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Views and reviews about Surgery for traumatic intracerebral hematomas

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1. INTRODUCTION

Traumatic intracerebral hematomas (ICH) are defined as hematomas 2 cm or greater in size not in contact with the surface of the brain. These are present in 15 percent of autopsy cases of severe head injury (Adams et al. 1977). They account for approximately 20% of all intra cranial hematomas.

Classifications

1. International Classification of Diseases (WHO – ICD-NA code)

List is exhaustive only few given here which relates to the chapter (Table 1).

2. Clinico-Pathological Classification

(1) Acute traumatic ICH is obvious at the time of injury and found at initial scan although they may go on enlarging hours to days. This is most common variety.

(2) Delayed traumatic ICH (DTICH) is not obvious on initial scan but appears after hours to days or even weeks after initial negative scan. Bollinger's spate apoplexy is a type of delayed hematoma which occurs even after weeks or months and patient remains asymptomatic. While common variety of delayed ICH occurs most commonly within 72 hours and patient is rarely asymptomatic (Atkinson, 2003). Spontaneous ICH has different etiology, pathophysiology of development, management and prognosis. It occurs without any obvious trauma and is due to some other pathology mostly in older age than traumatic variety and also have poor prognosis.

3. CT scan classification

CT scan classification of post traumatic ICH (Blumberg and Fukamachi, 2005) is given in table 2.

Table 1

ICD-NA classifications (Source: WHO)

SN	Disease entity	ICD-NA(Neurological application) code
1	Traumatic cerebral oedema-	S 06.1
2	Diffuse brain injury	S 06.2
3	Traumatic Intra cerebral hemorrhage	S06.3*
4	Intra cranial injury with prolonged coma	S 06.7
5	Spontaneous Intra cerebral hemorrhage	I 61

*(if frontal- S 06.2x0,temporal S 06.3x1,Parietal -S 06.3x2, Occipital- S 06.3x3, Deep cerebral hemisphere- S 06.3x4,corpus callosum -S 06.3x5, Brain stem -S 06.3x6 ,Cerebellar -S 06.3x7 and so on)

Table 2

CT scan classification of post traumatic ICH

Type I	Type II	Type III	Type IV
Evident of initial scan	Evident on initial scan enlarges on subsequent scan	Develop although initial scan showed no abnormality	Develops in area of abnormality(salt & pepper or "flecked high density" change

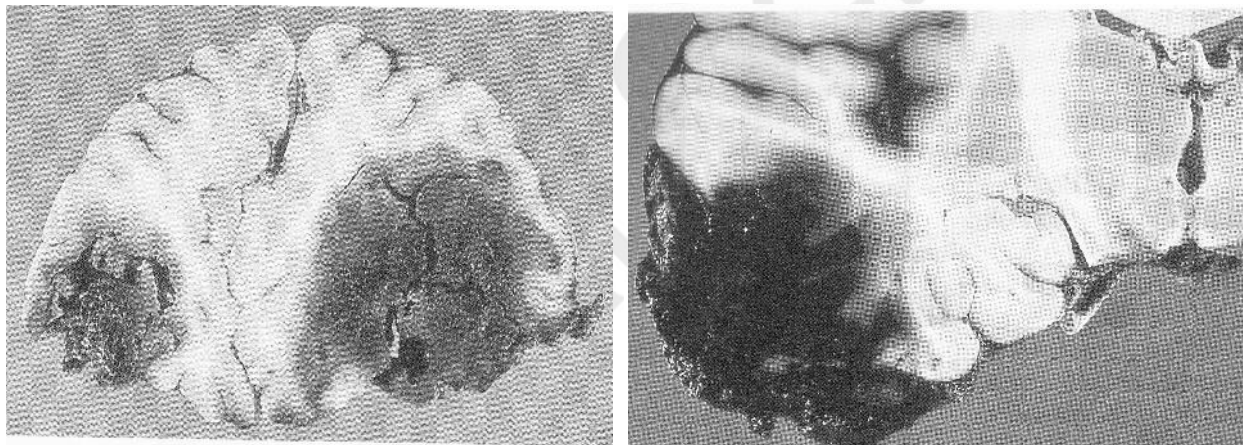


Figure 1 a & b

Burst frontal and left temporal lobe (Blumberg and Fukamachi, 2005)

There are primary brain injury due to trauma and secondary brain injury due to secondary insult following trauma. There are two types of primary brain injury, focal and diffuse. Focal brain injury comprises of epidural hematoma, coup contusion, countercoup contusions, inter mediate coupe contusions, intra cerebral hematoma, tissue tear hemorrhages, and sub dural hematoma. While diffuse brain injury can be classified in to cerebral contusions and diffuse axonal injuries (Gennarelli and Meaney, 1996).

Holbourn, and Ommaya and Gennaralli have documented experimentally and, clinical and autopsy observation have confirmed the predisposition of frontal and temporal poles as the common sites of injury (Holbourn, 1943; Naffziger and Jones, 1982), (Figure 1a & b). It was pointed out that from a pathological stand point it is difficult to draw a clear-cut line between a lesion which could be called cerebral contusion with hemorrhage and a real intracerebral hematoma (Kristiansen and Tandon, 1960). Adams described the entity they call burst lobe and distinguish them from traumatic ICH where ICH is in direct communication with cortical laceration which in itself is in continuity of subdural hematoma (Mc Cormick, 1996), (Figure 1a); most of the time they coexist. Basal ganglion, thalamus and brain stem lesion mat occur in isolation or in combination with lobar lesion (Blumberg and Fukamachi, 2005), (Figure 2).

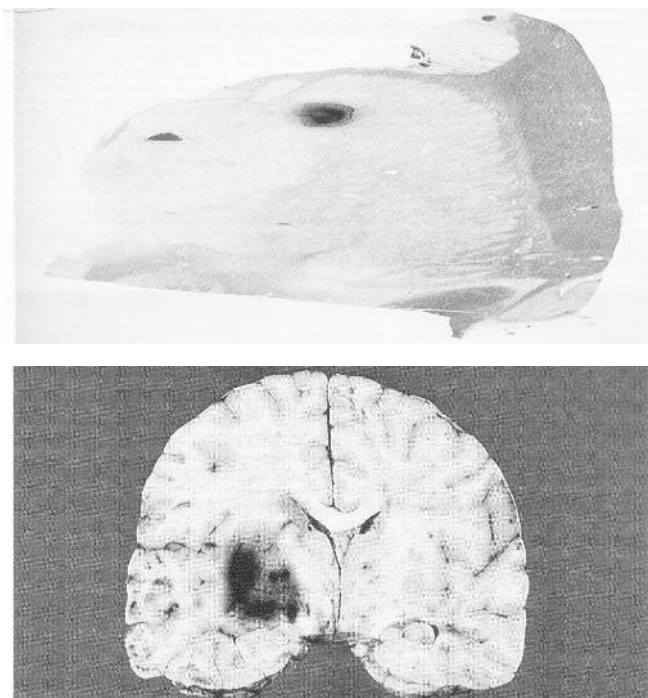


Figure 2

Basal ganglia hematoma (above) & thalamic hematoma (Blumberg and Fukamachi, 2005) below in the figure

2. VIEWS FOR THE MECHANISM OF ICH FORMATION AND DAI DEVELOPMENT (PATHOPHYSIOLOGY)

Larger traumatic ICH is uncommon most often associated with extensive cortical contusions in which larger, deeper vessels have been disrupted. Smaller, single hematoma that is not associated with contusion probably occurs because of stress wave concentration resulting from impact or because of acceleration-induced tissue strain deep within the brain. These may represent form frusta of the tissue tear hemorrhages that accompany diffuse axonal injury. Tissue tear hemorrhages (TTH) are due to intra cerebral damage to blood vessels and axons in association with diffuse axonal injury (DAI). They are due to inertial or head motion effect and not contact phenomenon. They are distinct from ICH and are part of severe form of DAI that result in immediate prolonged coma. TTH are typically numerous, small (usually varying from petechial to 1 cm in diameter, and located parasagittally in the central (not polar) portion of the brain. Delayed variety may be because of vessel fragility in elderly or change in intramural intra cranial vascular pressure gradient, decompression following evacuation of subdural or extra dural hematoma, and with release of temponade effect or hypotension due to shock followed by correction of shock leading to hematoma disseminated intra vascular coagulation and coagulopathy associated with alcohol intake.

3. SIGN AND SYMPTOMS

An intracerebral hematoma may produce specific localizing signs associated with the damaged eloquent cortex, in addition to

deterioration in sensorium due to the mass effect resulting in raised intracranial pressure and herniation and seizure. Deterioration in sensorium occurs in proportion of the degree of shift of the midline structures to the contra lateral side, but cerebral edema or diffuse axonal injury may present in a similar fashion and may associated in case of severe head injury. In mild case without significant mass lesion and mid line shift (less than 5mm mid line shift) conservative therapy may be tried. Progressive neurological deficit or deterioration in consciousness may prompt measurement and finally surgical decompression. Delayed development of a mass lesion may necessitate surgery during hospitalization, even if surgical decompression was not required initially.

Delayed intra cerebral hematoma most commonly occur in first 24 to 48 hours though in some cases many days or even few weeks may pass the hall mark of delayed intra cranial hematoma is progressive deterioration in clinical status such as seizure, progressive focal neurological deficit, or decreasing level of consciousness. But sometimes simply appears on subsequent CT scanning in unchanged patient. It mostly occurs in elderly or alcohol users and have elevated ICP relieved by medical or surgical means (Atkinson, 2003).

Decompression of a mass lesion on one side may lead to expansion of a contra lateral lesion intraoperatively or postoperatively. Cerebral edema develops around an area of contusion or a hematoma within 24 to 48 hours following head injury and maximizes in 2 to 5 days. Significant edema around the contusion- hematoma may increase the effective mass by 25 % or more.

4. DIAGNOSIS

CT scan head is the modality of choice and can be performed within 10 minutes only and clearly defines the location, extent and type of intracranial lesion. Bone window shows skull fractures also thus obviating skull x-rays. Fracture is noted in 40-80% of ICH. If CT is not available emergency cerebral angiography can often demonstrate traumatic hematoma and midline shift. If both are not available, in a rapidly deteriorating patient can undergo twist drill ventriculostomy and CSF drainage followed by air ventriculography showing ventricular shift, indicative of mass lesion can be demonstrated. An acute hematoma is less clearly visualized on MRI than CT scan. It also takes longer time during procedure, as well as being expensive. MRI requires ventilator with nonferrous compound, if patient is intubated and requires ventilator. Exploratory burr holes may be required when patient is rapidly deteriorating and investigating facility is either not available or cannot be utilized. Exploratory burr hole can also be made just before formal craniotomy if patient is rapidly deteriorating (Dusick, 2006). Other options like Near Infra red spectroscopy

Table 3

Treatment Options for ICH and Indications for surgery (Hsieh et al. 2010)

Component	ICH score points
GCS score	
3-4	2
5-12	1
13-15	0
ICH Volume ,ml	1
=or > 30	0
< 30	
IVH	
Yes	1
no	0
Infratentorial origin of ICH	
Yes	1
no	0
Age ,years	
= or > 80,	1
80	0

Table 4

Treatment Options for ICH and Indications for surgery (Dusick et al. 2006)

Treatment option for ICH	
Progressive neurological deterioration referable to lesion, refractory ICP, or mass lesion in CT	Surgical evacuation
GCS score 6-8 and frontal or temporal contusion >20 ml and mid line shift =or > 5mm and /or cisternal compression	Surgical evacuation
➤ 50 ml volume	Surgical evacuation
No neurological compromise, controlled ICP ,no significant sign of mass effect on CT	Neurological observation and serial CT scan

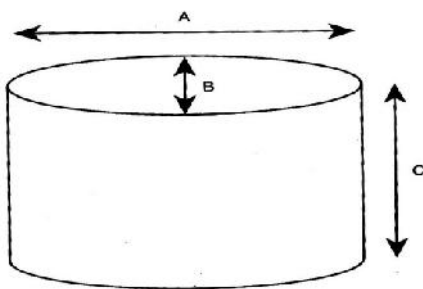


Figure 3

Calculation of volume (Dusick, 2006)

(NIRS or cerebral oxymetry), (Upadhyay, 2002), SPECT (single photon emission computed tomography), PET (positron emission tomography) TCD (trans cranial Doppler), etc can be adjuvant to CT scan.

Determination of ICH volume

ABC method (Dusick, 2006) of determining ICH volume (Figure 3) is volume in cc = $A \times B \times C / 2$. Where scan area with largest ICH was selected and the largest length and breath is measured in cm in the selected scan. C is the total depth or height (thickness) of the hematoma in going through all slices of the Scan. Morbidity and mortality is closely associated with key variable including patient age, volume of hematoma, location of hematoma, neurological status at presentation (GCS). supra tentorial hematoma with volume more than 60 ml have a 71% to 93 % mortality rate while cerebellar hemorrhage with 30 to 60

ml have a mortality of 75 % mortality rate. Pontine hematoma greater than 5 ml is almost 100% lethal (Hsieh et al. 2010).

ICH Scoring

An ICH scoring has been devised that takes into consideration GCS (Glasgow coma scale), ICH volume, IVH (intra ventricular hemorrhage) location of hematoma, and age. Higher score corresponds with 30 days higher mortality rate (Table 3).

5. HISTORY

The first written record of man's surgical practices comes from Egypt as seen in the Edwin Smith Papyrus. In this manuscript, 27 cases of head trauma have been described, out of which 14 involved the soft tissue only and in 13 cases, a fracture was present. Rhazes (*Rhazes. 1511 Continens*) is said to be the first to state that intracranial hemorrhage is more important than fracture of the skull. Intracerebral hemorrhage following trauma was apparently known to Roger of Salerno (*Roger, 1938*). Delayed development of hematoma first described in four case over a century ago (*Atkinson, 2003*). Surgical intervention, depending upon the clinical recognition of the condition, was not advocated until early in the twentieth century. In 1903, Cushing stated that he could find no reference to surgical therapy; he reported a case in which he found an intracerebral clot while exploring for a subdural hematoma (*Cushing, 1903*). The patient did not recover. Naffziger and Jones were more successful with their cases treated by craniotomy and evacuation of the clot. In the first half of this century, the recommendations for operative management have been far more varied (*Naffziger and Jones, 1982*). Though isolated cases of temporal lobe hematomas were described under a variety of names' burst temporal lobe, pulped lobe, his first large series of such lesions was described by various authors who elaborated the pathogenesis, clinical picture and management (*Tandon, 1978*). The need for adequate exposure and use of an osteoplastic flap was also stressed. A large fronto-temporo-parietal craniotomy initially described by Murray Falconer for epilepsy surgery was popularized for head injury and later on, its revised or extended version became popular as the 'trauma flap'.

6. GOALS OF SURGERY

The guiding principles of surgery for head injury are preservation of neurological function, prevention of further injury to the neuraxis and prophylaxis against the secondary complications of head injury. Specific goals include the following:

1. Prevention of increased intracranial pressure: Mass lesions that increase in size in the cranium (Monro-Kellie-Burrows doctrine) displace CSF and venous blood. Failure after this compensation will lead to global compression of the brain with progressive increased intracranial pressure (*Rengachary, 2005*), impairment of cerebral perfusion pressure, cerebral blood flow and herniations. In central downward herniations leads to Cushing's triad (hypertension, bradycardia and respiratory irregularity) and may lead to basilar artery stretching and petechial hemorrhages in the brain stem area (i.e. Duret hemorrhage). Occasionally opposite cerebral peduncle is compressed against tentorium causing ipsilateral hemi paresis, ipsilateral to papillary dilatation contrary to normal phenomenon in uncal herniations called kernohan phenomenon (*Dunn and Ellegala, 2010*).
2. Prevention of focal brain parenchymal compression and dysfunction: Hemorrhage involving the eloquent cortex should be decompressed if the dysfunction is considered to be the result of the focal pressure effect rather than neuronal disruption. The treatment of multiple or deep, relatively inaccessible hemorrhage, is more problematic; large hemorrhages of the basal ganglia or the internal capsule are usually associated with fixed deficits and the indications for surgery depend on midline brain shifts or increasing intracranial pressure effect rather than neuronal disruption.
3. Prevention of anatomical compression of the brainstem and diencephalon: Mass lesions producing midline shift (more than 5mm) and brainstem compression need evacuation.
4. Prevention of compression of brain vasculature: Anatomical distortion of the brainstem may result in avulsion injuries to the perforating vessels. Temporal lobe hematomas, by uncal herniation may compress the posterior cerebral artery as it traverses the tentorial incisura causing occipital ischemia. Posterior fossa lesions may compress the superior cerebellar arteries in a similar fashion. Lateral mass lesions may cause subfalcine herniation of the cingulate gyrus and its incarceration with compromise of the anterior cerebral artery. Prolonged compression of these arteries will result in infarction of the brain in their respective territories. Tonsillar herniation may cause cheyne stokes or neurogenic hyperventilation and opisthotonus position (*Rengachary, 2005*).

7. TREATMENT OPTIONS FOR ICH AND INDICATIONS FOR SURGERY

General principles of treatment options and indications are summarized in table 4 (*Dusick et al. 2006*). Although controversy remains, the above guide lines published in 2006 offer useful recommendations. It is mostly because

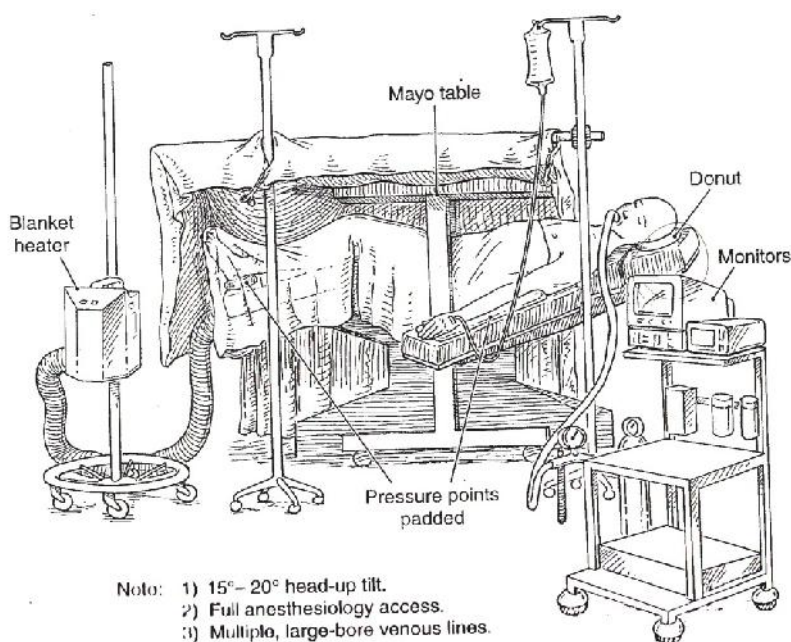


Figure 4
 Patient position and equipments for craniotomy or craniectomy (Prabhu et al. 2004)

intravenously may also given in addition to mannitol. If there is a history of seizures, intravenous diphenylhydantoin in a loading dose of 20mg/kg body weight is administered. Even if there is no history of seizures, the drug is given prophylactically in a dose of 10 mg/kg body weight. Check list for craniotomy—

- (1a) A minimum of two units of blood should be available. When the patient's GCS is less than 9 and there are signs of herniation or evidence of aspiration into the lungs, intubation and ventilation are started in the emergency room before shifting the patient.
- (1b) coagulation studies-(i) prothrombin studies, (ii) partial thromboplastin time, (iii) platelets count.
- (1c) blood gas analysis.
- (1d) routine full blood count and electrolytes.
- (2) Radiograph of chest, and cervical spine if suspected.
- (3) Consent for surgery.
- (4) Foley catheter in bladder.
- (5) Two large bore peripheral intra venous lines or one peripheral and one central line to maintain central venous pressure >5cm H₂O.
- (6) Arterial catheter.
- (7) Protection of both eye from chemical fluids and pressure.
- (8) Adequately secured cuffed endotracheal tube.
- (9) Both lower extremity placed in sequential compression device to minimize the risk of deep vein thrombosis.
- (10) Adequate antibiotics and anti convulsants before surgery (Prabhu et al. 2004).

Smooth intubation and induction of anesthesia in these patients with raised intracranial pressure is critical. Before intubation, the patient is paralyzed and given a bolus of thiopentone. Lidocaine is instilled into the trachea. Volatile anesthetic agents like halothane should not be used because of their cerebra vasodilator effect, which will result in increased intracranial blood volume and a rise in intracranial pressure. Various combinations of barbiturates, narcotics and tranquilizers should be used because of their minimal effect on intracranial pressure. Blood gases are monitored during the surgery and the arterial oxygen tension maintained above 80 mm hg and the paco₂ between 28 and 32mm hg.

9. OPERATIVE TECHNIQUE

Meticulous surgical technique and judgment are necessary for the successful management of traumatic intracerebral hematomas. Gentle handling of the brain is imperative; firm brain retraction should not be done, as acutely injured

diversity of lesions coexist rather than simply one type of lesion in case of head injury (Upadhyay, 2002; Upadhyay, 2007). Although conservative management of hematoma and contusion are also indicated and used successfully in selected case (Pankaj and Tiwary, 2001). Management must be balanced and closely observed if so chosen. Thus an intracerebral hematoma failure to improve is an important indication for surgery as a matter of fact waiting for deterioration to set in can often lead to irreversible damage.

8. PREOPERATIVE PREPARATION AND ANESTHESIA

In general cranial surgery should not be performed until a stable blood pressure and adequate lung function have been achieved in patient who is comatose and deterioration in level on consciousness or development of focal signs (dilated pupils), removal of hematoma is a matter of great urgency. Intubation with hyperventilation and shifting the patient to operation theatre is taken up.

Intravenous mannitol in the dose of 1g/kg body weight is given rapidly; 300 ml of 20% mannitol will suffice in an adult. Furosemide (lasix) 40 mg

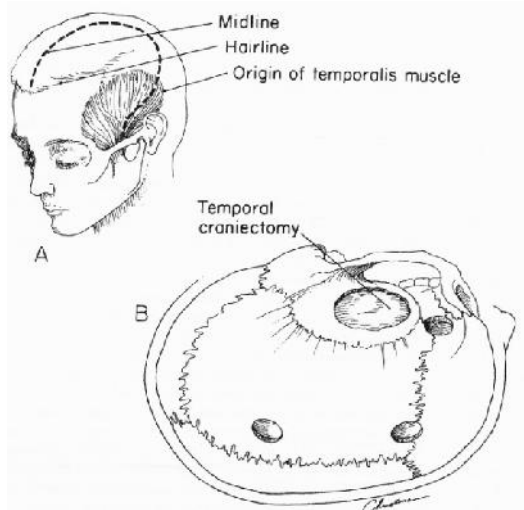


Figure 5
Scalp incision and burr hole placement for a standard trauma craniotomy. Note the temporal craniotomy for initial decompression.

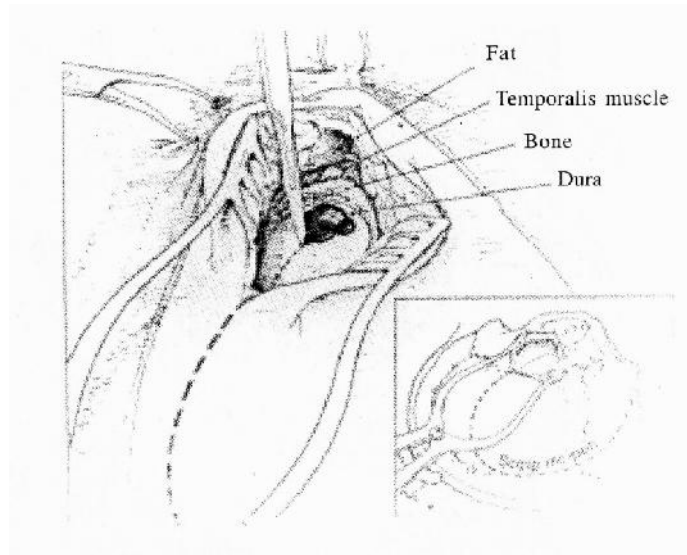


Figure 6
If the patient has been deteriorating prior to operation or if the intracranial pressure is known to be very high and there is a major shift, an initial quick subtemporal decompression should be done. One should then proceed with the formal craniotomy

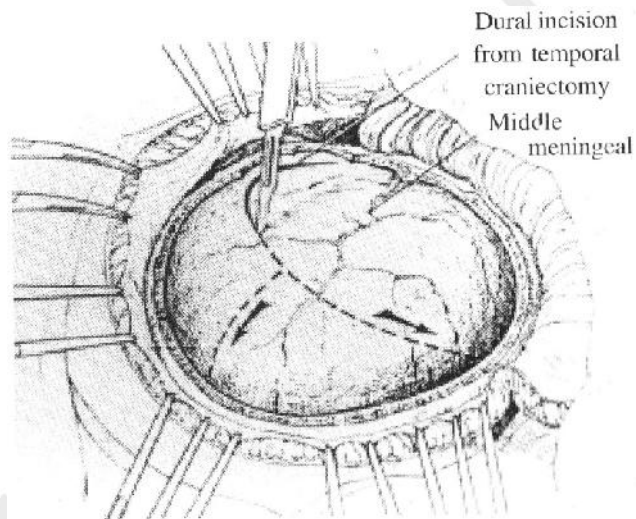


Figure 7
The dural opening shown here provides safe access to anterior, midline and basal regions

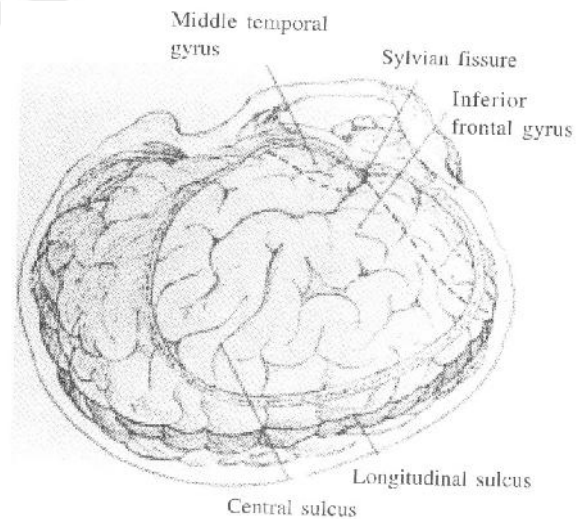


Figure 8
The extent of exposure is demonstrated here. The common sites of contusion hematoma are accessible

brain tissue is quite vulnerable to this type of insult. Open craniotomy is better than endoscopic or stereotactic evacuation as it also provides additional space for accommodating brain swelling.

The most frequent sites of traumatic intracerebral hematoma and contusions are the frontal and the temporal poles, and the area along the vertex. However, the majority of traumatic injuries are best dealt with through a generous fronto-temporo-parietal craniotomy that provides access to these areas. Sometimes, there can be a

relatively small localized polar contusion hematoma which can be evacuated through a relatively small flap craniotomy or medium sized trephine centered over the area of interest.

10. TECHNIQUE OF BASIC FRONTO-TEMPORAL CRANIOTOMY FOR TRAUMATIC LESIONS

The patient should be positioned supine with the head turned to the opposite side, supported on a doughnut or in three pin fixation and elevated above the level of the heart. A small sand bag or a rolled towel should be placed under the ipsilateral shoulder to prevent positional obstruction of cranial venous drainage (Figure 4). The skin incision is outlined as shown in figure 5. It is begun 1 cm in front of the tragus of the ear, starting just above the zygomatic arch then carried superiorly and posteriorly over the ear, posteriorly around the parietal bone to the midline, where it is brought anteriorly up to the hairline. In some patients, the hairline would have receded posteriorly, and then there is no choice except to extend the incision anterior or below the hairline over the forehead. It can later be closed by using plastic surgical techniques and the minimal visual scar is a small sacrifice for the added exposure of the frontal lobe gained by this flap. If the patient has been deteriorating rapidly prior to operation, the inferior end of the incision, i.e. just anterior to and above the ear should be opened first, down through the temporalis muscle to the bone (Figure 6) a burr hole and a limited craniectomy should be performed quickly and the dura opened in a cruciate fashion. Any subdural blood along with the contused/softened temporal lobe and even a part of the intraparenchymal hematoma will squeeze out spontaneously, thereby affording immediate relief of the elevated intracranial pressure. Then, the remaining scalp incision is completed and either a free bone flap or osteoplastic flap based on the temporalis muscle can be raised.

The medial portion of the craniotomy should be approximately 2 to 3 cm away from the midline. The bone flap should be brought low across the frontal bone and resection of the lateral sphenoid wing will further enlarge the exposure, if required.

The temporal dural opening, if already made, can be enlarged, otherwise the dural opening should begin over the temporal region, since, if the brain herniates through the dural opening here, relatively silent cortex is affected (Figure 7). The remainder of the dural opening can be completed with little further herniation of tissue. If intact cortex begins to herniate through the dural incision, further maneuvers to reduce brain swelling should be instituted. Immediately viz., additional mannitol, increased hyperventilation and perhaps even transient reduction of arterial blood pressure to reduce and relieve cerebrovascular engorgement. The dural opening should be curved gently as it is carried out anteriorly up to the anterior medial border of the bone flap. An incision from the center of this dural flap, directed posteromedially will complete the opening. This dural opening provides access to the frontal and temporal lobes, and the anterior and middle fossa (Figure 8). If there is raised pressure or brain herniated another method adopted is to make multiple slit opening in the dura and initial evacuation is done through it before making full dural flap (Figure 9), (Kjellberg and Prieto, 1971). Intraoperative ultrasound is a cheap portable option can be of help in locating hematoma intraoperatively and help in complete evacuation without adding to further injury and decrease the need of retraction over brain (Polin et al. 1997). Intra operative MRI systems can also help in location and asses' extent of evacuation but it costlier and less frequently available as other navigational tools.

The vast majority of unilateral contusion hematomas can now be dealt with. In the less common situation in which parietal or occipital contusion hematomas are present, the approach would have to be modified. A parietal lesion may become accessible by enlargement of the basic craniotomy flap or a reverse question mark scalp incision. Once the dura is opened, the exposed brain should be inspected for any surface contusion. If the contusion or laceration is either in the either in the polar region or the basal surface, it will not be seen on the surface (Figure 10). The preoperative CT scan would have already established its site and extent. The contusion may be restricted to either the temporal or the frontal lobe, or many are at multiple sites. The aim of surgery is not to 'chase' all contusions but only to attend to the one which is acting as a mass lesion due to an associated intraparenchymatous hematoma and/ or edema. This would already have established on the basis of the preoperative scan.

The brain tissue in a confluent contusion is irreversibly damaged and serves not only as a primary mass lesion but may go on to cause further major brain swelling in and around itself. Therefore, obviously necrotic contusions should be removed when exposed at operation. As a general rule, surface contusion larger than 1 to 2 cm in diameter should be removed. Often, a surface contusion 2 cm in diameter will extend several centimeters or more deep into the hemisphere and the cortical edge of the contusion is only the 'tip of the iceberg'. The contused brain is purple blue, soft, friable, necrotic and should be sucked gently by suction tube or better by using Cavitron ultrasonic suction apparatus (CUSA) until a circumferential margin of healthy brain tissue is reached. One should not hesitate to remove all the contused tissue even at multiple sites over the anterior temporal and frontal lobes. Contusions over the temporal lobe or in the region of the central sulcus should be evaluated carefully and if there are clear large areas of necrotic contusion they should also be removed following the same principles. Removing contusions in these areas

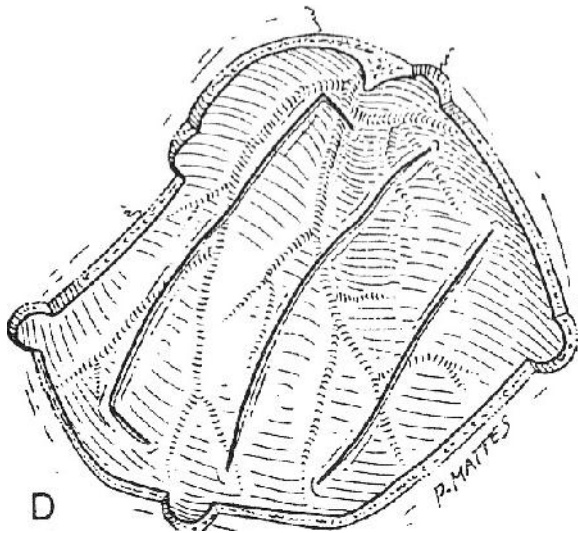


Figure 9

If ICP is raised pressure or brain herniates another method adopted is to make multiple slit opening in the dura (Kjellberg and Prieto, 1971)

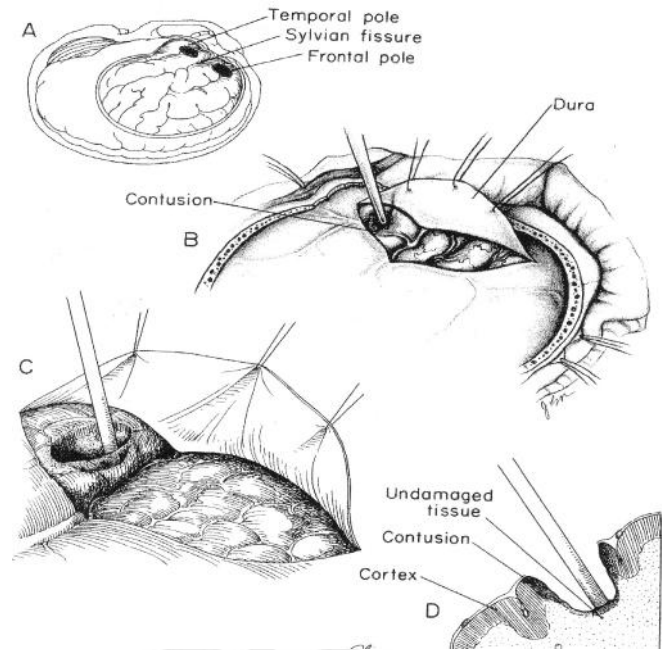


Figure 10 a-d

(a) The usual location of contusion hematomas. (b) The dural opening for access to contusion hematomas. (c, d) resection of contusion limited to damaged brain

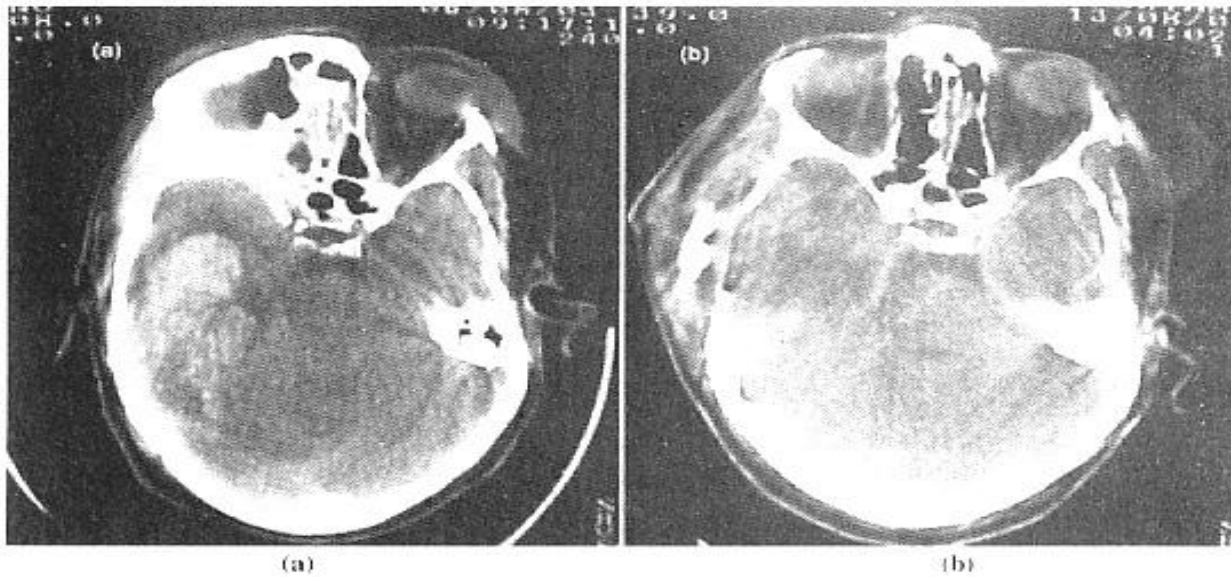


Figure 11

CT scans of a patient taken preoperatively (a) and following evacuation of a large hemorrhagic contusion in the right temporal lobe. (b). Note that the ambient cistern is free in the postoperative picture

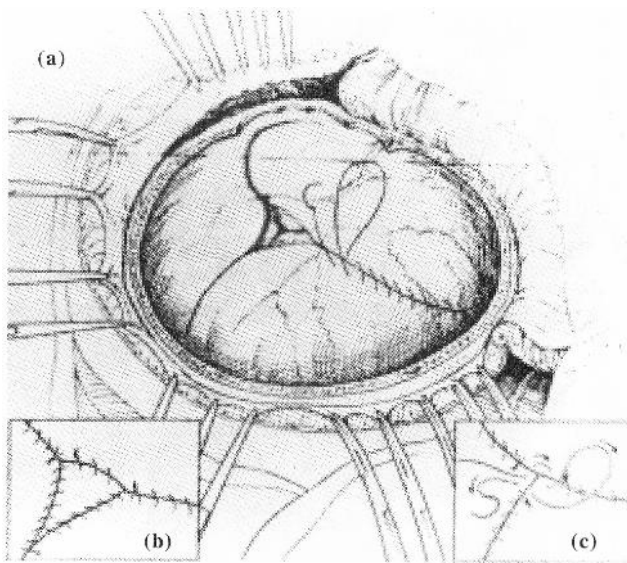


Figure 12 a-c

(a) dural closure is done with a continuous stitch; (b) the junction of the 'T' may be closed either with a dural patch or (c) primarily

will not increase the neurological deficit if one works entirely within the contusion. The secondary brain edema that occurs around the contusion can be reduced by operative removal and the ultimate neurological function may be good.

Intracerebral hematomas tend to present deeper in the brain tissue than contusions. They may also appear directly on the cortical surface and, often, the pathological characteristics are actually those of a combined hematoma and contusion. Hematomas presenting on or near the surface and greater than 1 to 2 cm in size should be evacuated. A deep intracerebral hematoma should be evacuated if it is causing a major mass effect with shift, or if it is associated with neurological deficit. Gentle handling of the injured brain is critical during the procedure, since the brain cannot tolerate further mechanical trauma from tough instrumentation and forceful retraction. Meticulous haemostasis is vital in avoiding a recurrent hematoma. The use of the operating microscope throughout the procedure is most helpful in (1) minimizing retraction of brain tissue, (2) minimizing the extent of necessary cortical incisions, (3) helping to recognize the necrotic areas of the brain and (4) helping in securing meticulous haemostasis. If the preoperative scan shows the lesion responsible for the mass effect to be restricted either to the anterior temporal or to the frontal lobe, it is best to plan a formal frontal lobectomy, including the lesion, rather than carry out a limited

removal restricted to the contused brain. As already mentioned, the contused brain is not always seen on the surface and may be exposed as the lobectomy proceeds and the intraparenchymatous hematoma is evacuated. The end point of evacuation of a hematoma is to have a relaxed brain with CSF flowing from the basal cisterns and return of brain pulsation if initially not present. It may be useful to visualize the tentorial edge in all cases and sectioning it to avoid uncal herniation and compression of brain stem. Open the arachnoids of the ambient cistern, especially in temporal lobe contusion (Figure 11).

Sudden massive intraoperatively brain swelling can develop. It may occur immediately after dural opening or, sometimes, even after a clot or contusion has been evacuated and the brain is initially relaxed. A loss of cerebral autoregulation with acute cerebral hyperemia is thought to account for this phenomenon. Intraoperatively brain swelling must be dealt with promptly and aggressively. The first step to counteract this is to request the anesthetist to ascertain that there is no airway obstruction, undetected hypercarbia or compression of the neck veins. After excluding these, a brief period of arterial hypotension to a systolic pressure of 60-90 torr in combination with hyperventilation and additional mannitol often reverses this swelling, although these measures may need to be repeated several times. After two to four minutes the blood pressure should be allowed to seek its own level naturally. Prolonged induced hypotension must be avoided as severe brain ischemia will ensue. Sodium Nitropruside should not be used to lower the blood pressure, because this drug not only causes cerebral vasodilatation but can directly disturb the integrity of cerebral autoregulation. Other agents such as Trimethaphan (Arfonad) should be used for rapid induction of transient hypotension. Brain swelling that does not subside by these means may respond to 500 mg boluses of thiopental up to a total dose of 1 to 2 g. Only if this maneuver also fails to relocate the brain within the skull, should internal decompression by temporal or frontal lobectomy be used.

In cases with large contusions and intracerebral hematomas involving most of the anterior temporal lobe it is better to plan an anterior temporal lobectomy rather than limiting the surgery to the removal of the contused brain alone. As long as the lobectomy is in front of the vein of Labbe, this procedure is unlikely to add to the deficit; and, in addition, it provides an opportunity to deliver any herniated medial temporal lobe, attend to the basal contusion and to section the tentorium.

Obtaining meticulous hemostasis with the use of bipolar cautery and haemostatic agents like avitene, Surgicel, Fibrillar, thrombin soaked gel foam or surgofoam (Dusick et al. 2006).

After the procedure is completed, dural closure is recommended by most surgeons and, if necessary, a pericranial or fascia lata graft should be used (Figure 12). Now a days artificial material (dural patch /membrane) or duragen type are also available which can be attached to make cavity more spacious and prevent CSF leak. Use of adhesive material in form of fibrin glue may also be used to seal the small dural opening so left during closure of help attach the dural membrane/ duragen. Dural tacking suture must be used to prevent hematoma formation. Some surgeons do not close the dura so as to allow some more space, in case there is edema in the postoperative phase. The bone flap

Table 5

Out come in ICH and contusion (Dusick et al. 2006)

ICH or contusion	Overall outcome	Factors associated with poor out come
	11%-30%	Presence of associated lesions(e.g. fracture, EDH SDH etc) Presence of edema, raised ICT, hydrocephalus, compression of cisterns /ventricles Location of lesions Larger volumes and increasing no of lesions increasing from clinical deterioration to surgery

should always be replaced and secured with nonmetallic sutures, so that it does not interfere with postoperative CT or MRI scanning. Sometimes, the brain swelling can be massive and cannot be controlled and, in these patients, the dura is left open and the bone flap removed. This procedure should not be used as a routine. When necessary, the bone flap can be placed in the subcutaneous fatty layer of the anterior abdominal wall or in deep freezer (at -70°) for later replacement usually after 6 week to 6 month interval to avoid cumbersome and artificial cranioplasty. For posterior fossa hematomas a wide sub occipital craniectomy is used and the dural opening is begun on the side of the lesion.

Note: a burr hole does not provide enough exposure to make it worthwhile even as a stop gap measure in treating a traumatic intracranial mass lesion. It is of value only when done as a diagnostic procedure by a surgeon prepared to perform an immediate formal craniotomy. With the availability of CT and MRI scanners, the use of diagnostic burr holes is hardly ever required.

Prognosis and outcome: of ICH and contusion are summarized in table 5

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