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Surgical Treatment of Severe Traumatic Brain Injury

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1. Introduction

Head injury is the number one cause of trauma-associated mortality, being directly associated with approximately half of all trauma-related deaths [1]. Every year in the United States, approximately 1.5 million head injuries occur, resulting in 250,000 hospitalizations and 52,000 deaths [2]. Traumatic brain injury (TBI) is the leading cause of death in persons less than 45 years of age [3]. Furthermore, by World Health Organization estimates, TBI will be the third leading cause of death and disability, across all age groups, by the year 2020 [4]. From a cost perspective, TBI results in an astounding $6 billion in direct costs and over $40 billion in indirect costs annually in the United States [5].

For neurosurgeons and intensivists involved, the management of TBI presents many challenges. Many patients with TBI also have traumatic injury to other organ systems, further complicating management. Centers treating a high volume of severe TBI may have better outcomes in terms of mortality and quality of life [6].

Current management of severe TBI consists of a host of surgical and non-surgical modalities. The majority of patients with severe TBI, defined by Glasgow Coma Scale (GCS) 3-8, will be managed nonsurgically. Medical interventions are generally used to optimize intracranial pressure (ICP), maintain cerebral blood flow and oxygen delivery, minimize cerebral edema and maintain a healthy metabolic environment [7]. Surgical treatment in severe TBI is most commonly used for evacuation of intracranial hemorrhage (ICH), especially when there is decreased level of consciousness, focal neurologic signs and/or evidence of intracranial hypertension [7]. Prior to publication of the "Guidelines for the Surgical Management of Traumatic Brain Injury" in 2006, the role of surgery was often based on individual surgeon preference or subjective factors [8]. As noted by the guideline authors, there is a paucity of prospective, randomized controlled trials for surgical

lesions in TBI, which precluded literature categorization with traditional "level of evidence" distinctions [8]. Instead, the guidelines offer "literature-based recommendations" for the following TBI lesion types warranting surgical consideration: acute epidural hematoma (aEDH), acute subdural hematoma (aSDH), traumatic parenchymal lesions, posterior fossa mass lesions, and depressed cranial fractures [8].

This chapter will review the aforementioned lesion types with respect to epidemiology, guidelines-based indications for surgery, timing, and technique. We will also review new data pertinent to each lesion type that has been published since the 2006 "Guidelines for the Surgical Management of Traumatic Brain Injury." We will pay particular attention to the surgical management of intractable intracranial hypertension, which has become the matter of intense debate in recent years. This debate relates to the publication of the DECRA (Decompressive Craniectomy in Diffuse Traumatic Brain Injury) trial in 2011 [9]. We will review this work in depth, as well as, the controversy surrounding its application to patient care.

2. Acute epidural hematoma

Epidural hematoma occurs in 2.7 – 4 % of TBI patients[10]. They most frequently occur in the temporal and temporoparietal regions as a result of linear skull fracture with subsequent damage to either the anterior or posterior divisions of the middle meningeal artery. Traumatic epidural hematoma of nonarterial origin may result from middle meningeal vein, diploic emissary vein, or venous sinus bleeding and accounts for roughly 25% of total cases [11]. Epidural hematoma in patients over 65 years of age is rare, due to the prominently adhered dura to the overlying skull. The classically described "lucid interval," with rapid deterioration after a period of post injury wakefulness occurs in about half of surgical cases[8]. Isolated EDH mortality is lower than that for ASDH and approximates 10%[12]. EDH represents one of the most urgent neurosurgical lesions, as severe brain compression can develop rapidly from high-pressure arterial bleeding often necessitating rapid evacuation. In the appropriate patient, surgical evacuation of posttraumatic epidural hematoma has been shown to be impressively cost-effective with regards to quality and duration of life preserved, when compared to other surgical procedures[13].

Guideline based indications for surgery [8]: aEDH larger than 30cc should be evacuated regardless of the patient's GCS score. In patients with GCS > 8 and no focal neurologic deficit, nonoperative management with serial CT scan and close observation in a center with available neurosurgical services can be considered if the hematoma meets the following criteria: volume < 30cc *and* less than 15mm thick *and* < 5mm of midline shift. It is important to mention that lesions in the temporal or posterior fossa may cause significant brainstem compression, associated with high mortality, in the absence of large size, significant midline shift, or elevated ICP [12,14,15]. These patients should have a much lower threshold for surgery.

Timing [8]: Patients meeting the above mentioned criteria should undergo surgical evacuation as soon as possible. Expeditious hematoma evacuation is particularly vital for comatose patients (GCS < 9) and/or with anisocoria.

Surgical technique [8]: There are insufficient data to support one surgical treatment method. Most authors would recommend craniotomy over simple burr hole evacuation in order to provide adequate access to and evacuation of the offending clot. Traditional teaching was for a large "question-mark" or "T" shaped incision and subsequent large trauma bone flap. With improved imaging quality and technology, the ability to localize the clot location prior to incision often allows for a smaller linear incision and more focused craniotomy which can be expanded if the need arises [7]. Also common practice is to make a small dural opening in order to inspect for a concomitant SDH which may sometimes develop with "reperfusion" or resuscitation of the trauma patient [7].

3. Acute subdural hematoma

Acute subdural hematoma is more common than aEDH, occurring in about 30% of severely head injured patients [16]. The most common causes of these lesions include motor vehicle accidents, falls (particularly in those > 75 years of age) and assaults. By definition, aSDH after trauma occurs within 14 days of injury and is associated with a higher mortality rate than EDH, with or without surgical intervention[12,16]. Compared to isolated EDH, the degree of underlying brain damage associated with aSDH is more severe [17]. Mortality rates traditionally quoted for those requiring surgery vary between 40 – 68% and is greatest in those with increased age, poor initial GCS, and other associated brain and systemic injuries [12]. A recent report observed 16% inpatient mortality for all comers with subdural hematoma, which did not vary significantly for those undergoing surgical intervention [18].

Guideline based indications for surgery [12]: Thickness greater than 10mm or midline shift greater than 5mm on CT should undergo surgical evacuation regardless of GCS score. Patients with GCS < 9 should undergo intracranial pressure monitoring. For comatose patients with less than a 10mm thick lesion or less than 5mm of midline shift, indications for surgical evacuation include a decrease of GCS by 2 or more points, ICP > 20mmHg, or asymmetric or fixed and dilated pupils.

Timing [12]: Most authors would agree surgical candidates should undergo evacuation as soon as possible. This principle was originally based on a study conducted over 30 years ago showing marked improvement in mortality if aSDH was evacuated within 4 hours of injury. Some have since questioned this notion claiming either no outcome difference, or a worsened outcome with more rapid time to evacuation [16,19,20]. However, a careful review of this data reveals that the patients who underwent rapid evacuatoin also had more severe neurologic injury prior to surgery, challenging the validity of the outcomes data [12,16,20].

Surgical technique: Craniotomy, generally via a large frontotemporoparietal approach, with or without bone flap removal and duraplasty is the preferred technique [12]. Multiple techniques for aSDH evacuation have been utilized in neurosurgery including trephination via twist drill or burr hole, craniotomy +/- duraplasty, subtemporal decompressive craniectomy, and large decompressive hemicraniectomy +/- duraplasty. For patients with poor GCS associated with aSDH, trephination and irrigation without craniotomy maybe associated with

poorer outcomes compared to craniotomy or craniectomy [12,21]. Most studies comparing outcomes of those undergoing craniotomy vs. craniectomy suffer from selection bias as patients with more severe injury undergo craniectomy and worse outcomes based on initial presentation[12,22]. A subgroup of patients with a higher level of brain injury may benefit from decompressive craniectomy [23], and it is our practice to consider decompressive craniectomy in patients with midline shift that significantly exceeds the hematoma thickness suggesting a greater level of associated brain injury and swelling. A ventriculostomy is placed intraoperatively if the patient's preoperative GCS is less than 8 or if significant swelling is noted at the time of operation. Because brain shift is common in subdural hematoma and external landmarks can be difficult to palpate during surgery, ventriculostomy can be quite challenging in this setting. In addition to this, patients can also become coagulopathic in this setting making multiple passes less desirable. In this setting, consideration should be given to intraparenchymal ICP monitor placement if unable to successfully place a ventriculostomy.

4. Focal traumatic parenchymal lesions

Intraparenchymal hemorrhages, contusions or infarcts are associated with severe traumatic brain injury in up to 35% of cases, but only 20% of trauma craniotomy is undertaken for their removal [7,24-26]. These lesions occur most commonly in the frontal or temporal lobes due to the brain impacting against the frontal bone and sphenoid ridges, whereas parietal and occipital lobe hematoma is most often secondary to direct impact [7]. Parenchymal lesions tend to evolve over time and the resulting mass effect from larger lesions may lead to worsened secondary brain injury, neurological deterioration, herniation and death [27]. In addition to lesion blossoming, delayed traumatic intracerebral hematoma (DTICH), which occurs in areas of radiographically normal brain on initial CT scan, may lead to delayed neurological deterioration [24,28]. It is important that any patient with abnormal findings on initial CT scan be monitored closely for the aforementioned phenomena, which may develop subsequent to initial physical and radiographic examination. Much work has gone into prognosticating which patient and lesion characteristics may be prone to worse outcome. Risk factors for worse outcome include, but are not limited to increased age, lower GCS on admission, presence of skull fracture, absence of brainstem reflexes, status of basal cisterns on CT, ICH volume, severity of surrounding edema, preoperative neurological deterioration, and concomitant SDH[24]. Nonfocal lesions, specifically diffuse injury with intractable intracranial hypertension will be discussed in the next section with indications, timing and method for surgical management of focal parenchymal lesions reviewed now.

Guideline based indications for surgery[24]: Progressive neurological deterioration referable to the lesion, medically refractory intracranial hypertension (see section titled *Intractable Intracranial Hypertension* below for discussion) or signs of mass effect on CT should be considered for surgical evacuation. Also considered for surgical evacuation are patients with GCS 6-8 with frontal or temporal contusions greater than 20cm^3 in volume with at least 5mm of midline shift and/or cisternal compression on CT scan, as well as, patients with any lesion greater than 50 cm^3 in size. These indications have generally been derived by review of several

studies which have focused on defining patient and lesion characteristics at high risk for subsequent neurological deterioration and assume that earlier operative intervention will improve likelihood for a more favorable outcome [12,24,29-31]. Candidates for nonoperative management with intensive monitoring and serial imaging, include those with lesions without significant mass effect on CT scan, no evidence for neurological compromise, and without intracranial hypertension. Such lesions are common and generally resorb in 4 to 6 weeks by macrophage phagocytosis and gliosis [7].

Timing and surgical technique [24]: For patients with focal lesions and the indications mentioned above, craniotomy with evacuation of the mass lesion as soon as possible is recommended. While stereotactic evacuation of focal posttraumatic lesions has been reported, we do not advocate this method as the majority of these patients also have a degree of diffuse injury with associated widespread secondary injury and a degree of cerebral edema with intracranial hypertension [24,25,32].

5. Intractable intracranial hypertension

Cerebral edema and subsequent intracranial hypertension are a major concern in combating the secondary injury of severe TBI [24,33]. At least 80% of severe TBI patients have elevated ICP and this is the major cause of death in those who die [7]. Intracranial pressure monitoring is currently recommended by clinical practice guidelines for patients with severe traumatic brain injury who have an abnormal CT scan of the head or those with a normal CT scan who meet other specified criteria [34]. The level II recommendation for treatment is for those with sustained intracranial pressure greater than 20mmHg [35]. Currently recommended nonoperative therapies for intracranial hypertension include head of bed elevation, hyperosmolar therapy (mannitol and/or hypertonic saline), intubation to ensure normocarbia with only short periods of hyperventilation as a temporizing measure if needed, analgesia, neuromuscular paralysis, ventricular drainage, hypothermia, and barbiturate or propofol induced burst suppression [7].

The role for surgery in the treatment of medically refractory intracranial hypertension has become the matter of intense debate in recent years. A variety of surgical procedures have been used for treatment of refractory intracranial hypertension, without a prominent mass lesion, including subtemporal decompression, temporal lobectomy, and circumferential craniotomy [24]. The two most utilized surgical procedures in this setting include the hemispheric decompressive craniectomy and the bifrontal decompressive craniectomy; the latter originally described by Kjellberg et al [24,36]. The 2006 "Guidelines for the Surgical Management of Traumatic Brain Injury" support bifrontal decompressive craniectomy within 48 hours of injury as a treatment option for patients with diffuse, medically refractory posttraumatic cerebral edema and resultant intracranial hypertension [24]. This is based on data associating intracranial hypertension with poor outcome and multiple studies showing that decompressive craniectomy can reliably manage intracranial hypertension [37-40]. The aforementioned data was noted to be less than ideal as it lacked data from prospective, randomized trials [24].

For example, particular attention was paid to the work of Polin et al, which retrospectively evaluated outcome in 35 patients undergoing bifrontal decompressive craniectomy for refractory posttraumatic cerebral edema, matched for age, admission GCS, sex, and maximal ICP with historical controls selected from the Traumatic Coma Data Bank [24,40]. Pertinent findings included favorable outcome association for surgery when performed less than 48 hours after injury compared to surgery performed longer than 48 hours after injury, especially in patients whose ICP had not yet been sustained above 40mmHg [40]. Also, medical management alone carried a 3.8 times relative risk of unfavorable outcome compared with decompressive craniectomy [40]. While this work and others argued in favor of bifrontal decompressive craniectomy as the potential intervention of choice in the proper patient, guideline authors also noted the lack of contemporaneous controls, and called for prospective, controlled trials to meaningfully compare outcome between surgical and nonsurgical groups in this clinical setting [24].

It was this goal that the DECRA (Decompressive Craniectomy in Diffuse Traumatic Brain Injury) trial was undertaken [9]. DECRA was a multicenter, randomized controlled trial conducted in 15 hospitals in Australia, New Zealand and Saudi Arabia designed to test the efficacy of bifrontotemporoparietal decompressive craniectomy in adults below 60 years of age with severe TBI in whom first-tier therapeutic measures failed to control ICP above 20 mmHg per Brain Trauma Foundation guidelines recommendation [9]. Randomization to either early decompressive craniectomy or standard medical management was undertaken for patients with an initial GCS <8 and when ICP was > 20 mmHg for > 15 minutes. At 6 months follow-up, 70% of patients in the craniectomy group had an unfavorable outcome vs. 51% of patients in the standard care group (odds ratio 2.21 [95% CI 1.14-4.26]; P = 0.002) [9]. This was despite the findings that decompressive craniectomy was associated with decreased intracranial pressure and shorter ICU stays [9]. Based on these results, the authors concluded that decompressive craniectomy was associated with more unfavorable outcomes and that the Australian health care system would save tens of millions of dollars by adhering to a medically-based treatment strategy rather than aggressive surgical decompression, and they predicted the trial would significantly alter clinical practice [38].

Many neurosurgeons and intensivists certainly did not share the aforementioned opinions of the DECRA trial authors, which provoked strong emotional reactions and has brought the methods and results of the trial under heavy scrutiny to explain the confounding lack of positive results [41-44]. Perhaps the most compelling criticism pertains to the baseline characteristics of the patients in each of the study's groups. The number of patients with bilateral unreactive pupils in the surgical group was nearly double that in the control group (27% vs. 12%) [9]. After controlling for this, the differences in outcomes became non-significant [9]. In addition to pupillary differences, radiologic findings were more severe in the surgical group (77% vs. 67% with Marshall grade III injury) and GCS scale was lower (5 vs. 6) in the surgical group; All of the aforementioned factors have prognostic significance [41]. The issue of patient crossover has also been noted as a potentially confounding factor in this trial [45,46]. A total of 19 patients (4 < 72 hours after randomization and 15 > 72 hours after randomization) in the standard care group had a decompressive craniectomy as a life saving procedure, which

some involved in the trial believe may have eliminated equipoise for the involved neurosurgeons who likely felt that these patients had genuinely increased ICP [41,46]. These 19 patients were analyzed in the standard care group as there being an intention treat [45].

In addition to the baseline differences among groups in the DECRA trial, other issues with the trial have been raised. DECRA excluded patients with any traumatic mass lesions, greatly reducing the number of real-world patients the data may be applicable to [42]. Secondly, some feel that the procedure used may not be as efficacious as that originally described by Polin et al, as the DECRA procedure did not involve division of the sagittal sinus and falx cerebri [40, 42]. Furthermore, many believe the threshold ICP elevation for inclusion in the study (spontaneous increase in ICP > 20mmHg for > 15 min, continuous or intermittently, within a 1-hr period), may have been too low and that many neurosurgeons and intensivists would not normally consider decompressive craniectomy in patients who have an intracranial pressure around 20mmHg for such a short time [42,43,47]. Indeed, the median ICP for both groups during the 12 hours before randomization was 20mmHg, which is the upper limit of normal [9,42]. Based on many of the aforementioned points, the Section on Neurotrauma and Critical Care of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons state "that no conclusions regarding management of the use of decompressive craniotomy in patients with traumatic brain injury should be drawn from this trial, and clinical practice should not be changed on the basis of these results [42]."

Despite its many criticisms, DECRA does represent the first randomized clinical trial of decompression to be completed in adult neurotrauma patients, and may "have placed us on the first rung of the evidence-based scientific ladder [41]." The protocol for the RESCUEicp (Randomized Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intracranial Pressure) differs from that of the DECRA trial in terms of intracranial pressure threshold (> 25mmHg > 1-12 hours), decompressive craniectomy techniques allowed, timing of surgery (any time after injury vs. within 72 hours), acceptance of contusions and duration of follow-up (2 years) [48]. At the time of this writing, recruitment is near completion with anxiously awaited results, which may shed more light on the heavily debated topic of where the role of decompressive craniectomy lies in treatment of diffuse traumatic brain injury. In the meantime, decompressive craniectomy will likely continue to be used by many for refractory intracranial hypertension. Furthermore, the available data highlights the need for novel ways of treating patients with TBI, whether with neuroprotective agents or regenerative therapeutics, in addition to improved prevention initiatives [49].

6. Traumatic posterior fossa mass lesions

Compared with the aforementioned traumatic brain injuries, traumatic posterior fossa mass lesions are rare. In a recent retrospective review of 4315 patients of hospitalized TBI patients, only 41 (1%) were noted to have posterior fossa hematomas [50]. In these 41 patients, there were 18 patients with posterior fossa EDH, 10 with SDH, and 17 with intracerebellar hematoma [50]. EDH is the most common posterior fossa lesion reguiring surgery, followed by SDH and

intracerebellar hemorrhage [7]. Though rare when compared with the incidence of supratentorial traumatic lesions, timely recognition and evacuation of surgical lesions is of the utmost importance for the patient [51]. Those caring for these patients must keep in mind that because of the limited volume of the posterior fossa and proximity of the neighboring brainstem, rapidly fatal deterioration can occur from obstructive hydrocephalus and brainstem compression from an expanding hematoma. Respiratory pattern changes and sudden increases in blood pressure may be a harbinger of impending crisis, while pupillary reflexes, ICP measurements, or altered sensorium are not reliable clues to impending herniation in this region [7]. With regards to data supporting surgery in this patient population, we must keep in mind as stated in the 2006 guidelines, "…surgery is generally viewed as required therapy in symptomatic patients with progressive dysfunction. Because of the potential adverse consequences of withholding or delaying surgery for such patients, studies depend on retrospective analyses. As a result, there is no Class I or Class II evidence to support recommendations for the surgical management of these injuries [51]."

Guideline based indications for surgery [51]: Patients with mass effect on computed tomographic (CT) scan or with neurological dysfunction or deterioration referable to the lesion should undergo operative intervention. Mass effect on CT scan is defined as distortion, dislocation, or obliteration of the fourth ventricle; compression or loss of visualization of the basal cisterns, or the presence of obstructive hydrocephalus. Management by close observation and serial imaging may be appropriate for patients with lesions with no significant mass effect on CT scan and without signs of neurologic dysfunction.

Timing [51]: In indicated cases, surgical evacuation should be performed as soon as possible because these patients can deteriorate rapidly, thus, worsening their prognosis.

Surgical technique: Suboccipital craniectomy is the predominant method for evacuation of posterior fossa mass lesions [51]. Generally, a ventriculostomy catheter should be placed before surgery for the purposes of CSF drainage and ICP reduction [7]. An important caveat to ventriculostomy in the setting of posterior fossa lesions is that many advocate slow CSF drainage to avoid the rare possibility of upward herniation [7,52]. Because optimal surgical positioning involves anterior flexion of the cervical spine, absence of cervical spine fracture must also be assured and documented. Careful attention must be paid to boney removal over the venous sinuses, which can be a major source of bleeding complicating surgery in this region, and should be prepared for [7]. With injuries that involve the subdural spaces and cerebellar parenchyma, a larger decompressive craniectomy including the rim of the foramen magnum inferior, up to the edge of the transverse sinus superiorly and laterally as far as the digastric groove should be undertaken to provide adequate decompression [7]. For lesions extending inferiorly with concomitant compression, the posterior arch of the atlas can also be removed [7].

7. Depressed cranial fractures

Depressed cranial fractures complicate up to 6% of head injuries in one series [53,54]. Compound depressed cranial fractures are depressed fractures with an overlying scalp laceration in continuity with the fracture site and with galeal disruption, while simple depressed cranial fractures have no galeal disruption. Besides sequalae from an associated hematoma with mass effect, the primary clinical concern for depressed skull fractures involve their association with infection and late seizure [54]. Depressed skull fractures overlying major venous sinuses are generally managed nonoperatively due to high associated risks of surgery, but have also been reported to be associated with delayed onset of intracranial hypertension [55]. As is common with the other injury types discussed previously, there is a lack of Class I literature evaluating indications, timing and surgical techniques which provide the best outcomes for these patients [54]. For all open cranial fractures, prophylactic antibiotics, specifically cefazolin or pipercillin/ tazobactam for 5-7 days is generally recommended [56].

Guideline based indications for surgery [54]: Open (compound) cranial fractures depressed greater than the thickness of the cranium should undergo operative intervention to prevent infection. Open (compound) depressed cranial fractures may be treated nonoperatively if there is no clinical or radiographic evidence of dural penetration, significant intracranial hematoma, depression greater than 1 cm, frontal sinus involvement, gross cosmetic deformity, wound infection, pneumocephalus, or gross wound contamination. Nonoperative management of closed (simple) depressed cranial fractures is a treatment option.

With regards to frontal air sinus fractures, closed fractures, which only involve the posterior wall of the sinus, do not generally require surgical repair beyond scalp closure [7]. Compound frontal sinus fractures, which involve both anterior and posterior walls of the sinus, should be considered for surgical exploration and repair due to risk of delayed infection and/or CSF leak [7,57].

Timing [54]: Early operation is recommended to reduce the incidence of infection.

Surgical technique [54]: Elevation and debridement is recommended as the surgical method of choice. Primary bone fragment replacement is a surgical option in the absence of wound infection at the time surgery. All management strategies for open (compound) depressed fractures should include antibiotics.

8. Conclusions

Optimal outcome in severe TBI requires a coordinated effort between neurosurgeon, intensivist, nusrsing and rehabilitation to provide both surgical and nonsurgical interventions. With regards to surgery, prompt recognition and evacuation of surgical hematomas is vital. While surgical indications for many lesion types are based on retrospective data, the reviewed guidelines provide us with a framework from which we can build on to optimize treatment. The recent publication of the DECRA trial and presumed completion of the RESCUEicp trial

provide hope that higher-level evidence may be gathered in this patient population. The role of decompressive craniectomy in intractable intracranial hypertension continues to evolve. The RESCUEicp study hopes to address the shortcomings of the DECRA study.

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