REVIEW

A review on decompressive craniectomy for traumatic brain injury: the mainstay method for neurotrauma patients

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Abstract

Raised intracranial pressure is common leading cause of mortality in patients suffering from a traumatic craniocerebral injury. Currently, head injury constitutes a major public health problem across the world. Decompressive craniectomy is currently emerging as a preferred treatment strategy for patients suffering from refractory intracranial hypertension, which is unresponsive to appropriate neurocritical care management. The meticulous execution of decompressive craniectomy requires an understanding of anatomy, the pathogenesis of raised intracranial pressure, meticulous surgical technique, proper planning in association with the competent anesthetic team and paramedical staff to provide improved neurological outcome, and a significant reduction in mortality and morbidity. We provide a review of the status and appropriate review of this surgical procedure.

High Lights

- After over 100 years of the decompressive craniectomy (DC) procedure, clear indications are not present.
- Great variability exists for this technique, yielding variation in outcomes and creating comparative data.
- Decompressive craniectomy has been shown to decrease intracranial pressure and mortality.
- Decompressive craniectomy might increase vegetative state rates.

Keywords Head injury, Raised intracranial pressure, Decompressive craniectomy, Management, Neurological outcome

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History of decompressive craniectomy

Decompressive craniectomy (DC) was first described by Annandale in 1894 [1, 2]. In the later part of the nineteenth century, almost all pioneers in neurosurgery have performed craniectomies as a palliative measure for raised intracranial pressure, however, in 1901, Kocher took the lead to suggest palliative decompressive craniotomy for patients with traumatic brain injury and uncontrolled raised intracranial pressure [3]. Kocher and Harvey Cushing collaboration resulted in the proposition of the use of DC for the treatment of other brain

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conditions like brain tumors and vascular malformations through subtemporal and suboccipital decompressions [4, 5]. In 1908, Cushing published subtemporal decompressive operations for intracranial complications associated with bursting fractures of the skull [6].

Annandale described a procedure of DC, which gained popularity in the early 1970's, but due to poor clinical outcomes, quickly fell into disrepute [1, 2], and it was almost abandoned when experimental evidence suggested that decompression worsen cerebral edema [7]. In 1971, Kjellberg et al. reported 73 cases, which underwent large bifrontal craniectomy with 18% of surveillance [8]. In 1975, Venes and Collins reported retrospective analysis involving a total of 13 patients who underwent bifrontal decompressive craniectomy for the management of posttraumatic cerebral edema and observed a drastic reduction in mortality; however, survivors continue to have severe morbidity [9].

However, the popularity of DC started too returned throughout the 1980's. In 1980, Gerl and Tavan reported the role of extensive bilateral craniectomy and of dura opening offered the possibility of rapid reduction in intracranial pressure in a study involving 30 patients, mortality was 70%, and full recovery in 20% of the patients [10]. In 1990, Gaab et al. [11] analyzed 37 patients in a prospective study where 19 cases underwent bifrontal craniectomies and 18 had hemicraniectomies performed; they reported 5 deaths, with the rest of the cases achieving full social rehabilitation or remaining moderately disabled, authors further established initial posttraumatic Glasgow coma scale (GCS) $\geq$ 7, as the best predictor of a neurological outcome. Recent studies also showed the efficacious role of DC in a traumatic brain injury with raised intracranial pressure [12–15].

Traumatic brain injury (TBI): a critical public health problem

Injury-related morbidity and mortality is increasingly being recognized as a major public health ranking among the leading causes of death and affecting people in all age and income groups [16]. A traumatic brain injury will account for an increasing number of deaths worldwide by 2020 [17]. The Centers for Disease Control and Prevention (CDC) have defined TBI as an injury to the head inflicted with blunt or penetrating trauma or from acceleration/deceleration forces associated with at least one or more of the following: amnesia decreased level of consciousness, objective neurologic or neuropsychological abnormality [18, 19].

Traumatic brain injury is associated with mortality rates as high as 30% and with high morbidity [20, 21]. There is a significant percentage of TBI related deaths that occur relatively late, these are secondary to multiple organ failures and infectious complications, such as pneumonia [20–23]. Traumatic brain injury is also the most common cause of death and disability in children and young adults [24, 25].

Some of the important features of patients without a severe TBI are the subsequent mental and medical problems [26, 27]. The acute consequences of TBI are just about half of the burden but long-term sequalae, especially among adolescents and young adults, whose brains continue to mature and develop, are substantial [28]. Approximately 293,000 persons with ages between 15 and 24 years old took advice at emergency department treatment for TBI related cases in the USA in 2010 [29].

In Latin America low-middle income countries (LMIC), the rate of good recovery is similar to results in high-income countries (HIC), but rate and settings outweigh any other variable in predicting the outcome [30].

Sustained, raised intracranial pressure (ICP) refractory to medical therapy

Morbidity and mortality related to brain injury are mostly caused by mass effect and brain edema. In neurotrauma, brain edema leads to an elevation in intracranial pressure (ICP), causing an alteration of the cerebral perfusion pressure (CPP) and brain oxygenation [31]. Edema development plays a role in the resulting pathology following TBI [32], a secondary injury caused by a cascade of mechanisms initiated at the moment of injury. The pathophysiology of the primary and secondary lesions in TBI is the targets to prevent and wane the progression of brain damage. Raised intracranial hypertension is a frequent complication of severe TBI [33–35], about 70% of brain-injured patients will present with raised ICP [36–39]. Traumatic brain injury is the most common cause of intracranial hypertension [40]. Intracranial hemorrhage is the most frequent cause of death and disability following severe TBI [41–43]. Sustained raised intracranial pressure is defined as the presence of ICP above 20 mmHg is a known independent risk factor for poor neurological outcomes [44].

Intracranial pressure is determined by the volume of the content inside the cranial cavity: brain, blood and cerebrospinal fluid (CSF). According to Monroe–Kelly doctrine [45, 46], "the sum of the intracranial volumes of blood, brain, CSF and other components is almost constant and an increase in any one of these must be offset by an equal decrease in another" (see Fig. 1) [47]; the skull is a rigid structure; in order to maintain a constant blood pressure, the volumes inside the cranium should be constant. Any increase or an additional volume (e.g., hematomas, edema, hyperemia) will carry an increase in the ICP. Alterations in brain autoregulation, blood flow and brain edema are consequences of raised ICP,
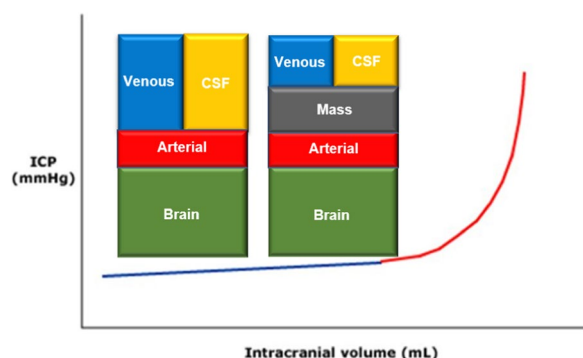
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Fig. 1 Monroe–Kelly doctrine

TBI patients with refractory ICP and have worse outcomes, more likely to develop herniation syndromes [48, 49]. A CPP lesser than 60–70 mmHg is associated with diminished oxygenation and altered metabolism in brain parenchyma [50].

The raised ICP results in "spatial compensation," extrusion of CSF and blood (mainly venous) from the intracranial cavity. Cerebrospinal fluid has a key role in spatial compensation because it can be expelled to the spinal theca, the reservoir [51, 52]. The intracranial cavity can store up to 150 mL of new volume without a significant increasing ICP; this occurs because the venous blood can be derived from the general circulation [45]. The CSF shift is time- and age-dependent variable. Older people can accommodate more of the expanding new volume due to the additional space created by cerebral atrophy; conversely, young people get symptomatically faster, due to the lack of space.

Among deleterious effects, increased ICP is the shift of brain parenchyma resulting in structural damage to the brain and to herniation syndromes and can cause compression on the brainstem causing bradycardia and hypertension and, if untreated, respiratory depression and death may follow [52–54].

In the context of raised ICP, the cerebral perfusion pressure (i.e., the difference between ICP and mean arterial blood pressure) generally decreases, contributing to the reduction in cerebral blood flow and producing ischemia and neuronal death, but reduced CPP is associated with the hypoxic/ischemic injury regardless of the ICP [40, 55].

According to the guidelines from the Brain Trauma Foundation, when ICP rises > 22 mmHg, measure should be taken to lower ICP [56], which include primary measures like elevation of head of the bed to 30 degrees (might cause hypotension), mechanical ventilation to achieve $PaCo_2$ 35–40 mmHg, normovolemia, Propofol (2–4 mg/kg/h) (may cause hypotension and Propofol infusion syndrome), evacuation of intracranial mass lesions (surgery associate risks), seizures treatment (adverse effects depending on the drug used); secondary measures include, increase sedation, neuromuscular blockage (neuropathy and myopathy), hyperosmolar agents (mannitol which may cause hypotension and hyperosmolarity, hypertonic saline), normothermia and CSF drainage (infection and bleeding risk); if the above fails, induced hypertension (risk of acute lung injury) is achieved with moderate therapeutic hypothermia (risk of arrhythmia, infection and electrolyte abnormalities) +/− barbiturates use [57]. if all the measures described fail, refractory intracranial hypertension is considered and a DC can be the best next step to improve ICP, CPP, MAP, pressure–volume compensatory reserve (RAP) index and cerebrovascular pressure reactivity index (PRx) [58].

The neurosurgical technique of decompressive craniectomy (DC)

Routinely, two type of craniectomies is performed in cases with traumatic brain injury with sustained raised intracranial pressure (i.e. hemi craniectomies and bilateral craniectomies) [59]. When deciding the surgical plan for craniectomy, multiple factors need to be considered, including location, hemisphere, size of the decompression, dural technique, the bone flap, etc. The CT scan may aid in deciding location (frontal, temporal, parietal, occipital or different combination), size, and extent, and unilateral or bilateral hemisphere need of other procedure like lax duraplasty, external ventricular drain.

Once the decision is made, the bone removal should be tried at its maximum as possible extent to make a larger size craniectomy, and possible recommended size of the decompression should be at least 14 cm (anteroposterior) by 12 cm (superoinferior) if the intention is to perform a frontotemporoparietal craniectomy to create spaces for expansion of brain [60].

The ideal technique implies the removal of bone in the entire supratentorial Hemi cranium. One of the most important landmarks for this procedure is the root of the Zygoma; it allows the identification the floor of the temporal fossa. Also, are important landmarks: the asterion (confluence of the lambdoid, occipitomastoid, and parietomastoid sutures, indicates the area of transition between the transverse and sigmoid sinuses), the keyhole (identifies the pterion and indicates the confluence location of the frontal, temporal, and orbital cavities), the inion, the glabella and the midline (delineates the course of the superior sagittal sinus). When the patient's head is placed in the head holder, it is ideal that the sagittal plane of the head is turned 0–15° to the floor on opposite side (Fig. 2) [61]. Two techniques for unilateral and bilateral decompressive craniectomy are described separately.
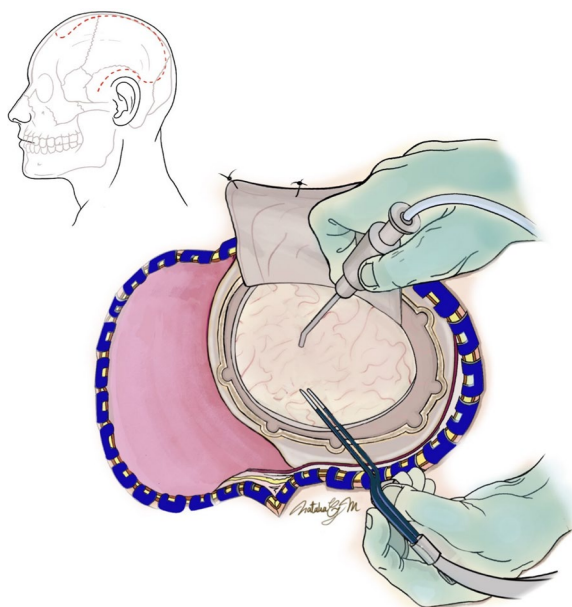
Fig. 2 Operative technique of decompressive craniectomy

A. Unilateral decompressive craniectomy
a. Scalp incision and flap raising for decompressive hemicraniectomy

Because the objective is the exposure of the entire hemicranium, there are two incisions that allow this goal to be achieved.

1. Reverse question mark frontotemporoparietal scalp cranial incision

This incision is quick and easy but has the potential risk of flap ischemia and dehiscence of the wound. It begins at the widow's peak, continues posteriorly along the midline to the inion, and it then turns sharply to the ear parallel to a line extending from the inion to the root of the zygoma [62]. The incision should skirt the superior and anterior portions of the ear as close as possible and extend 1 cm below the root of the Zygoma. With this incision, the hemi cranium is exposed at the midline, along with the line of the transverse sinus. Also, permits a great temporal fossa exposition. Skirting the ear avoids the superficial temporal artery; it ensures a good blood flow to the skin flap [63]. Now, the periosteum can be incised using an electrocautery knife, and then, the cutaneous flap can be reflected. The temporalis muscle can be reflected in two ways [63]:

Reflect the skin anteriorly as a separate layer of muscle, this facilitates the temporalis muscle reflection.

Reflect the muscular and the cutaneous flaps together, as a single piece. This allows to conserve the original muscle position in the absence of an underlying attachment to the bone and preserve cosmetic.

2. Modified L.G. Kemp scalp incision or midline sagittal incision with "T-bar"

With this incision, a better blood flow to the skin flaps is achieved, lessen the dehiscence risk, especially in the posterior portion of the incision, the weakness of the previously described incision [63]. The scalp is incised in the midline from the widow's peak to the inion; then, a limb to the incision is performed, forming the "T-bar" from 1-2 cm anterior to the tragus, extending superiorly, 1 cm behind the coronal suture until encountering the midline sagittal incision [61–63].

3. Proposed Novel Skin flap incision

In 2019, Feng and Cols proposed a new technical for skin flap in Craniectomy. In patients with anesthetic induction, head rotation to 60° to the contralateral side while positioned in a horseshoe head holder with a roll positioned under the ipsilateral shoulder. The midline of the skull is marked from the nasion to the inion [64]. The marked sites are: estimated locations of the sigmoid and transverse sinuses, 3 cm posterior to the ear and 1 cm above the ipsilateral transverse-sigmoid sinus junction. The incision is curved posteriorly as it approaches the midline and continues 1–2 cm off midline to 87 the hairline anteriorly [64].

Prior to the incision, a local anesthetic (lidocaine plus epinephrine) is injected. the incision is made all the way up to the skull, and all layers of the scalp are reflected inferiorly [64].

b. Raising bone flap for decompressive hemicraniectomy

Superior to the root of the zygoma, a single burr hole is made, to delineate the floor of the temporal fossa. The asterion should be exposed by reflecting the soft tissue caudally, through this manoeuvre the inferior extent of the temporal and occipital lobes can be visualized [63]. After doing the burr hole, a footplate is inserted and the bone flap is turned by extending the beginning of the craniectomy along the line toward the inion. To avoid the transverse, sinus is necessary to stay at least 1 cm rostral to the asterion. The lambdoid suture will be crossed when the bone flap is extended posteriorly; then, the drill is turned parallel to and 1 cm medial to the lambdoid suture until reach to a point 1 cm from the midline [63].

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The drill is then turned parallel to the sagittal sinus, again crossing the lambdoid suture. Drilling continues toward the supraorbital bar. The craniotomy is continued anteriorly by hugging the floor of the frontal fossa as closely as possible, staying as close to the orbital rim as the anatomy allows. Next, the drill is turned posterolaterally toward the keyhole and aimed as close to the pterion as possible. At this point, the drill is removed and re-inserted into the bur hole at the root of the zygoma.[63].

The second drill line is created by hugging the floor of the temporal fossa and extending it as far anteriorly as possible toward the temporal tip. The bone flap is removed by levering it using the pterion as fulcrum [63]. Usually, the pterion cracks on removal and the dura can be dissected using Rhoton dissectors. Leksell rongeurs are used to remove bone excess; it is necessary to smooth the edges of the bone flap [62, 63, 65].

B. Bilateral decompressive craniectomies
a. Scalp incision and flap raising for bilateral decompressive craniectomies

Bilateral craniectomies are especially useful in cases of bilateral frontal contusions or generalized cerebral edema without focal lesion [61]. Can be performed in two ways:

1. Performing separately two hemicraniectomies

This will result in the midline a strip of bone 2–3 cm wide, for covering the superior sagittal sinus. Can be used either the midline sagittal incision with "T-bar" or the large reverse question mark frontotemporoparietal incision [63].

2. Kjellberg type incision mark

In this, is made a standard bicoronal incision, beginning 1–2 cm anterior to the tragus, going superiorly behind the coronal suture until finding the opposite root of the Zygoma, also ending 2 cm anterior to the tragus [62]. The flap resulting is reflected anteriorly and inferiorly exposing the frontal and anterior temporal lobes [63].

b. Bone flap raising for bilateral decompressive craniectomies

The bur holes are placed in the keyhole and in the root of the Zygoma just below the superior temporal line. This type of craniectomy can be removed as a single piece or a strip of bone can be left over the sagittal sinus for protection, then resulting two bone flaps.

The bilateral frontal and subtemporal craniectomies will be performed, so the firs drill line extends from Zygoma, ascending and crossing the sagittal superior sinus until reaching the contralateral zygoma; the second drill line extends from keyhole to keyhole, crossing 1 cm parallel and superior to the orbital rim; then, other tow drill lines will be performed, are made from the zygomatic bur hole going anteriorly, hugging the floor of the temporal fossa toward the temporal tip and extending superiorly and anteriorly toward the keyhole [63]. This will result in the exposing of the frontal and anterior temporal lobes. The craniectomy can be enlarged using Leksell rongeurs, especially the subtemporal craniectomy [62].

c. Dural opening methods

For this step, it can be used three different ways of opening the dura with fish-mouth incision, stellate incision, C-shaped fashion incision and cruciate incision [62, 65]. The C-shaped fashion is one of the most used incisions for dural opening, it goes from the temporal tip of the temporal lobe, and curving back about 8 cm crossing the Sylvian fissure, and ending in the frontal region [63]. For allow, brain swelling can be practiced spoke-wheel relief cuts. The dural flap is reflected anteriorly. Now, the underlying hematomas can be evacuated. Once hemostasis is ensured, the dura leaves can be laid back over the brain surface [63], and a large piece of dural substitute is placed over the opened dura [62].

d. Usefulness of Dural substitutes and sealants for duraplasty in the DC procedure

In dural closure, it can be used absorbable gel sponges like dural substitutes and dural sealants. The dura substitutes are designed to be either placed as an only over dural defects or sutured into place [66]. There could be autologous tissues, such as pericranium or fascia lata, or artificial dural substitutes mainly derived from bovine tendon among others derived from foetal bovine skin [66].

With the aim to reinforce primarily repaired dura or as adjuncts to dural substitutes, dural sealants can be used including Dura Seal, Bioglue and Evicel [66].

e. Scalp closure

The scalp is closed over the absorbable gel sponge or the dura substitute or sealant using 2-0 Vicryl stitches for the galea. Typically, staples are used to close the skin.

f. Placement and storage of Craniectomy bone flap following Decompressive craniectomy

The craniectomy bone flap can be discarded, inserted in an abdominal subcutaneous pocket in the left lower quadrant or conserved in a bone bank [62, 63, 67]. The consequences of discarding the bone flap are obvious, require a cranioplasty with intraoperative reconstruction, making expensive the procedure, and sacrificing cosmetics. Some centers are preferred to discard it and use 3D methyl methacrylate prosthetic implants [62], especially because over one-half of the patients with severe CNS injury had concomitant systemic infectious processes of some type [68]. When the bone flap is conserved inside the body, it usually remolds the bone edges to some degree. So, keeping the bone frozen in a bone bank is an option with no risks of bone remodeling and offers great cosmetic outcomes [63].

When the autologous bone graft is not available for cranioplasty, synthetic materials such as tantalum, silastic, titanium plate, prefabricated acrylic, synthetic bone substitute and other similar material manufactured for the use of implantation into the body can be used [67].

Hinge or floating craniotomy

Patient with general anesthesia the surgeon make a scalp incision and bone flap is removed and put three titanium bone plates and screws placed around its periphery. A large dural opening is then created, and the hematoma is evacuated [69].

In cases of obvious cerebral edema, the dura is left open but laid back over the brain, and the exposed brain is covered with a sheet of compressed Gelfoam or Duragen. The bone flap is then returned to the operative field. An anterior superior titanium Y-shaped plate is secured to the surrounding skull in a manner that allow the bone flap to rise as cerebral edema occurs. Proper orientation of the hinged bone flap in the wound can be maintained by placement of a Y- or T-shaped titanium plate secured to the surrounding bone at the site of the hinge. The galea is closed with sutures, the skin with staples, and the head is gently wrapped with gauze. After a few weeks, with relaxation of brain swelling because of the mobility of the flap [69].

The important fact of DC

Decompressive craniectomy is a surgical procedure that reduces the secondary damage due to an uncontrolled increase in ICP but does not heal the primary lesion [70]. Inappropriate techniques for DC, e.g., do not smooth the bony edges; do not try at maximum to do bone removal as large as possible performing wrong approaches like only subtemporal decompression, or only frontotemporal decompression, can generate iatrogenic brain lesion, and even generate brain herniation through the craniectomy. Do not being faithful to the technique described can result in patient dead or in poor outcomes.

The inconsistent results and the conflicting opinions related to the DC can be due to the substantial variation of its use. It is imperative that DC must be performed with standardized technical guidelines as proposed by Quinn et al. in 2010 [61].

DC evidence

Table 1 summarizes the evidence from major published studies on decompressive craniectomy and its impact on the management of traumatic brain injury.

The following are the key clinical trials published on the role of decompressive craniectomy in traumatic brain injury (TBI):

- In 2001, a DC RCT in Melbourne, Victoria, Australia on 27 children older than 12 months-old found that children had better functional outcome (54% compared to control group 14%) and lower ICP (17.4 ± 3.4 mm Hg vs. 21.9 ± 8.5 mm Hg) after DC compared to medical management alone [69].
- In 2009, an RCT in Hangzhou, China on 74 adults between 18–65 years of age were assigned to a unilateral DC group and unilateral temporoparietal craniectomy group; lower ICP was seen in the DC group (15.98 ± 2.24 mm Hg vs. 21.05 ± 2.23 mm Hg) and lower mortality (27% vs. 57%) [71].
- In 2011, Cooper et al., in 15 tertiary care hospitals in Australia, New Zealand, and Saudi Arabia on 155 patients between 15–59 years of age with TBI and refractory intracranial hypertension (RICH), received bifront-temporoparietal (BFTP) DC or standard care, patients with BFTP-DC had lower ICP (14.4 mm Hg vs. 19.1 mm Hg, $p < 0.001$), shorter duration of mechanical ventilation (11 days vs. 15 days, $p < 001$), ICU stay (12 days vs. 18 days, $p < 0.001$), length of stay was reduced but not statistically significant (28 days vs. 37 days, $p = 0.82$), but greater risk of unfavorable outcome (51% vs. 42%, $p = 0.02$; OR = 2.21; 95% CI 1.14–4.26, $p = 0.02$). Please note that this particular trial recruited patients who presented with ICP > 20 mmHg for 15 min [72].
- In 2016, the largest RCT was published by Hutchinson et al. (RESCUEicp Trial), this study included 408 patients, 10–65 years of age from 73 institutions, from 23 countries (UK, Brazil, Canada, China, Czech Republic, France, Germany, Greece, Hong Kong, Hungry, India, Israel, Italy, Japan, Latvia, Malaysia, Peru, Russian Federation, Kingdom of Saudi Arabia, Singapore, Spain, Turkey and USA) also applied

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Table 1 Evidence of decompressive craniectomy in traumatic brain injury: a summary of the outcomes

Study	Country	Population	Outcome	ICP levels
Taylor et al. [69]	Australia	*N* = 27 Age (13.6–176.4 Months)	Favorable Functional Outcome (GOS > 5) *Decompressive Craniectomy group* 54% *Control group* 14%	*Decompressive Craniectomy* 17.4 ± 3.4 mm Hg *Control* 21.9 ± 8.5 mm Hg
Qiu et al. [71]	China	*N* = 74 Age (18–65 years old)	Favorable Functional Outcome (GOS > 5) *Decompressive Craniectomy group* 56.7% *Control group* 32.43% Mortality *Decompressive Craniectomy group* 27% *Control group* 57%	*Decompressive Craniectomy* 15.98 ± 2.24 mm Hg *Control* 21.05 ± 2.23 mm Hg
Cooper et al. [72]	Australia New Zeland Saudi Arabian	*N* = 15 Age (15–59 years old)	Unfavorable Functional Outcome (GOS < 5) *Decompressive Craniectomy (51%) versus Control (42%)* OR 2.21 (95% CI 1.14–4.26, *p* = 0.02)	*Decompressive Craniectomy* 14.4 ± 6.8 mm Hg *Control* 19.1 ± 8.9 mm Hg
RESCUEicp Trial [73]	23 Countries	*N* = 408 Age (10–65 years old)	Unfavorable Functional Outcome (GOS < 5) At 6 Months *Decompressive Craniectomy 72% (146/202) versus Control 70.4% (138/196)* At 12 Months *Decompressive Craniectomy 68% (132/194) versus Control 79.32% (142/179)*	*Decompressive Craniectomy* 14.5 (1.7–18) mm Hg *Control* 17.1 (4.2–21.8) mm Hg
DECRA Study [74]	10 Countries	*N* = 155 Age (18.5–34.9 years old)	Mortality OR 1.58 (95% CI 0.84–2.99; *p* = 0.16) Vegetative state at 6 Months OR 5.85 (95% CI 1.21–28.30; *p* = 0.03) Severe disability at 6 Months OR 2.49 (95% CI 1.21–5.11; *p* = 0.01)	*No reported*

to TBI and RICH. Extended Glasgow Outcome Scale (GOS-E) at 6 months showing death (26.9% vs. 48.9%), vegetative state (8.5% vs. 2.1%), lower severe disability (21.9% vs. 14.4%), upper severe disability (15.4% vs. 8.0%), moderate disability (23.4% vs. 19.7%) and good recovery (4.0% vs. 6.9%). The GOS-E at 12 months, death (30.4% vs. 52.0%), vegetative state (6.2% vs. 1.7%), lower severe disability, (18.0% vs. 14.0%), upper severe disability, (13.4% vs. 3.9%), moderate disability (22.2% vs. 20.1%) and good recovery (9.8% vs. 8.4%). This trial included patients with ICP > 25 mm Hg for 1 to 12 h [73].

- In 2020, a RCT DECRA Study was published in the Journal of Neurotrauma. The mortality rate in patients after craniectomy was 11% higher (59% compared with 48%), but it was not significantly different from standard care (OR 1.58; 95% CI 0.84–2.99; *p* = 0.16). Among the survivors after craniectomy, there were fewer good outcomes (OR 0.33; 95% CI 0.12–0.91; *p* = 0.03) and more vegetative outcomes (OR 5.12; 95% CI 1.04–25.2; *p* = 0.04). Similar outcomes were observed in survivors at 6 months after the injury, with an increase in vegetative outcomes (OR 5.85; 95% CI 1.21–28.30; *p* = 0.03) and severely disabled outcomes (OR 2.49; 95% CI 1.21–5.11; *p* = 0.01) [74, 75]. Bifrontal DC is not recommended

to improve outcomes as measured by the GOS-E score at 6 mo 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 min 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 ICU [56].

A large frontotemporoparietal DC (not less than 12 × 15 cm or 15 cm diameter) is recommended over a small frontotemporoparietal DC for reduced mortality and improved neurologic outcomes in patients with severe TBI [56]. The latest meta-analysis published by Zhang et al. [75, 76], which included the four RCTs, five retrospective studies and one prospective study and previous meta-analysis, found that DC lowers ICP, reduce mortality rate but increase incidence of complications, but benefits on functional outcomes are not statistically significant just yet.

Conclusions

The cranial decompression techniques are lifesaving surgeries in expert hands. Neurotrauma surgery is a complex surgical procedure, usually performed by residents and young neurosurgeons. Authors suggest the use of a

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checklist during decompressive craniectomy to ensure that the procedure is performed in a proper guideline.

Abbreviations

DC   Decompressive craniectomy
TBI   Traumatic Brain Injury
CDC   Centers for Disease Control and Prevention
LMIC   Latin America low-middle income countries
HIC   High-income countries
ICP   Intracranial pressure
CPP   Cerebral perfusion pressure
CSF   Cerebrospinal fluid
MAP   Mean arterial pressure
RAP   Pressure–volume compensatory reserve
PRx   Cerebrovascular pressure reactivity index
CT   Computed tomography
Cm   Centimeter
RCTs   Randomized clinical trials
RICH   Refractory intracranial hypertension
BFTP   Bifront temporoparietal
ICU   Intensive care unity

Availability of data and materials
Not applicable.

Declarations

Ethics approval and consent to participate
The present manuscript has not been submitted to other journal for simultaneous consideration. The manuscript has not been published previously and is not split up in other parts, no data have been manipulated or fabricated, and no information is presented as if were from the authors.

Consent for publication
Valid informed written consent of the guardian was taken to publish this review. They were informed that the details of patient will not be disclosed.

Competing interests
The authors declare no competing interests.

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