

Decompressive Craniectomy (DC) - Views and reviews

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Kjellberg's and Prieto's wrote their most influential publication in 1971, which not only resurrected decompressive craniectomy (DC) but also challenged the field to validate or refute its utility in management of cerebral edema (Whitefield et al. 2001). There is near total absence of class I data on DC in TBI. And as we reach a discomfoting therapeutic plateau in the post operative medical management of sequel of severe TBI the use of DC in severe TBI, technical consideration in its implementation, the extant evidence regarding its efficacy has renewed interest. Two randomized trial, controlled trial RESCUE ICP and DECRA is going on to evaluate the results. Other indications of DC are malignant cerebral infarction (Dunn and Ellegala, 2010).

Rescue ICP trial multicentre trial by university of Cambridge and European brain injury consortium, on randomized to optimal medical management or DC on 50 patients (total of 600 patients at least including all centers). DECRA is also multicentre randomized trial that involves centers in New Zealand and Australia on the same principle and also including control of ICP (Dunn and Ellegala, 2010).

Primary DC is performed at the time of evacuation of an intra or extra axial hematoma to facilitate post operative management of intracranial pressure. Delayed or secondary DC is specifically performed to control raised ICP in hours or days after primary injury and its management if all measures to control ICP has failed (Dunn and Ellegala, 2010).

The defined boundaries of focal injury facilitate surgical resection. Management of diffuse injuries on other hand relies on hemodynamic, metabolic, and osmotic parameters, as well as ventriculostomy drainage of CSF. According to current American academy of neurosurgical society (AANS) and European brain stem consortium recommendations barbiturate-induced coma, hypothermia, and decompressive craniectomy represents second tier treatment.

In contrast to malignant cerebral edema, the evidence supporting emergent decompressive craniectomy in trauma remains controversial. It has been associated with increase cerebral edema, hemorrhagic infarct and cortical necrosis at craniectomy site. On other hand, decreased intra cranial pressure, improved oxygen tension and improved cerebral perfusion has also been reported. The views of more than 30 articles published on the subject of decompressive craniectomy in context of severe head trauma failed to demonstrate clear benefit. Proponents of DC

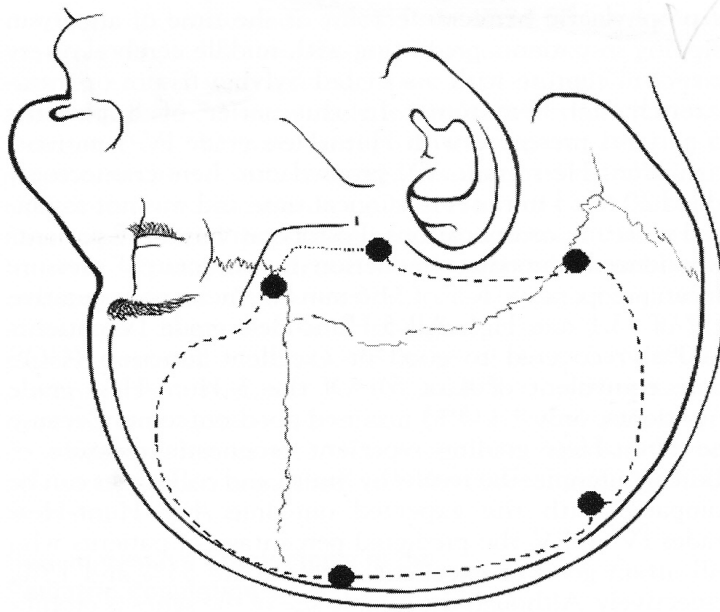


Figure 1

Standard Fronto- Temporal- Parietal (unilateral hemicraniectomy); (Chen et al. 2006)

site three studies (Polin and colleagues (Polin et al. 1997); Whitefield and colleagues (Whitefield et al. 2001); Guerra and colleagues (Guerra et al. 1999)) showing improved results. A prospective study in pediatric age group (Taylor et al. 2001) covering children over the age of 12 months showed favorable outcome but that was not statistically significant. The indication of DC in traumatic brain injury remains controversial. Most study suggests reduction in ICP but whether that alters the clinical course is controversial. The picture emerges that young patient (below 18 years) with GCS more than 4 may benefit from DC when intervention is performed early (Chen et al. 2006).

Salient surgical principles of DC

Wide range of DC has been reported. In general three approaches are used fronto-temporo-parietal (hemicraniectomy), frontal and temporal. All of them can be performed unilaterally as well as bilaterally. The removed bone is transplanted in abdomen or cry-preserved for future use.

Fronto-temporo-parietal craniectomy (unilateral hemicraniectomy) provides the most extensive decompression for unilateral lesions while for diffuse cerebral edema bilateral craniectomy may be

performed.

Surgical technique Patient is placed in supine position with head elevated and rotated 30 to 45 degrees. Vertex is pushed downward so that the zygomatic arch comes in the upper most plane. Skin incision is in form of trauma flap with goal to expose following margins. Anteriorly to superior border of orbital roof (avoiding entry into the frontal sinuses), posteriorly to at least 2cm behind the external auditory meatus, medially to 2cm lateral to mid line (avoiding the sagittal sinus), and inferiorly to floor of the middle cranial fossa (Figure 1).

The temporalis muscle is reflected anteriorly. Burr holes are placed at the key hole, at the root of zygoma and along the planned craniectomy margin using high speed drill. The sphenoid wing is fractured and removed up to superior orbital fissure. Dural edges are tacked up (called as tacking sutures, hitch sutures, tenting sutures or sleeper sutures, first introduced by sir Walter Dandy) to bony margin or to muscles, to prevent epidural hematoma formation. Duraplasty is performed using dural substitute after dura is opened in stellate fashion. Duraplasty should be nonconstricting and loose to allow for further expansion of intra cranial contents. Craniectomy increase space by 15% but duraplasty increases it by 55%, which shows the importance of proper duraplasty in the procedure. If additional space is required non eloquent portion of brain may be removed in form of lobectomy.

Craniectomy size and margins: Recommendation of 10 x 15 cm craniectomy with lower margin extending to less than 1cm from the middle cranial fossa. Less than 8cm of craniectomy has been found to provide suboptimal expansion and also associated with infarct and hemorrhage in vicinity of craniectomy. Frontal approach and temporal approaches are well described are based on above principles.

Complications

1. Respiratory complications

Up to 65 % of comatose head injured patients become hypoxic even with apparently adequate respiration or while on hyperventilation. The exact cause remains obscure. But disseminated miliary atelectasis with pulmonary shunting may underlie this phenomenon. Pulmonary edema can occur as a direct result or server head trauma and is presumed to stem from massive sympathetic discharges consequent to injury to the hypothalamus, which results in acute pulmonary hypertension (neurogenic pulmonary edema). Fluid overload can also result in pulmonary edema. Patients with head injury are also at risk of developing fat embolism from long bone fractures and pulmonary emboli from deep venous thrombosis. All these can trigger the development of adult respiratory distress syndrome (ARDS).

In an intubated patient, the first indication of ARDS is a fall in pao₂. Therapy should be aimed at supporting the level of oxygenation while reversing any underlying treatable lesion. Fio₂ is increased to maintain the pao₂ above

80 torr. If fio₂ of 100% fails to accomplish this, then positive end-expiratory pressure (PEEP) ventilation should be instituted. Patients usually tolerate this to levels of 10 cm water without an increase in the intracranial pressure when the head is elevated to 30 degrees. Pulmonary edema secondary to fluid overload responds to diuretics and fluid restriction. Aspiration pneumonitis can be treated with antibiotics, steroids and diuretics. Low molecular weight heparin (if there is no coagulopathy) and compression stocking have been shown to be safe after craniotomy to prevent pulmonary emboli from deep vein thrombosis.

2. Fluid and electrolytes

The gold standard is to maintain isovolaemia. Fluid restriction may be harmful and this is particularly true in the multitrauma patient in whom tissue perfusion is vital. Fluid replacement at maintenance levels should be done with an isotonic fluid (0.9% saline) at a rate adjusted to avoid hypo tonicity and to maintain electrolyte balance. Fluid intake and output should be monitored and the serum electrolytes checked frequently. A CVP line will be helpful in fluid management. Hyponatraemia is the most important electrolyte imbalance since it promotes cerebral edema and, if severe enough or rapid in onset, can precipitate seizures. It may be iatrogenic in origin as a result of inappropriate volume replacement with hypotonic fluid or as a result of ADH secretion caused by the trauma, surgery, anesthesia and mechanical ventilation. The syndrome of inappropriate antidiuretic hormone secretion (SIADH) results in hyponatraemia serum hypo-osmolality, increased urine sodium and increased urine osmolality. Mild degrees of hyponatraemia can be managed by restricting fluid intake to 500 ml/day. Whereas more severe symptomatic levels of hyponatraemia may require 3 to 5 % of hypertonic saline, keeping in mind that correction should not be more than 12 meq /lit /day. The rapid correction of hyponatraemia can result in central pontine myelinolysis. As far as possible, the hyponatraemia must be corrected slowly with normal saline only. Diabetes insipidus can also result and should be treated with carbamazepine initially and DDAVP if it is very severe. Hypokalaemia may result from diuretics and steroids, and can be corrected by potassium supplementation.

3. Intracranial hypertension

Intracranial pressure can be lowered by elevation of the head to 30 degrees, avoidance of jugular vein compression and sedation, especially if the patient is restless. If the patient is on a ventilator he can be paralyzed, but paralysis alone without sedation should be avoided since noxious stimuli can cause an elevation of intracranial pressure even in a comatose patient. Hyperventilation sufficient to lower the paco₂ to 28-32 torr is the main stay of therapy. Diuretics, in combination with mannitol, are very effective in lowering elevated ICP. If elevated intracranial pressure does not respond to these measures, a CT scan of the brain should be done to look for surgically treatable lesions like a hematoma at the site of surgery, enlargement of a contra lateral clot or the enlargement of other clots which were not found to be significant at the time of the first CT scan. Development of hydrocephalus can also lead to an increase in intracranial pressure. If a surgically treatable cause is not found and the ICP remains elevated, barbiturate coma can be used, titrated to the burst suppression pattern on the EEG.

4. Seizures

Prophylactic anticonvulsants should be used in all patients with cerebral contusion and/or intracerebral hematoma. In spite of prophylactic anticonvulsants, seizures can occur in 5 % of patients in the post-injury period. They can cause an increase in intracranial pressure even in a paralyzed patient because of an increase in cerebral blood flow. If the blood flow cannot meet this metabolic demand because of intracranial hypertension or if the seizure activity is prolonged, further ischemic injury may result. If seizures occur despite medication, an underlying cause such as hypoxia, electrolyte abnormality or infection must be excluded. Major Head injury can lead to other complications like gastrointestinal ulceration (stress ulcers) and coagulopathy, but their detailed description is beyond the scope of this chapter.

5. Post operative intra cranial infections

Osteomyelitis, subdural empyema, meningitis and brain abscess are seen in 2% to 10% of patients. Perioperative prophylactic antibiotic and meticulous operative procedure and closure minimizes the risk. Once detected broad spectrum antibiotics are begun (third generation cephalosporin for example cefotaxime or ceftriaxone and amikacin with metronidazole). Once culture and sensitivity is received the appropriate antibiotics may be substituted. Use of higher end antibiotic should be kept in reserve for serous and life threatening infections.

6. Hyperthermia

It associated with worse out come. Conversely the use of mild systemic hypothermia (from 32° C to 33° C duration of 24 hours, and rewarming rate of 24 hours or less) in severe head injury yielded some promising early results. But it

was also found to increase the complication rate like increase in infection rate, hematologic and metabolic complications. The present trend is to aggressively maintain normothermia (core temperature <37.5°C).

7. Intracranial complications

Recurrent hematoma(EDH,SDH,ICH,IVH),hydrocephalus may developed post operatively and there for observation specially by scanning as well continuous monitoring of ICP by subdural intra parenchymal or intraventricular probes (Upadhyay and Srivastav, 2007). Intra ventricular tube can also be use for CSF drainage and control of ICP. Early detection of complications should be aim. If so found, appropriate, urgent therapy give better prognosis and outcome.

REFERENCES

1. Chen C, Smith ER, Ogilvy CS, Carter BS. Decompressive craniectomy: physiologic rationale, clinical indications, and surgical considerations. In: Schmidek & Sweet Operative Neurosurgical Techniques: Indications, Methods, and Results. Elsevier, Philadelphia, 2006, 70-80
2. Dunn IF, Ellegala DB. Decompressive hemicraniectomy in the management of sever Traumatic brain injury in Acute Brain and spinal cord Injury. Ed Bhardwaj, Ellegala & Kirch, 2010 page 3-4
3. Guerra WK, Gaab MR, Dietz H, Mueller JU, Piek J, Fritsch MJ. Surgical decompression of traumatic brain swelling: Indication and result. *J Neurosurg.* 1999, 90(2), 187-196 [PMID: 9950487]
4. Polin RS, Shaffrey ME, Bogaev CA, Tisdale N, Germanson T, Bocchicchio B, Jane JA. Decompressive bifrontal craniectomy in the in the treatment of severe refractory post traumatic cerebral edema. *Neurosurgery*, 1997, 41(1), 84-92 [PMID: 9218299]
5. Taylor A, Butt W, Rosenfeld J, Shann F, Ditchfield M, Lewis E, Klug G, Wallace D, Henning R, Tibballs J. A random trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. *Childs nerv syst.* 2001, 17(3), 154 -162 [PMID: 11305769]
6. Upadhyay PK, Srivastav A. Supra and Infra Tentorial Acute Extra Dural Hematoma. *Indian Journal of Neurotrauma*, 2007, 4(1), 59-61
7. Whitfield PC, Patel H, Hutchinson PJ, Czosnyka M, Parry D, Menon D, Pickard JD, Kirkpatrick PJ. Bifrontal decompressive craniectomy in the management of post traumatic intra cranial hypertension. *Br J Neurosurg.* 2001, 15(6), 500-507 [PMID: 11814002]