgery – see p. TrH13 >>

IMMEDIATE EXPLORATORY BUR HOLES

- dura at burr hole is cauterized and cut in cruciate manner* (using *SMALL SCALPEL*).
  *cutting dura in cruciate manner produces four corners of dura that may be shrunk using cautery to expose what lies beneath
- if only outer dural layer is incised initially, *SHARP HOOK* is placed between dural leaves → dura is elevated away from brain → inner dural layer is incised (avoid injury to underlying brain but brain is supposed be separated by subdural clot).
  - if subdural hematoma has been present for some time, there will be membrane (just deep to dura) that must also be incised carefully.
- solid subdural blood is cautiously aspirated (liquid blood is allowed to escape).
  - burr hole may be enlarged with rongeur, but craniotomy is required to adequately remove fresh subdural blood (pressure release via burr holes serves only as temporizing treatment).
- cautious irrigation (with saline) to rinse out blood clots.

CRANIOTOMY

INDICATIONS

see p. TrH13 >>

TECHNIQUE

- make ***initial burr hole*** (even before opening entire incision), open dura → early decompression (gentle suction + irrigation) to forestall herniation.
- ***burr hole evacuation is insufficient for acute SDH*** → large **question mark-shaped flap CRANIOTOMY** (should encompass nearly extent of hematoma).
  - exposure should include sylvian fissure (likely source of ruptured cortical vessel).
  - if hematoma extends near temporal fossa, bone flap may be hinged on temporalis muscle, but *dura is turned toward sagittal sinus*!
  - if exact SDH location is known, LINEAR SCALP INCISION may be used (reduced surgical time).
- may *fenestrate dura in mesh-like fashion* rather than opening it (to prevent brain herniation through wound).
- remove blood (biopsy forceps, Penfield 3, gentle suction, irrigation) and surrounding "subdural membranes".
  - organized hematoma peels off surface of brain.
  - segments of hematoma, not directly exposed, can be irrigated and gently loosened with suction and Penfield #3.
  - explore areas under dura along entire perimeter by depressing brain gently* with widest ribbon (narrow ribbon cuts into brain).
    *beware pulling on bridging veins
- stop any active bleeding:
  1) arterial bleeding → bipolar electrocautery.
  2) venous oozing → Gelfoam, Surgicel; bridging veins → tamponade with muscle pieces.
- intraoperative ultrasound may locate *INTRAPARENCHYMAL CLOTS* (also may require evacuation).
- if associated brain injury and edema are present → place ***ICP monitor***; consider bone flap removal.
- bone flap management:
  a) bone flap is replaced
  b) bone flap is replaced loosely ("hinge craniectomy").
  c) leave bone flap out (craniectomy)
  N.B. craniectomy is never wrong with slightest anticipation of (postoperative) brain swelling
- subgaleal (±subdural) **drain** is placed.

MALIGNANT CEREBRAL EDEMA

See below >>

POSTOPERATIVELY

- obtain **CT** within 24 hours of removing acute SDH (symptomatic recurrent / residual hematoma → repeat operative intervention).

DECOMPRESSIVE CRANIECTOMIES

- allow brain to swell → reduced ICP → prevention of cerebral herniation and death.

References:
R. **Jandial** "Core Techniques in Operative Neurosurgery" (2011), procedure 12
E. **Sander Connolly** "Fundamentals of operative techniques" (2002), no info in this book
E. **Sander Connolly** "Fundamentals of operative techniques" (2010), ch. 20
Richard G. **Ellenbogen** "Principles of neurological surgery" 3rd ed. (2012), p. 318-319
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A. Quiñones-Hinojosa "**Schmidek and Sweet** Operative Neurosurgical Techniques: Indications, Methods, and Results", 6th ed. (2012), ch. 135 (p. 1551-1557)
Roberto C. Heros "**Kempe's** operative neurosurgery" (2003), no info in this book
Nader "Neurosurgery Tricks of the Trade – CRANIAL" (2014), ch. 107 (p. 505-511)
H. Richard Winn "**Youmans** Neurological Surgery" 6th ed. (2011), ch. 338

Suggested further readings:
1. Coplin W.M., Cullen N.K., Policherla P.N., et al: Safety and feasibility of craniectomy with duraplasty as the initial surgical intervention for severe traumatic brain injury. J Trauma 2001; 50:1050-1059.
2. Munch E., Horn P., Schurer L., et al: Management of severe traumatic brain injury by decompressive craniectomy. Neurosurgery 2000; 47:315-322.
3. Schneider G.H., Bardt T., Lanksch W.R., et al: Decompressive craniectomy following traumatic brain injury: ICP, CPP and neurological outcome. Acta Neurochir Suppl 2002; 81:77-79.
4. Valadka A.B., Robertson C.S.: Surgery of cerebral trauma and associated critical care. Neurosurgery 2007; 61:203-220

INDICATIONS

1. **Primary decompression – prophylactic measure** during emergency evacuation of traumatic mass lesion when bone is not replaced in anticipation of postoperative elevated ICP.
2. **Secondary decompression – therapeutic to elevated ICP refractory** to medical treatment (e.g. in severe TBI, malignant MCA stroke, some nontraumatic pediatric cases with refractory ICP such as Reyes syndrome) – surgery is lifesaving but not restorative (of what is lost)!

malignant MCA stroke aspects – see p. Vas5 >>

- some experts do not include age in making the decision whether to proceed with decompressive craniectomy; however, on the basis of data suggesting that old age does correlate with poor outcomes after decompressive craniectomy for TBI, they temper the expectations of outcomes in older patients.

Two issues to talk to family:

1) primary brain injury is so severe that, if patients survive, they are most likely to remain severely disabled.
2) how do people feel about survival with severe neurological disability for themselves? "How do you feel about surgical intervention that may leave you severely disabled?"

- it is important for family members to know dire prognosis to avoid unrealistic expectations (older patients, patients with limited brainstem reflexes and low GCS from time of injury are at greatest risk for poor outcome).

BENEFITS & DISADVANTAGES, TRIALS

- when performed correctly, decompressive craniectomy can reduce ICP, increase cerebral blood flow and oxygenation, and reduce therapeutic intensity levels, potentially preventing cerebral herniation and death.

Decompressive craniectomy decreases mortality but does not improve good outcomes!

Guidelines for the Management of Severe Traumatic Brain Injury (4th ed): 2020 Update:

GWJ Hawryluk et al. Guidelines for the Management of Severe Traumatic Brain Injury: 2020 Update of the Decompressive Craniectomy Recommendations. Neurosurgery 2020 August 6

- incorporated RESCUEicp data + DECRA 12-month data → 3 new, 1 retained, 1 removed level 2A recommendations.

- both RESCUEicp and DECRA are class 1 studies with good quality evidence.
- both RESCUEicp and DECRA studied secondary craniectomy:

	DECRA	RESCUEicp
Timing of surgery	**Early** - ICP > 20 mmHg for 15 min over a 1-h period despite the optimization of tier 1 treatments within the first 72 h of care; intracranial mass lesions excluded	**Late (more established intracranial hypertension)** - ICP > 25 mm Hg for 1-12 h refractory to 2 tiers of treatment within 10 d of admission; also included mass lesions (but most pts were diffuse injury)
Type of surgery	Exclusively **bifrontal DC**	Mostly **bifrontal DC**
Outcomes	Mortality the same but more worse outcomes and less good outcomes with early DC Mortality DC vs medical management 6 mo. 19% vs 18% 12 mo 21% vs19% GOS-E 12 mo (DC N = 73 % / Medical N = 82 %) Death 21 / 19 Vegetative 11 / 3 Low severe disability 19 / 16 Upper severe disability 8 / 10 Low moderate disability 14 / 12 Upper moderate disability 19 / 19 Lower good rec 5 / 16 Upper good rec 3 / 5 DC fewer ICU days during hospitalization 3(10-18) vs 18(13-24), $P < .001$. 12 mo exploratory analyses: Fewer good outcomes in survivors with DC: 0.33 (95% CI, 0.12 to 0.91) $P = .03$ More vegetative outcomes in survivors with DC: 5.12 (95% CI 1.04 to 25.2) $P = .04$	Improved mortality and favorable outcomes with late DC Mortality: DC vs medical management 6 mo 16.9% vs 48.9% 12 mo 30.4% vs 52.0% GOS-E 12 mo (DC N = 185 % / Medical N = 175 %) Death 30.4 / 52.0 Vegetative 6.2 / 1.7 Lower severe disability 18.0 / 14.0 Upper severe disability 13.4 / 3.9 Lower moderate disability 10.3 / 7.8 Upper moderate disability 11.9 / 12.3 Lower good recovery 19.7 / 3.9 Upper good recovery 2.6 / 4.5 DC "patients had fewer hours than medical patients with ICP above 25 mm Hg after randomization (median, 5.0 vs 17.0 h; $P < .001$) but had a higher rate of adverse events (16.3% vs 9.2%", $P = .03$).

Level IIA recommendations – to improve mortality and overall outcomes.
1. NEW–Secondary DC performed for late refractory ICP elevation is recommended to improve mortality and favorable outcomes.
2. NEW–Secondary DC performed for early refractory ICP elevation is not recommended to improve mortality and favorable outcomes.

N.B. recommendation #2 should not be extrapolated to primary DC in which the bone flap is left off when an intracranial mass lesion is evacuated early after injury.

3. A **large frontotemporoparietal DC (not less than 12 × 15 cm or 15 cm in diameter)** is recommended over a small frontotemporoparietal DC for reduced mortality and improved neurological outcomes in patients with severe TBI.
4. NEW–Secondary DC, performed as a treatment for either early or late refractory ICP elevation, is suggested to reduce ICP and duration of intensive care, though the relationship between these effects and favorable outcome is uncertain.

Removed recommendation: "***Bifrontal DC is not recommended*** to improve outcomes as measured by the Glasgow Outcome Scale-Extended (GOSE) score at 6 months post-injury in severe TBI patients with diffuse injury (without mass lesions), and with ICP elevation to values > 20 mm Hg for more than 15 minutes within a 1-h period that are refractory to first-tier therapies. However, this procedure has been demonstrated to reduce ICP and to minimize days in the intensive care unit (ICU)."

The most important conclusion of these studies is that ***choosing to perform a DC is not a simple decision*** and that the potential benefits should be balanced against the complications and likely outcomes ***on a case-by- case basis*** (e.g. doing MRI to determine brainstem injury).

Unanswered questions:

1) relative risks and benefits of ***lateral DC vs bifrontal DC*** (should DC be tailored to the intracranial pathology?)

2) role of ***primary DC*** - ongoing RESCUE-SDH RCT will help with high-quality data.

RESCUEicp (Randomized Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intracranial Pressure) trial

*Hutchinson PJ et al. RESCUEicp Trial Collaborators Trial of Decompressive Craniectomy for Traumatic Intracranial Hypertension. N Engl J Med. **2016** Sep 22;375(12):1119-30. doi: 10.1056/NEJMoa1605215. Epub 2016 Sep 7.*

- 408 patients (10-65 yrs).
- conducted in 20 countries over a 10-yr period and included 73 medical centers.
- refractory ICP > 25 mm Hg for 1-12 hours.
- surgery was associated with better ICP control, reduced mortality, and higher rates of worse functional outcomes at 6 months (although this improved at 12 months)

Lifesaving procedures may not restore normal functions.
Maximal medical therapy may not be adequate in ICP control.

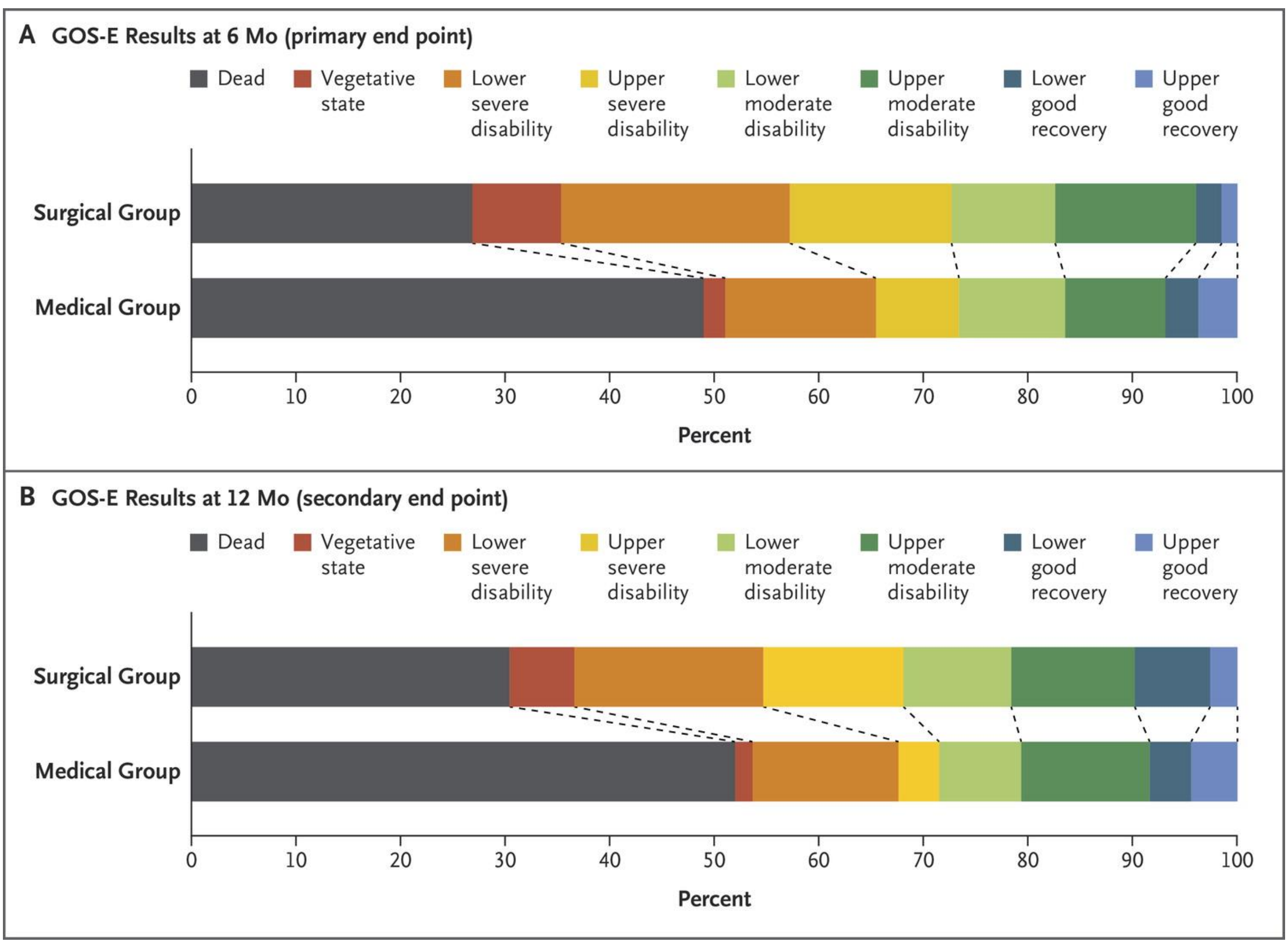


Table 3. Analysis of Primary and Secondary Outcomes.*

Variable	Surgical Group (N = 202)	Medical Group (N = 196)	Absolute Difference (95% CI)† *percentage points*	P Value
GOS-E result — no./total no. (%)‡				<0.001
At 6 mo				
Death	54/201 (26.9)	92/188 (48.9)	−22.1 (−31.5 to −12.7)	
Vegetative state	17/201 (8.5)	4/188 (2.1)	6.3 (2.0 to 10.7)	
Lower severe disability	44/201 (21.9)	27/188 (14.4)	7.5 (−0.1 to 15.1)	
Upper severe disability	31/201 (15.4)	15/188 (8.0)	7.4 (1.1 to 13.8)	
Lower moderate disability	20/201 (10.0)	19/188 (10.1)	−0.1 (−6.1 to 5.8)	
Upper moderate disability	27/201 (13.4)	18/188 (9.6)	3.9 (−2.5 to 10.2)	
Lower good recovery	5/201 (2.5)	6/188 (3.2)	−0.7 (−4.0 to 2.6)	
Upper good recovery	3/201 (1.5)	7/188 (3.7)	−2.2 (−5.4 to 1.0)	
At 12 mo				<0.001
Death	59/194 (30.4)	93/179 (52.0)	−21.5 (−31.3 to −11.8)	
Vegetative state	12/194 (6.2)	3/179 (1.7)	4.5 (0.6 to 8.4)	
Lower severe disability	35/194 (18.0)	25/179 (14.0)	4.1 (−3.3 to 11.5)	
Upper severe disability	26/194 (13.4)	7/179 (3.9)	9.5 (3.9 to 15.1)	
Lower moderate disability	20/194 (10.3)	14/179 (7.8)	2.5 (−3.3 to 8.3)	
Upper moderate disability	23/194 (11.9)	22/179 (12.3)	−0.4 (−7.1 to 6.2)	
Lower good recovery	14/194 (7.2)	7/179 (3.9)	3.3 (−1.3 to 7.9)	
Upper good recovery	5/194 (2.6)	8/179 (4.5)	−1.9 (−5.7 to 1.9)	
GCS score or death at discharge from ICU — no./total no. (%)§				<0.001
Death	42/185 (22.7)	83/171 (48.5)	−25.8 (−35.5 to −16.2)	
GCS score				
3–5	13/185 (7.0)	11/171 (6.4)	0.6 (−4.6 to 5.8)	
6–8	22/185 (11.9)	10/171 (5.8)	6.0 (0.2 to 11.9)	
9–12	67/185 (36.2)	37/171 (21.6)	14.6 (5.3 to 23.9)	
13–15	41/185 (22.2)	30/171 (17.5)	4.6 (−3.6 to 12.9)	
Intracranial-pressure control¶				
Median mean intracranial pressure after randomization (IQR) — mm Hg	14.5 (1.7–18.0)	17.1 (4.2–21.8)	−3.0 (−4.1 to −1.8)	<0.001
Median duration of intracranial pressure >25 mm Hg after randomization (IQR) — hr	5.0 (0.0–17.0)	17.0 (5.0–35.0)	−8.0 (−12.0 to −5.0)	<0.001
Median intracranial hypertension index 20 (IQR)	18.1 (9.9–36.7)	31.4 (18.2–54.2)	−10.4 (−14.5 to −6.7)	<0.001
Median intracranial hypertension index 25 (IQR)	6.6 (3.1–13.6)	11.8 (5.6–27.8)	−4.2 (−6.2 to −2.5)	<0.001
Median cerebral hypoperfusion index 60 (IQR)	6.8 (3.1–16.6)	11.1 (4.4–24.8)	−2.8 (−4.9 to −1.0)	0.002

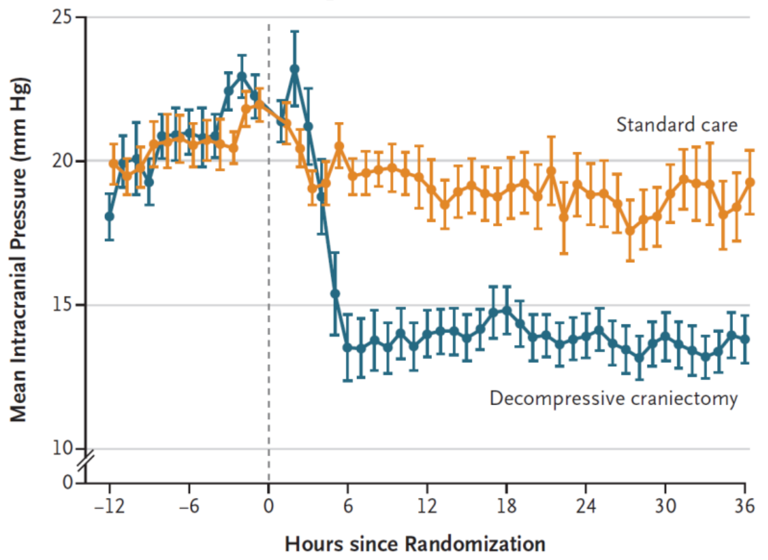
* ICU denotes intensive care unit, and IQR interquartile range.
† Absolute differences between percent values are percentage points and may not sum exactly owing to rounding. For median values, the treatment groups were compared with the use of the Mann–Whitney U test and the corresponding confidence interval. The estimated difference between the median values is not simply the observed difference between the median values.
‡ P values for the comparisons of the Extended Glasgow Outcome Scale (GOS-E) results were calculated by means of unordered chi-square tests. The eight outcome categories on the GOS-E are death, vegetative state (unable to obey commands), lower severe disability (dependent on others for care), upper severe disability (independent at home), lower moderate disability (independent at home and outside the home but with some physical or mental disability), upper moderate disability (independent at home and outside the home but with some physical or mental disability, with less disruption than lower moderate disability), lower good recovery (able to resume normal activities with some injury-related problems), and upper good recovery (no problems). See the Supplementary Appendix for additional descriptions of the outcome categories.
§ The GCS was used for assessing impairment of the level of consciousness. Scores range from 3 to 15, with lower scores indicating greater impairment. The P value was calculated by means of an unordered chi-square test.
¶ The mean intracranial pressure after randomization and the duration of intracranial pressure of more than 25 mm Hg after randomization could be calculated for 165 patients in the surgical group and for 160 in the medical group. The three indexes could be calculated for 192 patients in the surgical group and for 183 in the medical group. The intracranial hypertension index 20 is the number of end-hourly measures of intracranial pressure of more than 20 mm Hg divided by the total number of measurements, multiplied by 100. The intracranial hypertension index 25 is the number of end-hourly measures of intracranial pressure of more than 25 mm Hg divided by the total number of measurements, multiplied by 100. The cerebral hypoperfusion index 60 is the number of end-hourly measures of cerebral perfusion pressure of less than 60 mm Hg divided by the total number of measurements, multiplied by 100.

DECRA (Decompressive Craniectomy in Diffuse Traumatic Brain Injury)

Cooper DJ et al. Decompressive Craniectomy in Diffuse Traumatic Brain Injury. N Engl J Med 2011;364:1493-502

- Australian & New Zealand & Saudi Arabia multicenter trial: randomly assigned 155 patients (15-59 yo) with **severe diffuse TBI** (Marshall class III CT - moderate diffuse injury) and intracranial hypertension refractory to first-tier therapies:
  a) early (within 72 hrs) ***decompressive bifrontotemporoparietal craniectomy***
  b) standard ***medical management***.
- intracranial hypertension: spontaneous (not stimulated) ICP ≥ 20 mm Hg for > 15 minutes (continuously or intermittently, i.e. cumulative) within a 1-hour period.
- first-tier therapies: sedation, normalization of PaCO2, osmotherapy, neuromuscular blockade, and CSF drainage (EVD).
- second-tier options: mild hypothermia (to 35°C), barbiturates.
- exclusion criteria: not deemed suitable for full active treatment, dilated unreactive pupils, mass lesions (unless too small to require surgery), SCI, cardiac arrest at the scene of the injury.
- outcome at 6 months (evaluated by telephone by trained assessors who were unaware of study-group assignments):

— craniectomy led to lower ICPs and shorter ICU stays (no significant between-group difference in the total time in the hospital):



— *rates of death* were similar in the craniectomy group (19%) and the standard-care group (18%).

— craniectomy led to *neurological outcomes worse than with standard medical therapy* (70% vs. 51%; N.B. cannot be explained by more patients surviving in vegetative state as death rate was similar!):

Such difference disappeared after post hoc adjustment for pupil reactivity at baseline (i.e. harmful effect of craniectomy was no longer significant).

- obtaining lower ICP is not necessarily translated into improvement in clinical outcome (i.e. having lower ICP after procedure cannot be considered as "substantial benefit" to patient).
- benefit provided by lowering ICP was likely offset by surgical morbidity.

Pediatric trials – see p. TrH20 >>

TYPES

Primary DC – done during the craniotomy for hematoma evacuation (in anticipation of refractory ICP)

Secondary DC – done for refractory ICP.

1. **Frontotemporoparietal hemicraniectomy**
2. **Bifrontal craniectomy**
3. **Suboccipital craniectomy**
4. **Cushing's subtemporal decompression, temporal lobectomy (debridement of contused brain)**

- complete temporal lobectomy performed within 2 hours of the development of clinical signs of transtentorial herniation in 10 patients with unilateral hemispheric swelling and a GCS < 7* resulted in 40% functional independence.

  *all patients had displacement of the brainstem, compression of the contralateral peduncle, and progressive obliteration of the parasellar and interpeduncular cisterns on CT, along with fixed pupillary dilation.
  Nussbaum E, Wolf A, Sebring L, Mirvis S: Complete temporal lobectomy for surgical resuscitation of patients with transtentorial herniation secondary to unilateral hemispheric swelling. Neurosurgery 29:62–66, 1991

UNILATERAL VS. BIFRONTAL

1) even in cases of bifrontal contusions* good ICP control can be obtained with unilateral surgery.
   *surgery on side of larger intraparenchymal injury
2) in unilateral surgery, larger decompression can be obtained without manipulation or exposure of sagittal sinus.
3) unilateral surgery enables more extensive decompression low in temporal region.
4) opening into frontal sinuses (look at CT) can be more easily avoided in unilateral decompression.
5) cranioplasty after unilateral decompression is simpler and safer.
6) bifrontal approach is required only for bifrontal extraaxial mass lesions.

- hemicraniectomy can be performed bilaterally, however, it is difficult to position the head to do this.
- diffuse brain edema with no mass lesion or midline shift:
  a) nondominant hemisphere unilateral DC
  b) bifrontal DC.

BEFORE SURGERY

1) check coags!
2) type and cross **pRBC, FFP, platelets**
3) give AED, MANNITOL, ANTIBIOTIC (add 500 mg of METRONIDAZOLE if air sinus involved)
4) ventilate to $PaCO_2$ 25-30 mmHg
5) ask to have **plenty of hemostatic agents ready** (before incision!) – Surgicel, large pieces of Gelfoam, large amounts of Surgifoam/FloSeal (it takes time to mix them!!!), Avitene, peroxide soaked cotton balls or cottonoids.

PRINCIPLES

- **exposure of internal carotid artery in neck** for proximal control is always considered when injury to intracranial portion of artery is suspected.
- **large craniectomies** - to prevent brain strangulation over bone edges.
- minimal **brain debridement**.
- deep **bone fragments** should not be chased.
- **adequate brainstem decompression** - squamous portion of temporal bone and lateral greater wing of sphenoid are removed; anterior temporal lobectomy is performed if needed.

- adequate **venous drainage decompression** – craniectomy extends over vein of Labbé, over parasagittal bridging veins.
- **dural onlay substitutes** (e.g. DuraGuard) for dural closure – cover entire craniectomy defect (not just dural gaps) – prevents dura scarring to scalp flap (easier plane dissection during cranioplasty).
  N.B. dural opening must be large (some leave dura intact just excoriate its surface – inadequate for decompression)
- trauma patients are often coagulopathic - risk of EDH is high in the absence of the tamponading effects of a bone flap – leave 2 subgaleal drains!
- to prevent compression of large cortical veins as the brain herniates against the dura, may use **vascular tunneling technique (Csokay)** - place ***Gelfoam rolls*** as supporting pillars ***between the dura and cortex*** on each side of large cortical vessel traversing underneath the dura.
- galea and skin should be closed with closely spaced sutures to prevent CSF leak.

Bone flap management

a) wash with bacitracin irrigation, saturate with sterile solution (e.g. RPMI medium 1640 www. invitrogen.com/GIBCO), sterilely wrap and double bagged (e.g. intestinal bags which are then placed in a sterile plastic container), and *store at −30°F (−80 Celsius)*
b) implant into *subcutaneous pocket in abdominal left lower quadrant* to avoid contamination by feeding tube placement and to decrease confusion with appendectomy scar; indicated if the patient's own skull is preferred and the patient does not live in the area where he/she is having the surgery; bone flap may be split in half for patient's comfort; risk of resorption, rhabdomyolysis, and infection.
a) place back into skull defect without securing (*"floating" craniectomy*) – not recommended!
b) discard (if bone is fragmented or grossly contaminated)

History

- treatment of wartime penetrating head injury has evolved over the last century from Cushing's recommendation of aggressive brain debridement and watertight dural closure in World War I to minimal brain debridement with maximal surrounding bone removal instituted by Israeli physicians during the Lebanon War of 1982 with improvement in long-term seizure outcomes.

POSTOP

- **head CT** ASAP.
- after decompressive craniectomy, the new ICP treatment threshold is > 15 mmHg.
- patient need protective **helmet** if out of bed and a **sign at the bedside** "No bone flap".

COMPLICATIONS

Intraop complications

1) venous sinus injury – see below >>
2) precipitous external herniation (malignant cerebral edema) intraoperatively (soon after decompression) – see below >>

Postop complications

(up to 50-55%)

1) decompressive procedures may aggravate cerebral edema formation → increased secondary injury.
   Cooper PR, Hagler H, Clark WK, Barnett P: Enhancement of experimental cerebral edema after decompressive craniectomy: Implications for the management of severe head injuries. Neurosurgery 4:296–300, 1979
   – in one study, hemicraniectomy lowered ICP but resulted in significantly greater cerebral edema (attributed to reduction in the interstitial pressure within the brain after decompression, resulting in a greater hydrostatic pressure gradient between the intravascular and interstitial spaces).
   – "kinking" of cortical veins against bone edges (when bone flap is too small) may lead to further engorgement and venous ischemia.

2) expansion of hematomas after decompression.
   - ***skull fracture*** contralateral to side of decompression is significant risk factor for postoperative EDH - routine early postop CT should be considered in cases with skull fracture remote to site of decompression, esp. if no ICP monitor was placed.

3) **wound complications** are most common source of surgical morbidity:
   a) traumatic injury to skin at incision → nonhealing, dehiscence
   b) infection
   c) CSF leak (open durotomy + CSF absorption problems [CSF is absorbed passively due to pressure gradient, at least 5 mmHg, across arachnoid granulations, which is lost after craniectomy])

4) abnormalities in CSF absorption (subdural hygroma and hydrocephalus) – due to ruptured arachnoid creating a one-way valve for CSF flow, pressure gradients between hemispheres due to reduction of ICP and decompression of one hemisphere, and alterations in the brain's shape as a result of surgery.
   - CSF absorption is disturbed after craniectomy (CSF is normally absorbed passively due to pressure gradient, at least 5 mmHg, across arachnoid granulations, which is lost after craniectomy).
   - definitive treatment is cranioplasty; VPS may not be successful due to low ICPs.

5) syndrome of the trephined
   - fatigue, headache, mood disturbances, seizures, and even motor weakness.
   - mechanism underlying this syndrome is unknown (CBF or CSF flow abnormalities, direct atmospheric pressure on the brain).
   - symptoms improve in recumbency.
   - often resolves with replacement of the bone flap.
   - no evidence that it is harmful or that delay of cranioplasty can result in long-term consequences.

<u>Intracranial Pressure changes</u>

Lilja-Cyron et al. Long-Term Effect of Decompressive Craniectomy on Intracranial Pressure. Neurosurgery 86:231–240, 2020

- telemetric ICP sensors (Neurovent-P-tel; Raumedic, Helmbrechts, Germany) were implanted in 16 patients (traumatic brain injury: 7, stroke: 9) → scheduled weekly ICP monitoring sessions
- mean ICP gradually decreased from 7.8 ± 2.0 mmHg to – 1.8±3.3mmHg (P=.02) during the first month.
- normal postural ICP change was abolished for the entire follow-up period, ie, there was no difference between ICP in supine and sitting position (P = .67).
  — normally, ICP drops by 5-10 mmHg going from supine to standing
- intracranial pulse wave amplitude (PWA) was markedly reduced and decreased from initially 1.2 ± 0.7 mm Hg to 0.4 ± 0.3 mm Hg (P = .05).

— using phase contrast MR imaging, it has previously been shown that pulsatile CSF flux in the cerebral aqueduct is absent following DC.

- these abnormalities might have implications for intracranial fluid movements (e.g. CSF and/or glymphatic flow).
  — pulse wave propagation is believed to be an important driver of CSF flow and emerging evidence also supports its role in other intracranial fluid movements, such as glymphatic flow; hence, the reduced intracranial PWA may contribute to development of complications related to CSF flow (hygroma, hydrocephalus, "syndrome of the trephined") occurring after DC

Subsequent cranioplasty also carries the risk of infection, cerebral swelling, and resorption of the bone flap.

DECOMPRESSIVE HEMICRANIECTOMY ("TRAUMA FLAP") / FRONTOTEMPORPARIETAL CRANIECTOMY

PLANNING

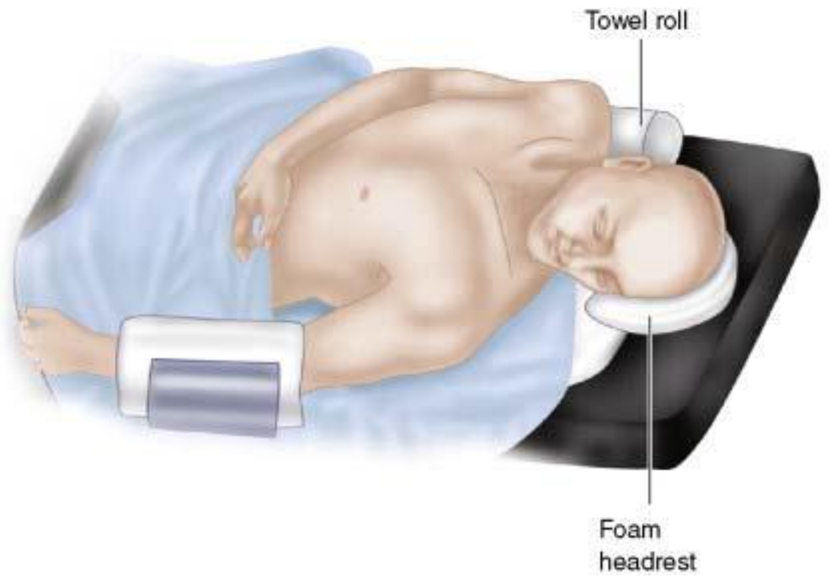
- patient is positioned supine.
- **towel roll** under ipsilateral shoulder.
- **head turned** to contralateral side (do not compress jugular veins!).

N.B. in setting of trauma, it is important to position patient with ***cervical spine precautions*** (if cervical spine fracture – position patient on bean bag)

HEAD SUPPORT

B) head on foam **headrest / gel donut** - allows for repositioning of head intraoperatively if venous outflow obstruction is suspected; but pinning in Mayfield frame is most convenient

N.B. it is very important to make ***large craniectomy but not to damage venous sinuses*** – draw midline and transverse sinus projection on scalp (thus, highly recommended to shave the scalp to have good landmarks; if leaving hair on [e.g. in less emergent cases, such as MCA stroke], mark scalp and incision before pining the patient and while keeping head in neutral position)



Source of picture: R. Jandial "Core Techniques in Operative Neurosurgery: Expert Consult - Online and Print", 1st ed (2011), Saunders; ISBN-13: 978-1437709070 >>

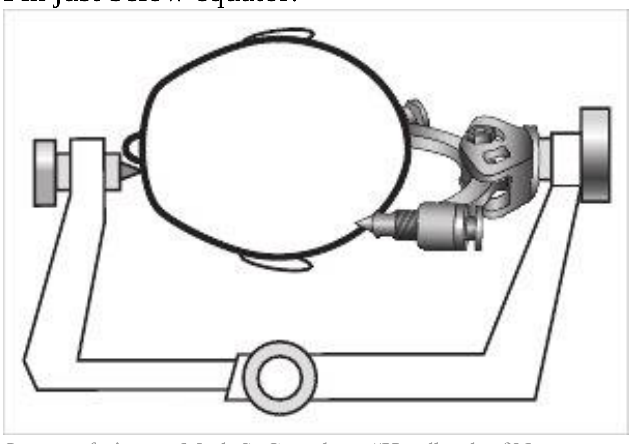
C) Dr. JRC likes to **pin** the patient – greater access and stability (e.g. do not need assistant to stabilize head when cutting with footplate or elevating bone flap).

- AP axis of head is placed horizontal to floor (unless C-spine not cleared or if neck too immobile – one may compensate for this by rotating table)

N.B. pins are contraindicated in skull fractures (skull fractures parallel to the plane of CT imaging can be missed)

N.B. ***mark incision (esp. midline) before*** pining the patient (see above) or else it is difficult to see midline.

Pin just below equator:



Source of picture: Mark S. Greenberg "Handbook of Neurosurgery" 8th ed. (2016); Publisher: Thieme Medical Publishers; ISBN: ISBN 978-1-62623-241-9 eISBN 978-1-62623-242-6 >>

Draping

- it is easy to get off **midline** in emergency setting (especially if head is not pinned); H: mark skin in midline → place drapes up to midline so that you are always oriented to midline; watch for sagittal suture intra-operatively.
- prepping contralateral Kocher point for EVD can save some time after case.

PROCEDURE

SKIN INCISION

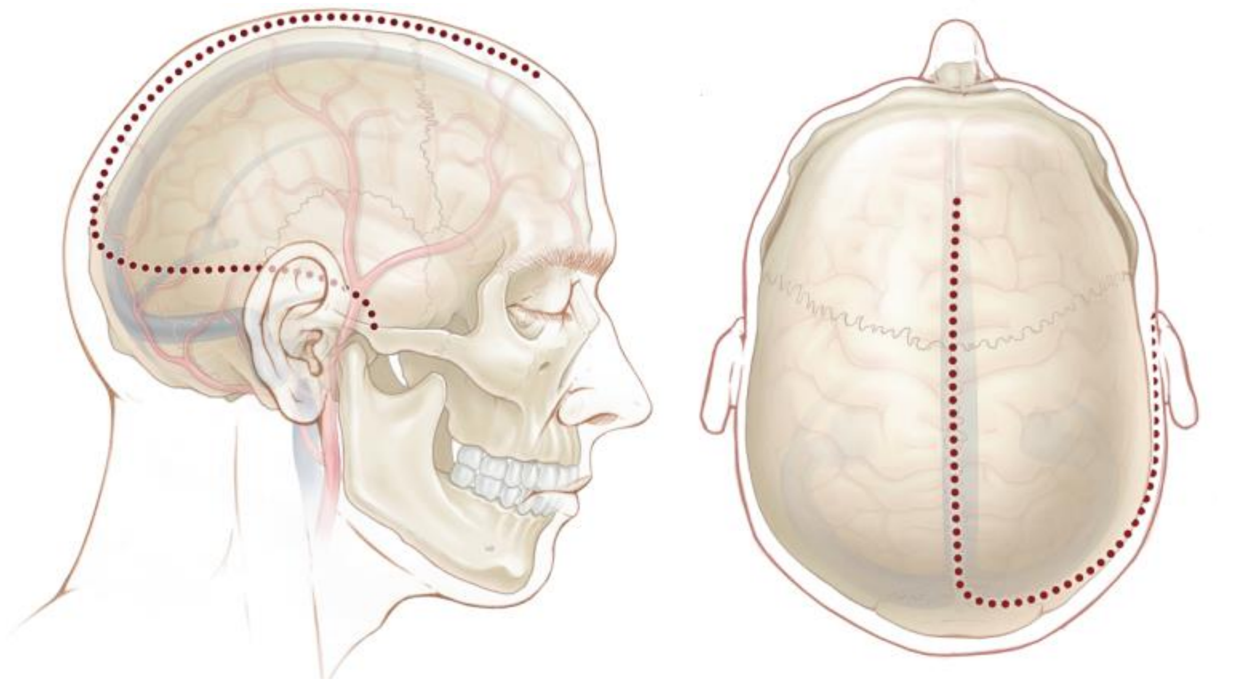
- protect superficial temporal artery - to preserve blood supply to flap.
- incorporate scalp lacerations if feasible.

In rapidly deteriorating patient with acute SDH/EDH, immediate temporal decompression is performed by incising skin and temporalis muscle down to bone just anterior to ear and above zygoma → burr hole and, if necessary, small craniectomy are created to partially decompress temporal lobe, before entire skin incision is completed.

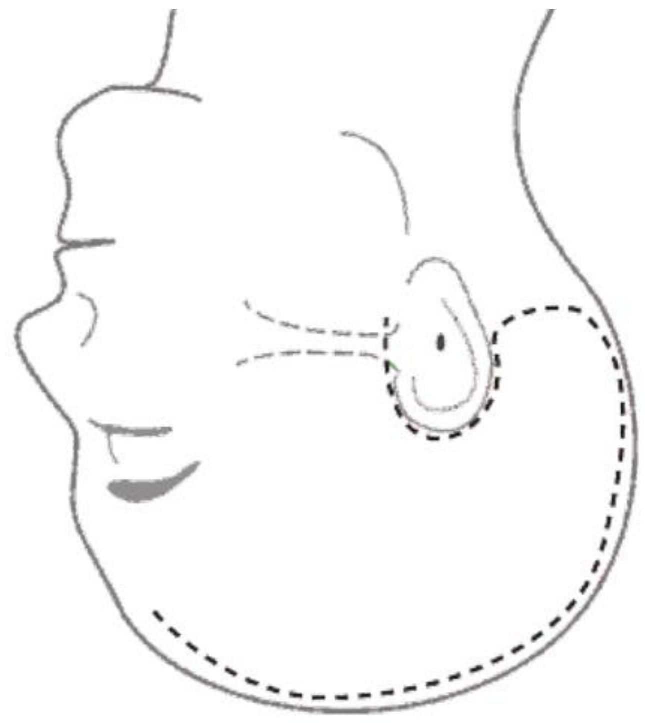
A. Large (REVERSE) QUESTION MARK:

— starts 1-2 cm anterior to tragus at temporal root of zygoma, curves posteriorly above and gently behind ear (or just superior to pinna) to asterion;
— posterior extent of the incision should be more than 15 cm behind the keyhole.
— sweeps around parietal boss to few centimeters lateral* to sagittal suture and forward to widow's peak;
— may cross over to opposite frontal region in curvilinear fashion along hairline for 3-4 cm.

*N.B. one wants scalp incision away from craniotomy edge – postop scalp shrinks and drags scar over dura (cranioplasty is easier if scalp scar is over the bon). Plus, it is safer to mark / make incision paramedian and not in midline (to leave margin of error if midline was not clearly marked, plus, during surgery it may be difficult to visualize sagittal suture); posterior corner may hug the lambdoid suture



Source of picture: Brian T. Ragel et al. "Wartime decompressive craniectomy: technique and lessons learned". Neurosurg Focus / Volume 28 / May 2010 >>



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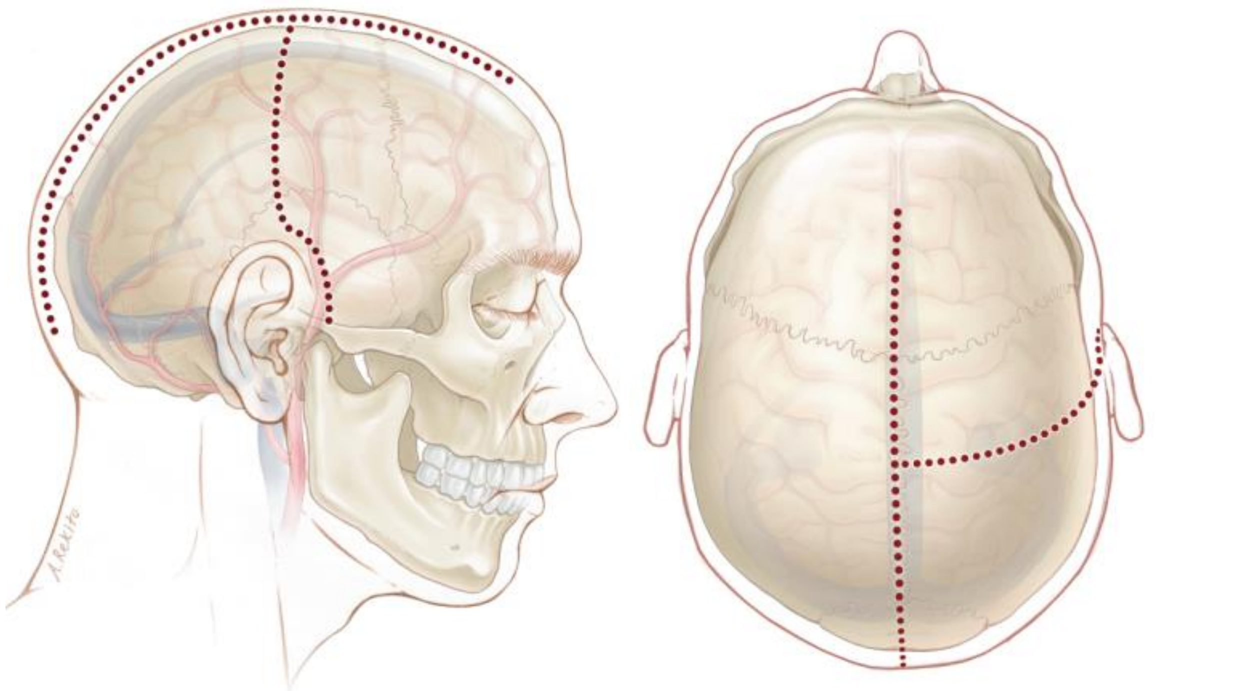
B. Ludwig G. Kempe hemispherectomy incision (midline sagittal incision with "T-bar" extension)
- spares STA, posterior auricular and occipital arteries, unlike large reverse question mark incision:
— starts at widow's peak and is carried posteriorly along the sagittal suture to inion;
— "T-bar" extension is started 2 cm anterior to tragus at temporal root of zygoma, curving slightly above ear and then incised superiorly to meet midline sagittal suture (approximately 1 cm behind the coronal suture)
— advantages over "?" craniotomy - much better healing (preserved vascular supply, no pressure on posterior incision), also allows for easy surgical access to contralateral side by placing second "T-bar" extension if bilateral surgical access is needed.



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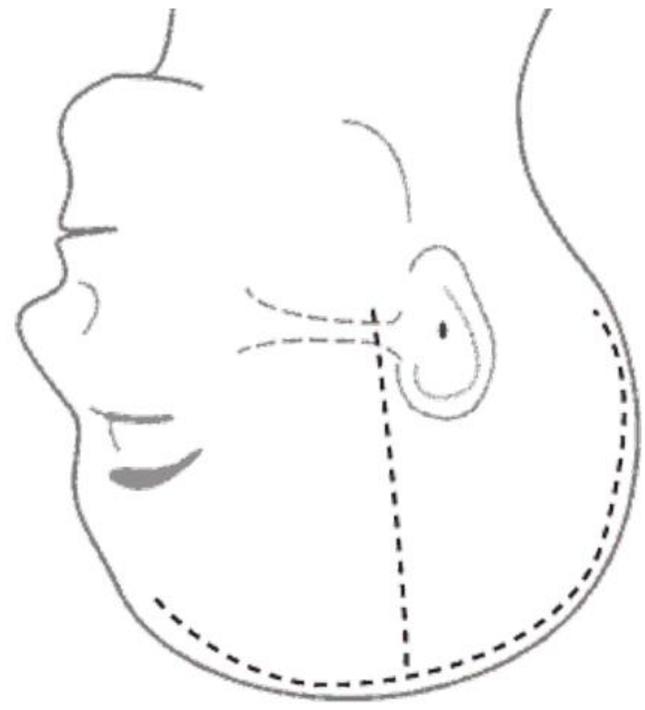

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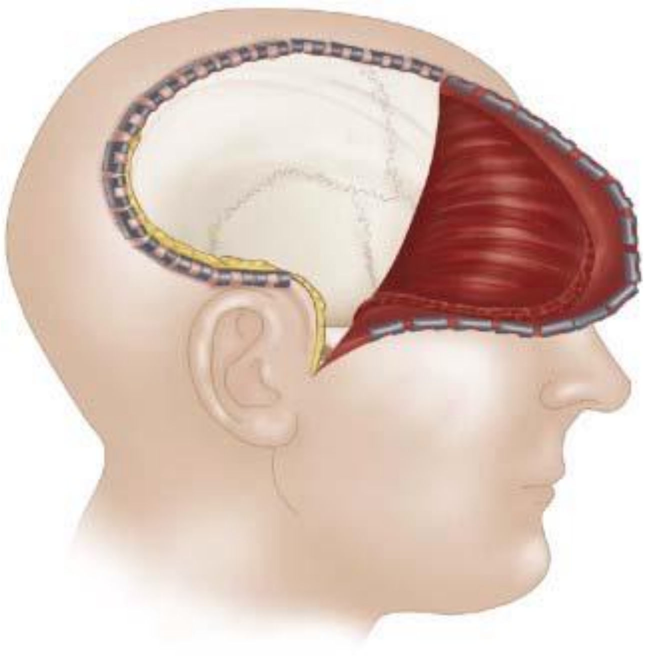
MUSCLE AND SOFT TISSUE DISSECTION

- skin incision is carried down to cranium; incise temporalis muscle with Bovie along skin incision.

- expose superior portion of temporalis fascia (along temporal line) and make incision leaving fascial cuff for reconstruction (only if planning to place the bone flap back at the end of procedure; otherwise elevate full thickness myocutaneous flap leaving naked bone).
- musculocutaneus flap is reflected anteriorly (i.e. temporalis muscle is elevated off bone using periosteal / Bovie) and fixed with scalp hooks/towel clamps.
- ideally, muscle dissection extends down to root of zygoma and as far below keyhole as possible, to maximize temporal decompression achieved.



Source of picture: R. Jandial "Core Techniques in Operative Neurosurgery: Expert Consult - Online and Print", 1st ed (2011), Saunders; ISBN-13: 978-1437709070 >>

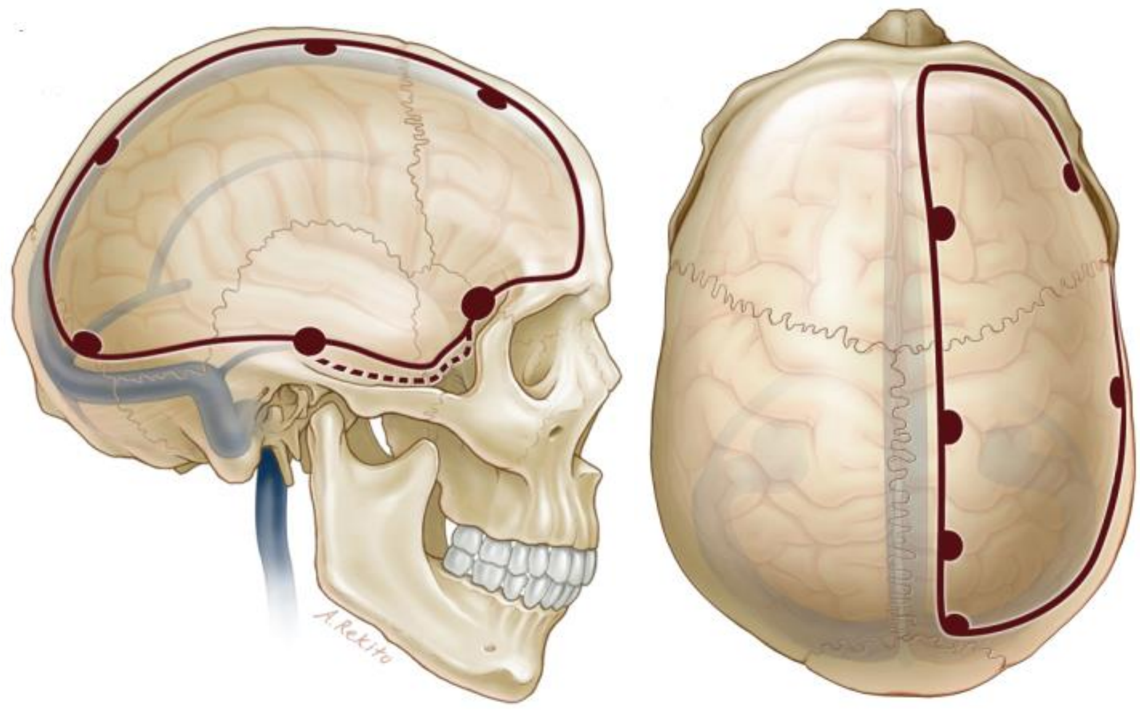
BURR HOLES AND BONE FLAP

- may use *14 mm perforator* at **calvarium** – quick; at **keyhole** and **zygoma** need to use *Acorn drill bit* as bone there is very thin (until perforator reaches disengagement depth, dura is already shredded).
- bur holes placed at:
  1) pterion or keyhole (exposing frontal and temporal dura) – absolutely necessary
  2) just above the posterior root of zygoma – absolutely necessary (all other but holes are optional)
  3) about 1-2 cm above asterion (to avoid transverse sinus)
  4) inferior to parietal boss
  5) superior to parietal boss
  6) additional one or two bur holes 2 cm off midline on ipsilateral side of craniectomy.
- bone flap perimeter: hug the floor of middle fossa above mastoid air cells to get as low as possible (beware transverse sinus) → extending back (1 cm beyond the lambdoid suture*) → around parietal boss (crossing lambdoid again) toward midline, leaving 2-cm lip of bone adjacent to sagittal suture → coronal suture is crossed and the drill is taken as low as possible in the frontal fossa near the midline → staying as low as possible, the orbital roof is followed posteriorly towards the keyhole burr hole.

  *this leaves a small amount of bone posteriorly on which the head can rest post-op

  — decompressive craniectomy led to significant improvement in the visibility of mesencephalic cisterns and a significant decrease in midline shift; this change in cistern visibility correlated with the *distance between the lower craniectomy border and cranial base*.

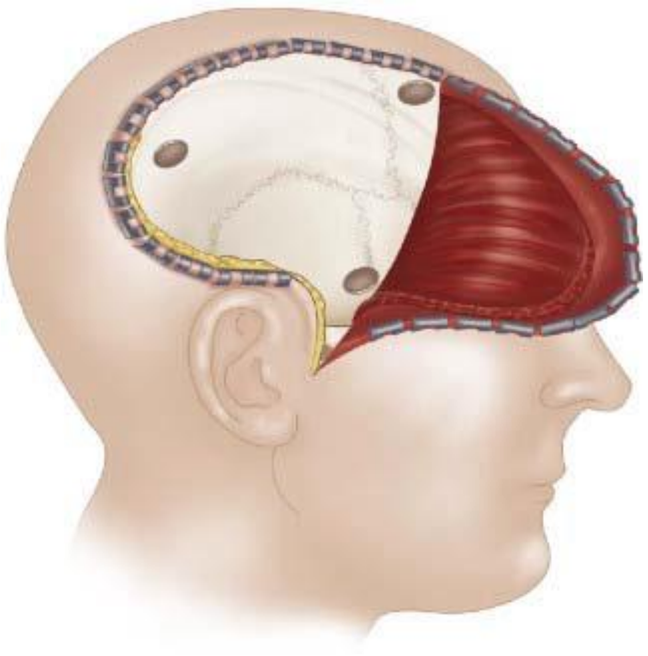
  Munch E et al: Management of severe traumatic brain injury by decompressive craniectomy. Neurosurgery 47:315–322, 2000



Source of picture: Brian T. Ragel et al. "Wartime decompressive craniectomy: technique and lessons learned". Neurosurg Focus / Volume 28 / May 2010 >>

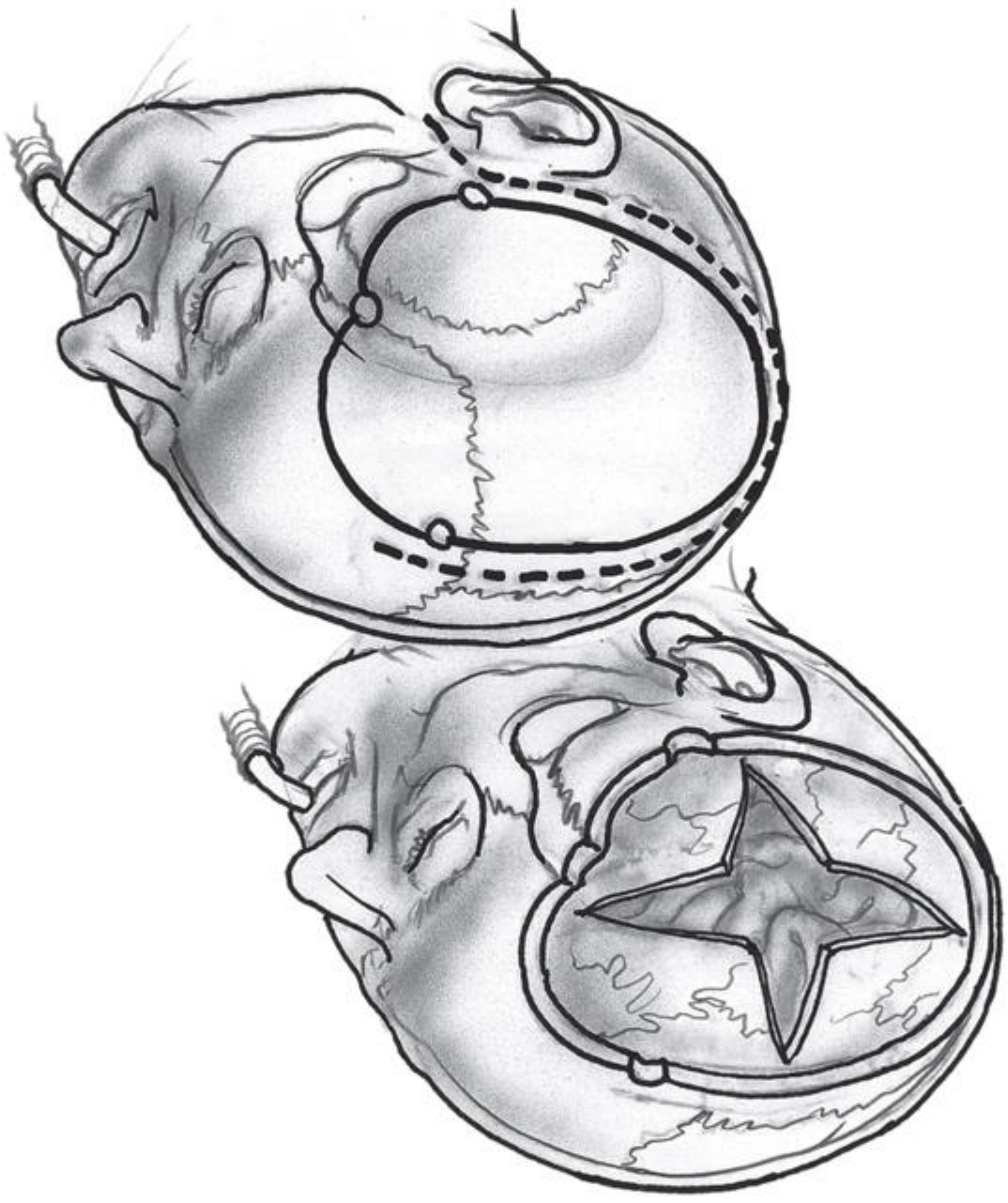
- several burr holes (at least three) are made to create bone flap that is at least 12 cm × 15 cm (smaller bone flaps would not sufficiently decompress brain but may be sufficient for EDH or SDH evacuation when brain edema is not expected) – use ruler to measure back from keyhole to ensure anteroposterior extent of bone flap is 15 cm.

  Tanrikulu et al. compared different sizes of DC (12–15, 15–20, and 20–24 cm) and found no additional benefit in wider craniectomies provided that the lower limit of 12 cm is observed.
- large burr hole is placed in temporal squamosa at zygoma root; additional burr holes can be placed posterior (parietal) and 1.5 cm off midline (frontal):



Source of picture: R. Jandial "Core Techniques in Operative Neurosurgery: Expert Consult - Online and Print", 1st ed (2011), Saunders; ISBN-13: 978-1437709070 >>

- flap should extend 1.5 cm from midline, 1-2 cm above transverse sinus – to decompress vein of Labbé, parasagittal bridging veins

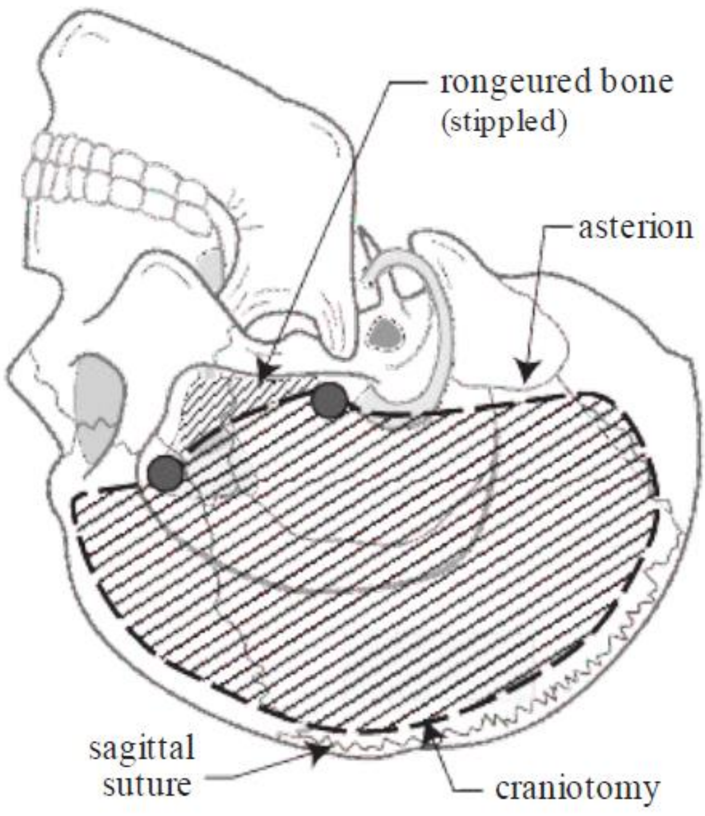


Source of picture: E. Sander Connolly "Fundamentals of operative techniques in neurosurgery" (2010); Publisher: Georg Thieme Verlag; ISBN-10: 1588905004; ISBN-13: 978-1588905000 >>

- in especially urgent cases (recent anisocoria and underlying SDH), making cruciate opening in dura through first burr hole may provide some relief of intracranial hypertension.
- ***dura over anterior frontal lobe*** is commonly torn (typically in emergencies and older patients) - it is good practice to assume that dura may be incompetent in frontal area and to begin to strip dura and elevate bone flap away from this site.
- Dr. Villanueva makes parasagittal bone cut from front to back (rationale – dura is more commonly is damaged at the beginning of cut – so better to tear SSS anteriorly).
- fate of bone flap – see above >>

TEMPORAL FLOOR

- after removal of bone flap, remaining TEMPORAL BONE must be cut with rongeur down to floor of middle cranial fossa (subtemporal craniectomy) to provide maximal decompression of lateral brainstem.



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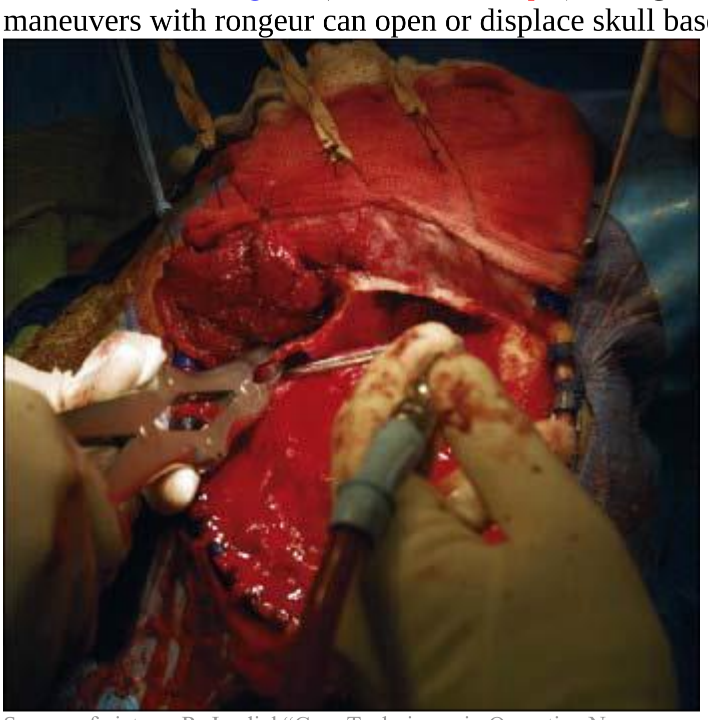
N.B. ***BITE with rongeur*** (not twist or torque) during bone removal low in middle fossa; aggressive maneuvers with rongeur can open or displace skull base fractures → uncontrolled bleeding.



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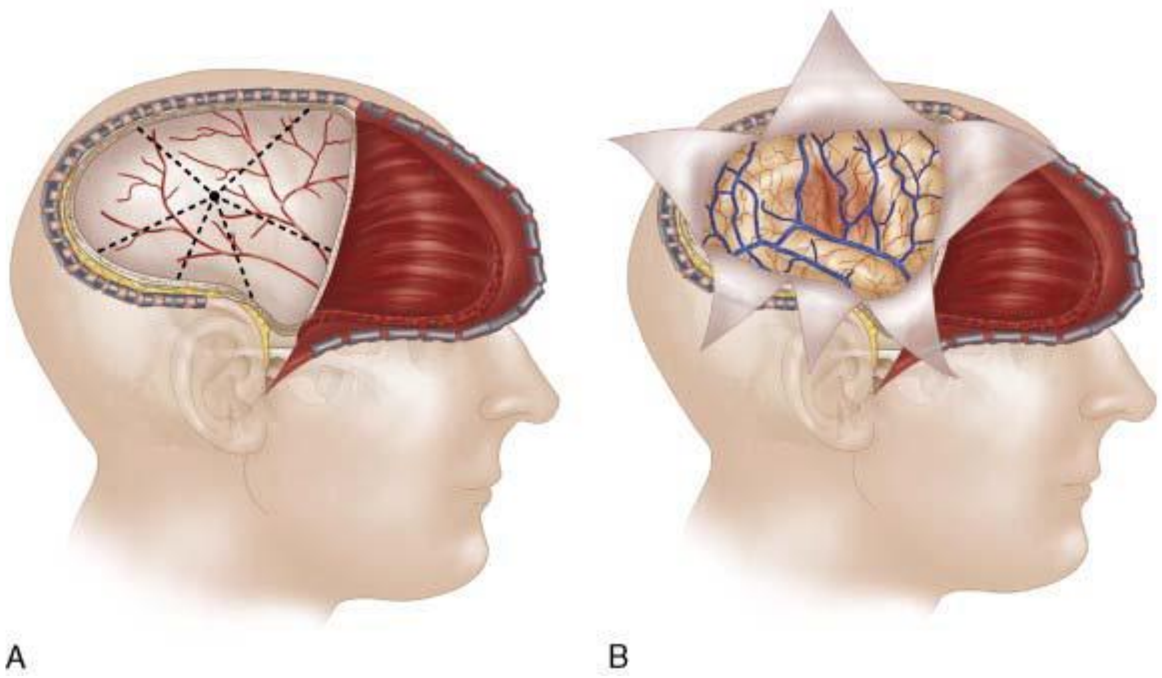
- sphenoid wing is removed with rongeurs or high-speed drill

- wax bone edges.
- line cranial edges with hemostatic material (e.g. Surgicel) and Cottonoids.
- large amounts of bleeding arising from the middle cranial fossa warrant further attention and usually come from the middle meningeal artery or the sphenoid wing - in this situation, a ***slightly more conservative temporal craniectomy*** can provide bone to which the dura can be tacked to help tamponade such bleeding.

DURAL OPENING

- several choices:

c) only vertical slit incisions ("pie crusting") – if expect malignant cerebral edema (e.g. in GSW) so will be difficult to close even scalp flap; slide brain ribbon through slit and then suction tip over brain ribbon – evacuate clot; if brain looks collapsed after clot evacuation, may connect cuts by horizontal dural cut (so result is H).
d) H incisions
e) C flap
f) ***C-shaped with spoke-wheel relief cuts*** 1-cm short of the craniotomy edge with dural releasing incisions made at intervals up to the bone margin to avoid strangulation of the brain on the dural edge
a) ***multiple radial incisions*** (in stellate fashion) to provide maximal cerebral decompression:



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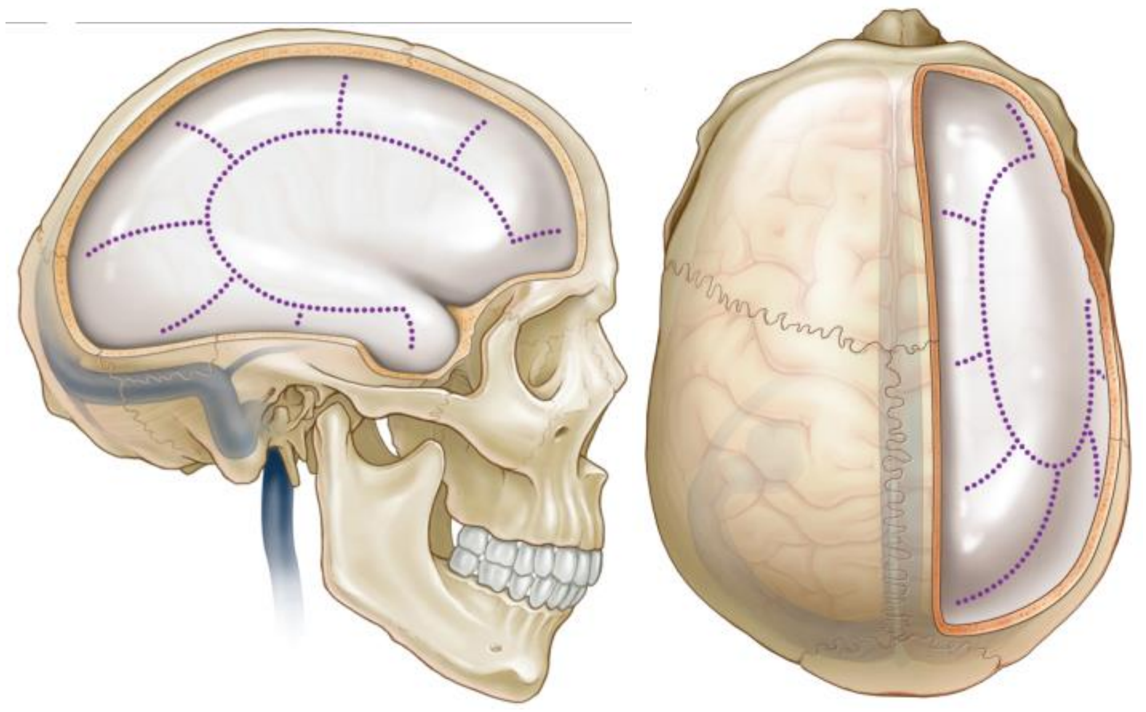

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- open dura slowly (!!! – risk of sudden cardiovascular collapse; H: adequate resuscitation by anesthesia - central line is a must).
- remove hematomas (gentle scraping and sucking), obtain hemostasis (bipolar), irrigate copiously.
  N.B. no attempt should be made to chase elusive pieces of clot in subdural space!
- ***arterial blood exiting from middle fossa*** in large amounts warrants exploration and often arises from middle meningeal artery or sphenoid wing. H: slightly more conservative temporal craniectomy provides bone to which temporal dura can be tacked, which may stop bleeding; consider waxing foramen spinosum, peroxide soaked cotton balls or cottonoids.
- large frontal and temporal **contusions** can be removed with gentle suction and bipolar cautery.
- ultrasound can be used to identify **intracerebral hematomas** that do not come to surface.

CLOSURE

- using dural substitute (DuraGuard, SepraFilm).

Options

A. Duraplasty – suturing graft to dural edges.
B. Leave durotomy open to permit brain expansion ("rapid-closure DC"); cover brain with onlay dural substitute to protect brain surface and reduce adhesions to scalp flap - leaves of dura are folded over dural substitute (do not suture patch to dura!)

- **epidural tack-up sutures** to bone edge.
- **subgaleal drains** are placed over surface of dural substitute and tunneled externally; ***at least two Jackson-Pratt drains*** (patients often do not clot properly, and without tamponading effect provided by bone flap, risk of EDH is high)
  N.B. routine prolonged use of drains is needed!?
- galea is closed with numerous, closely spaced interrupted 2-0 absorbable braided sutures.
- skin is closed with running 4-0 absorbable monofilament suture / staples – to prevent CSF leak
  N.B. to ensure watertight closure, sutures are placed very close together!
- sterile **head wrap** (not tight if bone removed + label side of head without bone)

POSTOP

Head CT ASAP:

Decompressive hemicraniectomy. Note the extensive boney decompression from the frontal bone to the occiput and the remaining interhemispheric subdural hematoma. The brain parenchyma on the left can be seen protruding beyond the previous boney-boundary indicating how severe the intracranial hypertension was. The hyperdensity seen above the left frontal lobe is part of the Jackson-Pratt subgaleal drain:

Source of picture: Richard G. Ellenbogen, Saleem I. Abdulrauf, Laligam N. Sekhar "Principles of neurological surgery" 3rd ed (2012); Publisher: Saunders; ASIN: B007V2NOGM >>

DECOMPRESSIVE BIFRONTAL CRANIECTOMY (KJELLBERG)

pronounced "Shellberg"

R.N. Kjellberg, A. Prieto Jr.: Bifrontal decompressive craniotomy for massive cerebral edema. J Neurosurg. 34:488-493 1971

INDICATIONS

Level II A recommendation

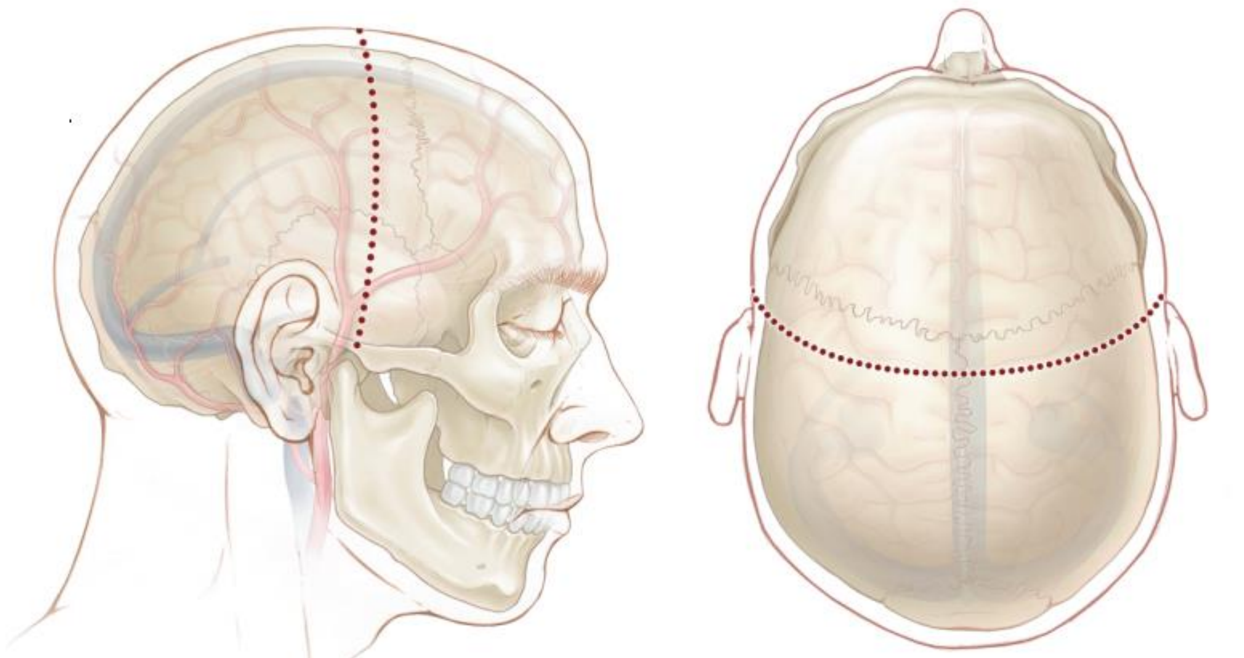
- bifrontal DC is *not recommended to improve outcomes* (as measured by GOS-E score at 6 months post-injury in severe TBI patients with diffuse injury and with ICP > 20 mm Hg for > 15 minutes within a 1-hour period that are refractory to first-tier therapies).
- demonstrated to reduce ICP and to minimize days in ICU.

PROCEDURE

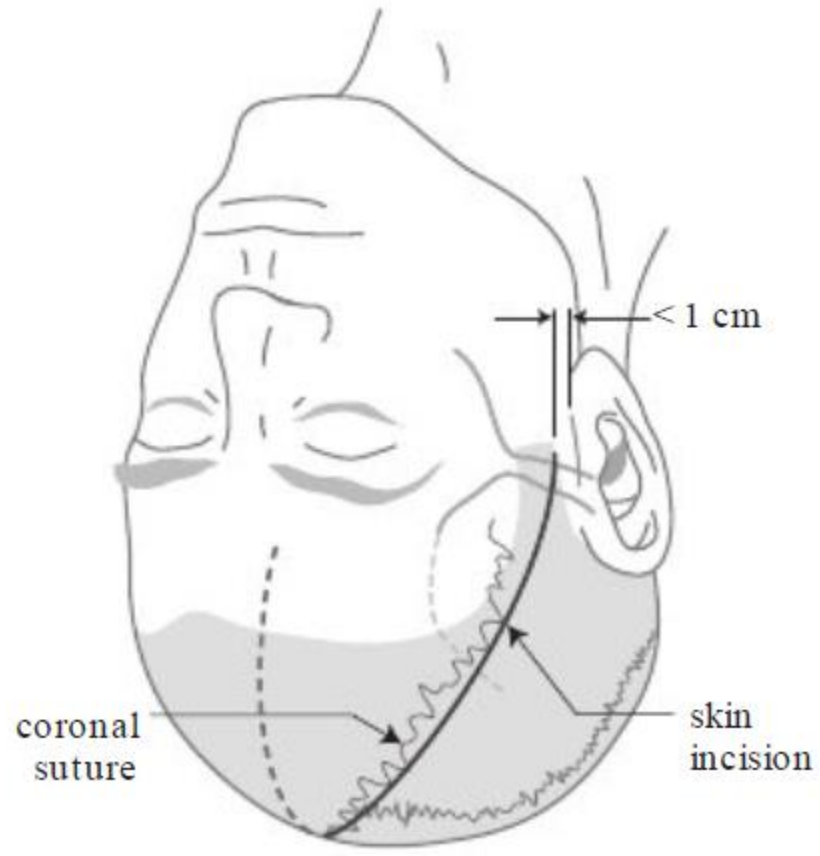
- supine position with the head in 15-30 degrees of flexion.

INCISION

- bicoronal (Souttar) incision - starts at root of zygoma, 1-2 cm anterior to tragus, extends superiorly to or just behind coronal suture, and ends at root of opposite zygoma, 1-2 cm anterior to tragus.
- Dr. Villanueva uses wavy (vs. straight) incision – scar does not interfere with chewing.



Source of picture: Brian T. Ragel et al. "Wartime decompressive craniectomy: technique and lessons learned". Neurosurg Focus / Volume 28 / May 2010 >>



Source of picture: Mark S. Greenberg "Handbook of Neurosurgery" 8th ed. (2016); Publisher: Thieme Medical Publishers; ISBN: ISBN 978-1-62623-241-9 eISBN 978-1-62623-242-6 >>

- myocutaneous flap is brought forward over the orbital rim.
  – dissect out the *supraorbital nerves* from the supraorbital notch on either side (if the supraorbital notch is closed, a small osteotome can be used to open it so that the supraorbital nerve can be freed) - allows further advancement of the myocutaneous flap.

BONE

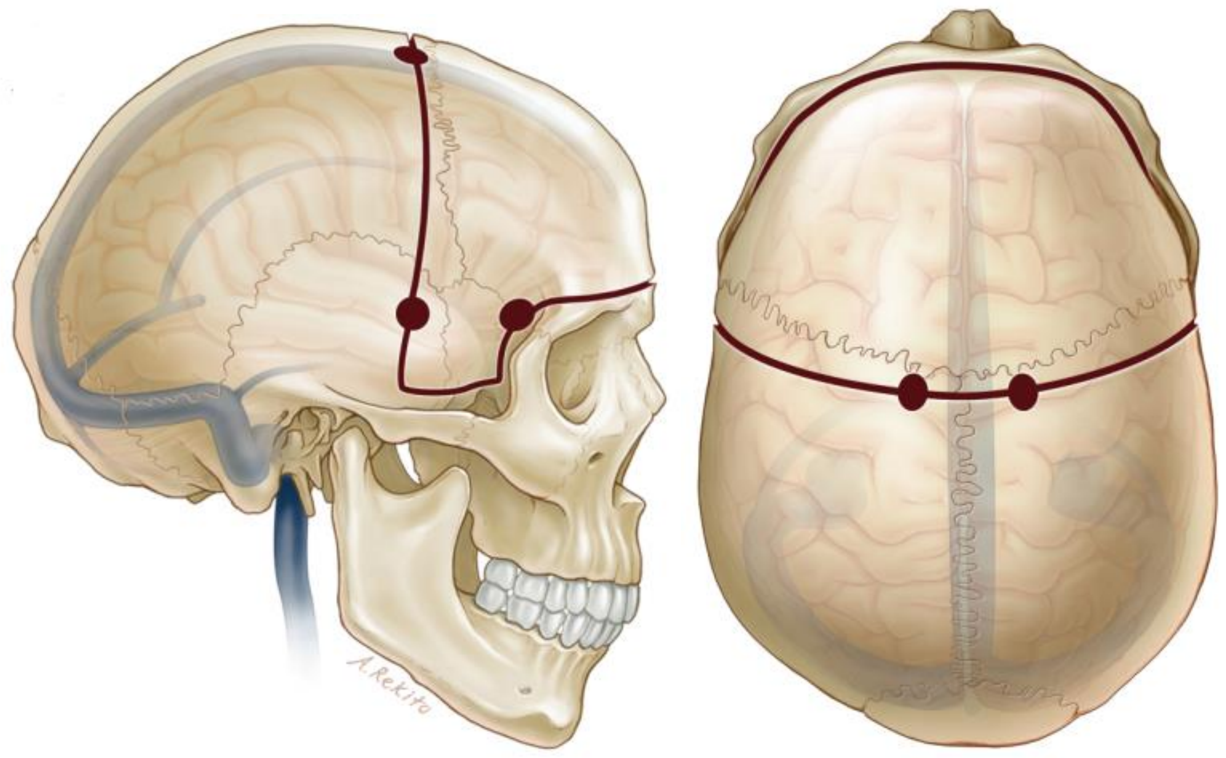
- bur holes are placed in:
  1) pterion (keyhole)
  2) root of zygoma

3) just below superior temporal line*
4) over superior sagittal sinus (last bur hole to make) – if a large single bone flap is planned**

* posteriorly – behind coronal suture!!! (Dr. Villanueva extends even further to parietal bossings – the adequate decompression of sagittal sinus).
** alternatively, strip of bone can be left over sagittal sinus for protection.

- bilateral frontal and subtemporal craniectomies are performed, exposing frontal and anterior temporal lobes.
- anteriorly, drill passes just above the orbital rims and nasal bridge.
- additional (optional) removal of squamous portion of temporal bone and greater wing of sphenoid bone is accomplished with rongeur, removing bone to floor of middle fossa.
- immediately after removing bone put lap pad with H_2O_2 over entire dura – air embolism prophylaxis (in case SSS was damaged) plus hemostatic.
- if frontal sinus is entered, it must be cranialized (dissect the flap of pericranium to cover it).

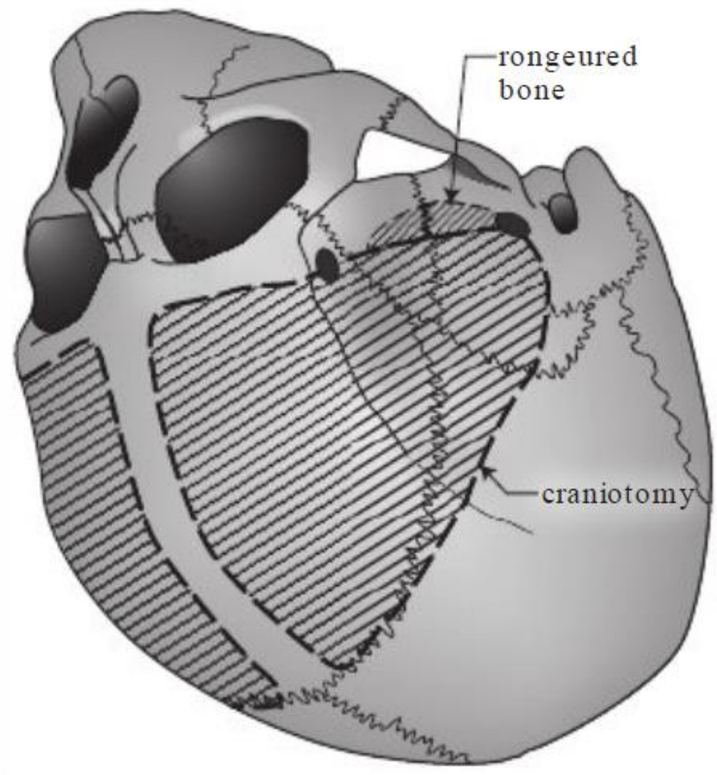
A. One bone flap:



Source of picture: Brian T. Ragel et al. "Wartime decompressive craniectomy: technique and lessons learned". Neurosurg Focus / Volume 28 / May 2010 >>

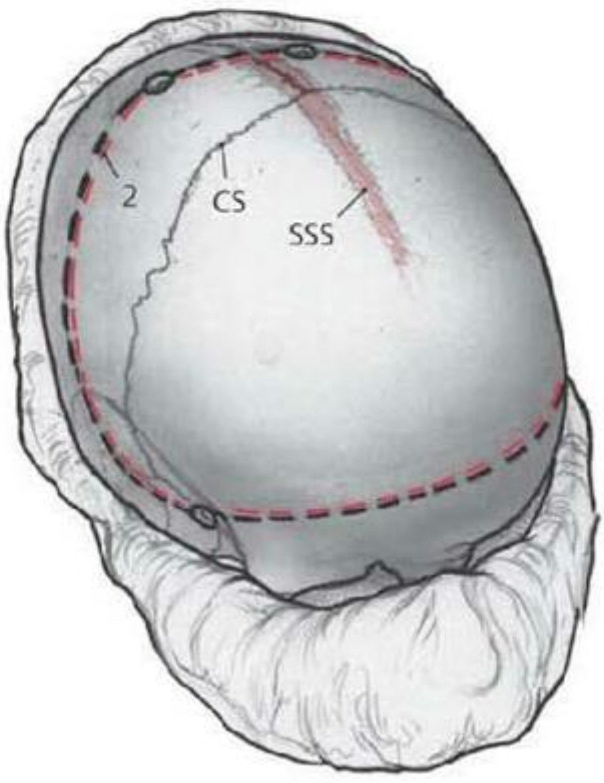
N.B. cross the superior sagittal sinus with the footplate last!

B. Two bone flaps:
N.B. if midline bone strip is too wide, it can damage the brain!



Source of picture: Mark S. Greenberg "Handbook of Neurosurgery" 8th ed. (2016); Publisher: Thieme Medical Publishers; ISBN: ISBN 978-1-62623-241-9 eISBN 978-1-62623-242-6 >>

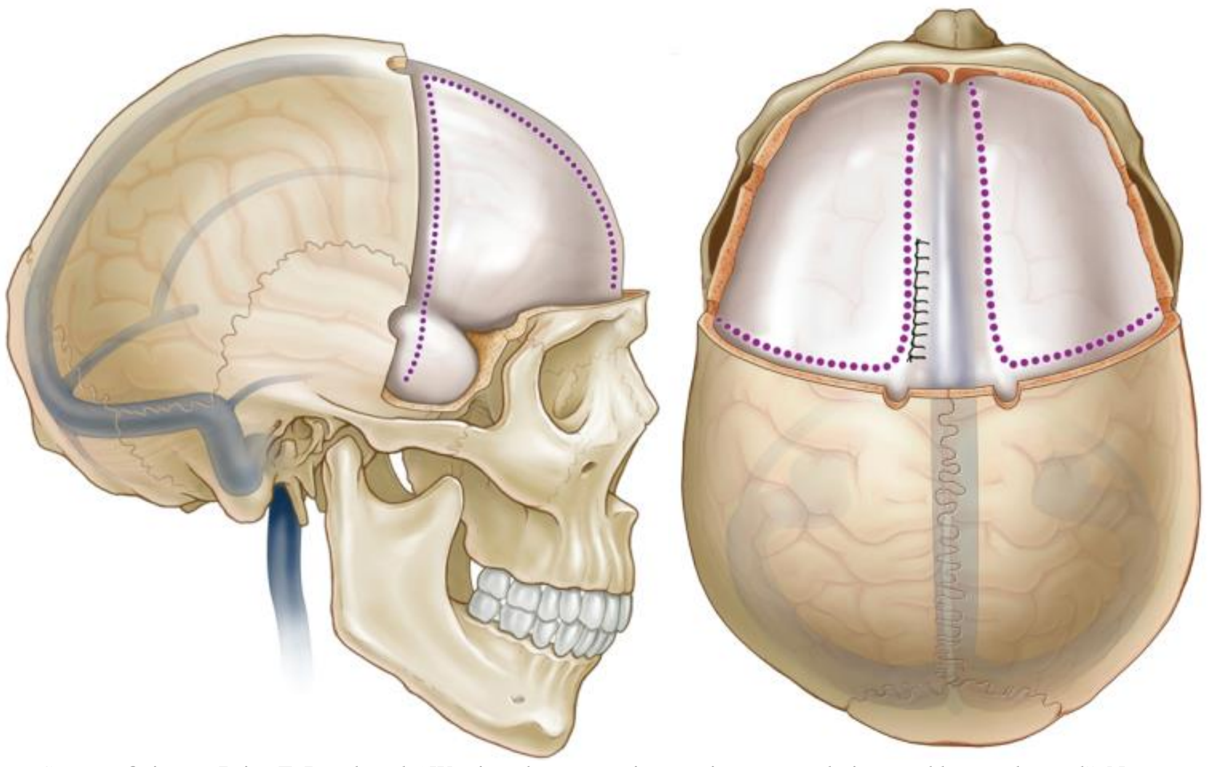
Variant without temporal decompression (avoiding zygomatic bur hole):



Source of picture: R. Nader "Neurosurgery Tricks of the Trade – Cranial" (2014); Publisher: Thieme; ISBN-10: 1604063343, ISBN-13: 978-1604063349 >>

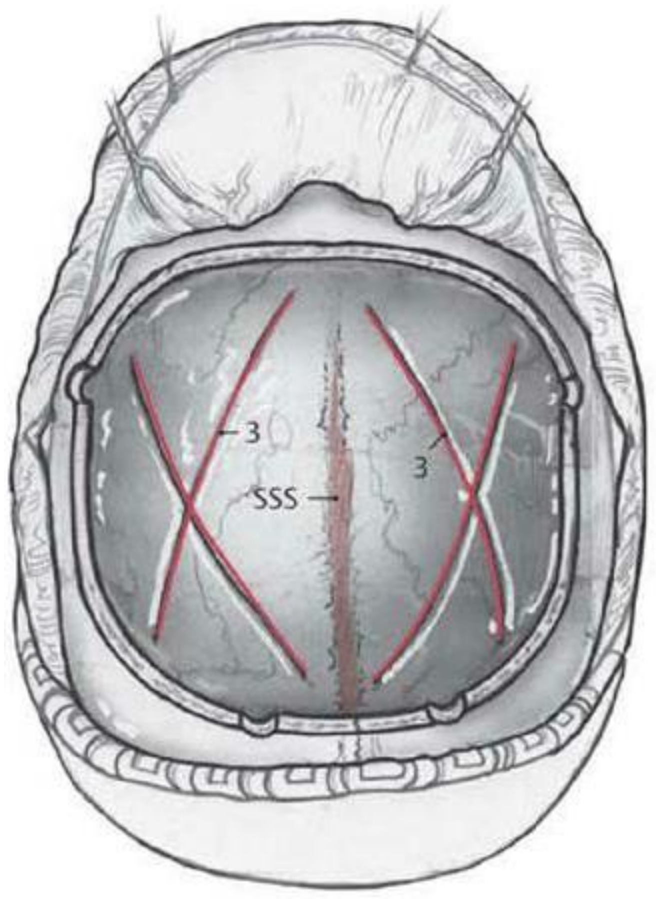
DURA

- some experts insist, that the division of the anterior superior sagittal sinus and falx is crucial.
- dural cuts:
  a) standard Kjellberg open fish-mouth cuts made along floor of anterior fossa with release of inferior aspect of interhemispheric falx and then curved up (along posterior bone edge) towards SSS.
  b) mitral valve–type dural incisions - parallel to superior sagittal and parallel to posterior bone edge (some recommend connecting bilateral incisions along floor of anterior fossa with release of inferior aspect of falx):



Source of picture: Brian T. Ragel et al. “Wartime decompressive craniectomy: technique and lessons learned”. Neurosurg Focus / Volume 28 / May 2010 >>

c) bifrontal cruciate incisions:



Source of picture: R. Nader “Neurosurgery Tricks of the Trade – Cranial“ (2014); Publisher: Thieme; ISBN-10: 1604063343, ISBN-13: 978-1604063349 >>

POSTERIOR FOSSA DECOMPRESSIVE CRANIECTOMY

- incision: midline skin incision from above inion to ≈ C2 spinous process.
- bone opening: laterally to sigmoid sinuses, superiorly to transverse sinus.
- C1 laminectomy is typically performed as well.
- dural opening: “Y” shaped incision.

PENETRATING BRAIN INJURIES

- systemic antibiotics + tetanus prophylaxis + seizure prophylaxis!!!
- rapid local debridement - clean from bone fragments, necrotic brain, debris, foreign objects.
  – debridement of devitalized brain is gentle (use combination of suction and irrigation).
- exploration for hemorrhage and necrotic tissue (recent data question practice of pursuing bone chips spread deeply into brain).

  N.B. retained fragments have not been associated strongly with infection, most authors *remove fragments only if they are accessible*! (secondary removal is performed only for unusually large retained fragments - complication rate for repeat exploration may exceed rate of complications of retained fragments)
- watertight closure - after hemostasis, all layers of wound are closed tightly (drains are added when hemostasis is not absolute).

Options (class III) for penetrating TBI:

a) **small entrance bullet wounds** → local wound care and closure.

b) **extensive wounds with nonviable (devitalized) scalp, bone, dura** → more extensive debridement before primary closure or grafting to secure a watertight wound.

c) **significant fragmentation of the skull** → debridement with either craniectomy or craniotomy.

d) **significant* mass effect** → debridement of necrotic brain tissue and safely accessible bone fragments, evacuation of intracranial hematomas.

N.B. **in the ABSENCE OF SIGNIFICANT MASS EFFECT**, surgical debridement of the missile track in the brain is not recommended (class III evidence - outcomes are not measurably worse in patients who do not have aggressive debridement); routine surgical removal of fragments lodged distant from the entry site and reoperation solely to remove retained bone or missile fragments are not recommended.**

e) **open air sinus injury** → watertight dura closure

— any repairs requiring duraplasty can be at the discretion of the surgeon as to material used for closure.

— the question of timing of surgery has not been adequately studied to make evidence-based recommendations but general practice it to operate promptly.

N.B. **CSF leak** is the variable most highly correlated with intracranial infection after PBI! (CSF leak increases risk of infection 10-20 fold).
Highest risk of CSF leak - transventricular trajectories (suggesting a course of CSF drainage in such cases), trajectories through air sinuses (suggesting sinus cranialization in such cases).

*“significant” - displacement of the midline > 5 mm, compression of basilar cisterns from edema or hematoma, deteriorating clinical condition.

**in the United States military, thorough debridement of intracranial bone and metal fragments was official military policy through the Vietnam War.

- the literature from recent military conflicts when broad-spectrum antibiotics were widely used suggests that retained bone fragments no longer appear to be independently associated with increased risk of infection - the argument for aggressive debridement no longer appears to be supported!
  Aarabi B et al. Central nervous system infections after military missile head wounds. Neurosurgery. 1998;42:500–509
- studies cannot confirm that fragment removal has obvious efficacy in preventing epilepsy; interestingly, metal fragments (vs. bone fragments) correlated with the development of epilepsy.
  Brandvold B et al. Penetrating craniocerebral injuries in the Israeli involvement in the Lebanese conflict, 1982-85. J Neurosurg. 1990;72:15–21
  Salazar AM et al. Epilepsy after penetrating head injury, I: clinical correlates—a report of the Vietnam Head Injury Study. Neurology. 1985;35:1406–1414.

> Vigorous debridement and pursuit of intracranial fragments are not necessary to prevent infection, has no obvious efficacy in preventing epilepsy, and is associated with morbidity and mortality without resulting in any clear advantage in mortality!

GUNSHOT WOUNDS

Military vs. civilian penetrating brain injury (PBI):

- Most PBIs incurred on the battlefield are from shrapnel, not bullets, like most civilian PBIs.
- Most PBIs on the battlefield by a high-velocity bullet never make it to medical care - extremely damaging cerebral injuries that these bullets inflict (field triage under battle conditions requires the corpsman to identify the injured who have a low probability of surviving and to not prioritize them for rapid transport).
- Wounds are much more likely to be contaminated on the battlefield.
- Suicides are much less frequent.
- Battlefield care conditions are inferior and evacuation times are much longer (mean evacuation time in the military setting is 2.3 hours vs. 30-45 minutes in civilian injures).
- In the military situation, 50% of CSF leaks may occur at sites remote from entry or exit wounds, caused by fractures and dural rents resulting from the concussive effect of the projectile.
- Injuries in the military setting occur primarily in young men in otherwise excellent physical condition; vs. civilian setting - broader age range.
- Literature reports a mortality under wartime conditions in the 20% range (8-43%; military gunshot wounds have 2.5-4 times higher mortality than from shrapnel wounds) vs. 94% for civilian cases.

- first surgery often represents "damage control" surgery - quick removal of mass lesions (hematomas), parenchymal debridement without overly aggressive pursuit of deep and small bone or foreign fragments hemostasis, and quick decision with respect to decompressive craniectomy; deep imploded bone fragments and foreign bodies are not chased.
  - scalp incision (during primary craniotomy) is better to locate away from penetration wound so that least possible scarring will overlie site of any future cranioplasty.
  - extremely important – watertight dural closure; use grafts to avoid tension on suture line (esp. if dura was extensively debrided); prefer autologous grafts – fascia lata, pericranium* or temporalis fascia; avoid artificial synthetic or biological dural substitutes.
    *pericranium is positioned so that surface which has been against bone is directed away from brain.

> Current standard is *debridement of first few centimeters of tract* → *watertight dural closure**

*to prevent centripetal infection and CSF fistula

– reports from Middle East conflicts in which patients were treated without any debridement but with only simple skin closure of bullet hole did not indicate worse outcome.

- second or third operations are sometimes necessary for further debridement of necrotic brain tissue; deep imploded bone fragments and foreign bodies would often deliver themselves to surface at this time.
  - ultrasound is helpful in finding retained fragments or hematomas.
    N.B. *bullet is not removed unless it is easily accessible* (because risk of brain injury from bullet retrieval exceeds benefit of its removal).

- cranioplasty is delayed for ≈ 1 year (when patient is medically stable and risk of infection is low).

STAB INJURY

a) jei ligonis pateko jau išėmus žalojančią priemonę, jei sužeidimas įvykęs prieš < 12 valandų, žaizda nėra stipriai užteršta, nėra intrakranijinės hematomos ar impresinio lūžio → pakanka pirminio chirurginio odos žaizdos sutvarkymo.

b) *jei šių reikalavimų neišlaikoma* → kraniektomija, įėjimo angos išvalymas, kietojo smegenų dangalo plastika;
   – į galvą įsmigęs svetimkūnis šalinamas tik po CT, operacinėje, atvėrus dura (removal under direct vision) pasiruošus galimoms komplikacijoms (atsinaujinęs ar suintensyvėjęs kraujavimas).
   – pašalinimas atliekamas atkartojant ginklo trajektoriją retrogradiškai.
   – ypač pavojingos *medžio liekanos* (sunkiai identifikuoja rentgenas) → meningitas, smegenų pūlinys.

DURAL VENOUS SINUS INJURIES

Traumatic dural venous sinus injuries carry high mortality (vs. slow sinus occlusions by tumor) – hard to control bleeding, ligation is dangerous.

– may be caused by *depressed skull fractures* overlying any of major intracranial venous sinuses.
– maintain hydration to prevent thrombosis.
– if major venous sinus injury is suspected: approach by exposing intact sinus above and below, open dura on both sides so that sinus can be clamped; ETOMIDATE for neuroprotection.
– anterior 1/3 of superior sagittal sinus ***can be ligated*** without any clinical sequelae; tears of posterior 2/3 of sinus ***need repair**** (primary repair or patching with galea or pericranium; alternatively, piece of muscle or Gelfoam may be sutured over sinus).
*because ligation may cause lethal venous congestion

MALIGNANT CEREBRAL EDEMA

- ominous sign!
- may happen if patient had period of hypotension / hypoxia – maximally dilated and brain vessels with paralyzed autoregulation.
- may happen precipitously intraoperatively (soon after decompression) → external herniation

MANAGEMENT

- make sure no venous outflow obstruction, make sure no tapes around neck.
- elevate HOB.
- avoid high SBP
- increase depth of anesthesia
- hyperventilate
- additional MANNITOL
- place EVD
- suspect **contralateral hematoma** - empiric surgical exploration on other side without interim CT (esp. in setting of contralateral skull fracture)
- avoid venous compression at sharp bone edges by making a gap in the bone above draining veins using a bone rongeur.
- subtotal temporal and/or frontal **lobectomies** may be necessary
- primary scalp closure maybe difficult (if this possibility is suspected in advance, it is wise to obtain hemostasis and be prepared to close before dura is opened); plus:
  - if head damp (Mayfield, Sugita) was used, it should be released to give the scalp laxity to be mobilized toward the incision.
  - dissection of the galea from the neighboring skull may be performed to provide more laxity.
  - make incisions in the galea from the underside of the scalp without cutting the epidermis (relaxing incisions).