

Decompressive Craniectomy: An Update

Margaret H. Lauerman¹ · Deborah M. Stein¹

Published online: 20 October 2015
© Springer Science + Business Media New York 2015

Abstract Craniectomy is a valuable technique in the management of evacuable mass lesions and elevated intracranial pressure (ICP). This review will examine the indications for and benefits of craniectomy in patients with traumatic brain injury (TBI). Recent data have questioned the utility of surgical decompression in the management of refractory intracranial hypertension; however, decompressive craniectomy (DC) remains a common practice at many trauma centers. Recent data have also questioned the use of invasive ICP monitors. However, despite this study, ICP monitors are standard of care in most major centers. Further research is needed before abandoning decompression for refractory elevated ICP or invasive ICP monitoring. While most patients with TBI are managed non-operatively, many patients do require decompression for refractory elevated ICP or evacuable mass lesions. Current guidelines help direct patient selection for DC, and specific indications for DC vary by the type of intracranial hemorrhage. DC can be lifesaving in patients with severe or progressive intracranial hemorrhage.

Keywords Intracranial hemorrhage · Decompressive craniectomy · Craniectomy · Craniotomy · Traumatic brain injury · Elevated intracranial pressure

Introduction

Traumatic brain injury (TBI) is a common cause of preventable death, especially in the younger patient population. Craniectomy remains a mainstay of care in patients with evacuable mass lesions. Often the mass lesion is evacuated and the cranium is not replaced due to cerebral edema, similar to a damage control laparotomy in which the abdomen is “left open” to prevent the development of abdominal compartment syndrome (ACS). Decompressive craniectomy (DC) for malignant intracranial hypertension can be a lifesaving operation, and can prevent mortality in patients in whom medical therapy is ineffective, similar to a decompressive laparotomy for elevated intraabdominal pressure and ACS. This review will elucidate how craniotomy and DC are used in modern management of TBI, and discuss recent developments in their use.

Epidemiology of Traumatic Brain Injury

TBI occurs over a spectrum of injury severity, with TBI ranging from a mild concussion requiring only outpatient treatment to devastating intracranial hemorrhage (ICH) with subsequent mortality. While most patients with a TBI do not require hospital admission, approximately 250,000 patients are hospitalized annually, with 53,000 subsequent deaths [1, 2]. Long-term disability in patients with TBI is a significant problem in those who survive [3]. With the worldwide incidence of TBI increasing, the personal impact, resource utilization, and costs associated with TBI are noteworthy [4].

This article is part of the Topical Collection on *Trauma Surgery*.

✉ Deborah M. Stein
dstein@umm.edu

Margaret H. Lauerman
mlauerman@umm.edu

¹ Program in Trauma, University of Maryland School of Medicine, 22 South Greene St, Baltimore, MD 21201, USA

Overview of Traumatic Brain Injury Management

Management of TBI encompasses both medical and surgical interventions, and patients with severe TBI often undergo both medical and surgical management concurrently. Medical management of TBI focuses on maintaining intracranial pressure (ICP) below 20 mmHg while optimizing cerebral blood flow (CBF) [5]. ICP management has been shown to be associated with functional outcomes, and thus tight ICP control remains a mainstay of TBI care [6]. Hyperosmolar therapy, head of bed elevation, pain control, appropriate sedation, temperature control, and barbiturate comas are all utilized to maintain an appropriate ICP [7, 8]. Cerebrospinal fluid (CSF) can be drained to decrease ICP. CBF can be decreased to minimize intracranial contents, although care must be taken to maintain mean arterial pressure (MAP). ICP is also influenced by intraabdominal and intrathoracic pressure through multiple compartment syndrome (MCS). Treatment of ICP should not only focus on the intracranial contents, but on the body as a collection of interconnected compartments [9].

Invasive ICP monitoring is an important aspect of TBI management. ICP measurements are used to guide management of cerebral edema. Performance of surgical decompression is based off these ICP measurements in patients with intractable elevated ICP. The need for ICP monitoring in TBI has become a recently controversial topic, with literature published asserting that invasive ICP monitoring does not have a beneficial effect on functional outcomes or mortality [10]. However, this data conflict with previous literature which found a survival benefit with ICP monitoring [11], and ICP monitoring continues to be frequently used to guide TBI management in most major medical centers.

Decompressive Craniectomy

Many patients with severe TBI meet criteria for decompression on presentation, and thus undergo operative intervention. Those patients that do not meet criteria initially for craniotomy or craniectomy should be reevaluated with any changes in clinical exam, progression of ICH, or failure of medical ICP management. Medical management of ICP targets decreasing cerebral tissue edema, arterial blood volume, venous blood volume, and CSF volume given the fixed volume of the cerebral vault. Unlike other body cavities such as the abdominal cavity, the skull cannot expand to increase the cerebral vault volume when the brain swells or an ICH is present. When the brain swells to a size larger than the space available in the cranial vault, herniation ensues. Medical management may not be

sufficient for severe ICH, and elevated ICP may persist despite maximal medical management. Operative decompression may be indicated in these circumstances.

To perform a craniectomy for anterior and middle fossa pathology, the patient is placed either supine or lateral, depending on their cervical spine clearance. The patient's head is elevated to maximize venous outflow, as intraoperative brain swelling is a serious consideration. A curved incision is made from anterior to the tragus to beyond the anterior hairline and clips are applied to the cut scalp edges for hemostasis. The temporalis muscle is reflected with the scalp, and multiple burr holes are created in the skull to allow elevation of the skull off of the underlying dura (Fig. 1). The dura is then opened to expose the underlying ICH and brain, and the intracranial blood is evacuated (Fig. 2). A dural pouch can be created with a substitute material, and the scalp closed over the dural pouch [12]. In a craniotomy the bone flap is replaced, while in a craniectomy the bone flap is left off at the procedure completion.

In contrast to the craniectomy used for anterior compartment ICH, the operation of choice in posterior fossa ICH is a suboccipital decompression. Herniation upwards and downwards can both occur with posterior fossa bleeding. Compression of the brainstem, given its proximity to the posterior fossa, is another known complication of posterior fossa ICH. Patients who decompensate may have hydrocephalus with obstruction of the usual CSF circulation. Upwards herniation with CSF drainage must be guarded against, and it is recommended not to drain CSF until after decompression. Either a lateral or a medial suboccipital decompression can be performed, depending on the distribution of the ICH, with the latter option opening the foramen magnum and C1 arch as well [13].

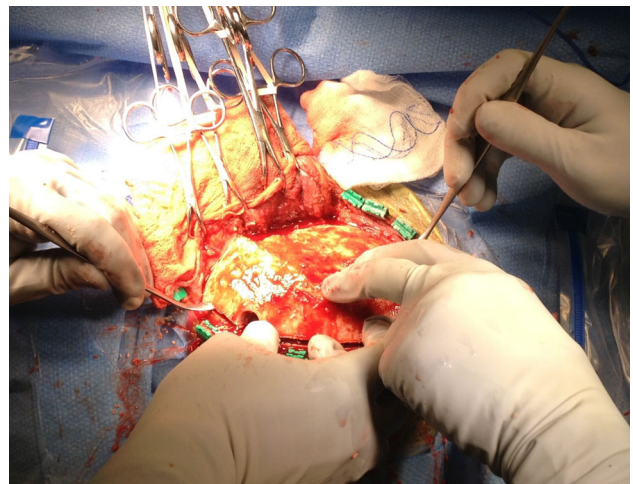


Fig. 1 Removing the flap of bone off of the underlying dura

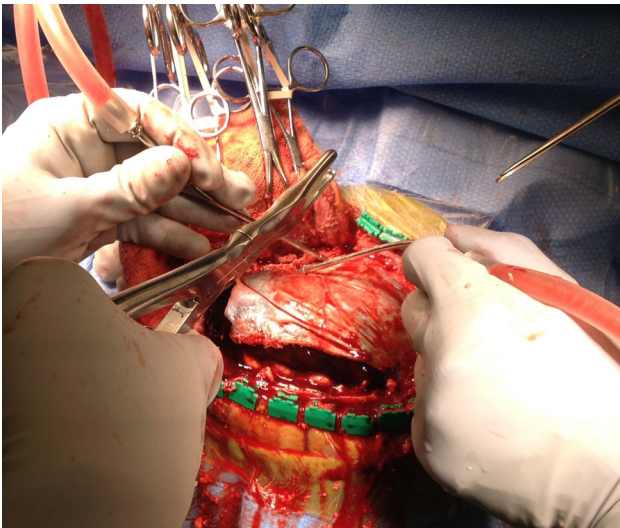


Fig. 2 Incising the dura to reveal the underlying brain and intracranial hemorrhage (ICH)

Surgical decompression has two mechanisms of benefit in TBI. Foremost, evacuable mass lesions are removed from the cranial vault during craniectomy, improving the space available into which the brain can swell. Second, at the completion of a craniectomy, the flap of skull can be left unattached, again creating increased volume into which the brain can swell similar in concept to leaving the abdomen open with a temporary abdominal closure to prevent ACS (Fig. 3). Both microvascular and macrovascular circulation improve after decompression of the cranial vault [14, 15]. Tissue oxygen delivery improves as well with improvement in CBF. In a study of patients with



Fig. 3 Computerized tomography (CT) image from a patient after a craniectomy for a subdural hematoma (SDH), with the bone flap absent from the right side of the skull

TBI, decreased tissue oxygen levels noted prior to decompression recovered after surgery [16]. Clinically, a decrease in ICP to below 20 mmHg is seen in 85 % of patients with elevated ICP, with a mean decrease in ICP of over 9 mmHg, after undergoing DC [17••].

When the bone flap is left off after decompression, care must be taken to not further traumatize the brain as the protective skull is no longer overlying. Care should be taken not to put pressure on that area where the craniectomy was performed, either through the patient's head resting on that side or with direct pressure. In patients who have recovered from their TBI, a specialized helmet should be used to protect their brain when they are more mobile and are out of bed. In those patients who survive, cranioplasty can be performed months later to reconstruct the cranial vault.

Craniectomy in the Anterior and Middle Fossae

Craniectomy in trauma is most often performed for evacuable mass lesions of the anterior and middle cranial fossae. Generally, evacuable mass lesions include subdural hematomas (SDH), epidural hematomas (EDH), and intraparenchymal bleeds. Subarachnoid hemorrhages (SAH) are not often treated with a craniectomy, as SAH is a more diffuse process. However, SAH can occur concurrently with cerebral edema, which may necessitate decompression if medical therapy is ineffective. ICH can occupy a significant volume of the cerebral vault, and can compress the underlying brain, leading to herniation in the most severe TBI cases.

Computerized tomography (CT) scanning is the best imaging modality for the initial diagnosis of TBI and for following ICH serially over time. Intravenous contrast is not required for initial TBI imaging, and intravenous contrast will actually obstruct visualization of the ICH. Magnetic resonance imaging (MRI) is not the primary test for ICH given the increased time required to perform the MRI and more limited accessibility when compared with CT.

Epidural Hematoma

Operative intervention in EDH depends on both radiologic imaging and clinical exam. On CT images, volume of EDH greater than 30 cc³ is an indication for evacuation. However, with an EDH volume less than 30 cc³, the decision to perform a decompression becomes more complex. If there is <5 mm of midline shift and the thickness of the EDH is <15 mm, the patient can be observed with serial CT imaging and serial clinical exams. However, to qualify for observation, the patient's Glasgow Coma Scale (GCS)

score must be greater than 8 and the patient cannot have focal neurologic deficits. Patients with EDH should be transferred to a center with neurosurgery capabilities in case they require operative intervention, as outcomes are related to the operative time delay after neurologic deficits appear [18]. Patients with EDH can rapidly decline given the arterial source of the bleeding, and should be closely monitored with frequent neurologic exams.

At our institution, our usual protocol for patients with TBI is to repeat their CT imaging at 6 and 24 h post-diagnosis. However, in patients with EDH that do not meet criteria for evacuation, we often rescan at 4 h following the initial diagnosis rather than the usual 6 h, and will often scan again at 12 h rather than waiting 24 h if there is any concern. This more frequent use of CT may help identify earlier any progression in the EDH and identify those who will fail non-operative management.

Subdural Hematoma

Operative indications in SDH (Fig. 4), much like in EDH, rely on both radiographic findings and the clinical exam; however, ICP monitoring plays a role in the decompression algorithm for SDH. Volume of the ICH is not a factor for SDH as it is for EDH. In patients with more than a 5 mm midline shift or with 10 mm of SDH depth, decompression should be performed irrespective of the patient's GCS. However, if the patient's GCS is decreased at 8 or lower, and the patient has lateralizing pupillary signs or a drop in GCS of at least 2 points, they should undergo craniotomy or craniectomy even if they do not meet the above radiographic criteria for evacuation. An ICP monitor should be placed for a GCS of 8 or lower in those not initially undergoing surgical evacuation [19] and craniotomy undertaken if the ICP remains elevated.

Multiple techniques have been described for evacuation of SDHs, including subdural drain placement alone, Burr hole creation, craniotomy, and craniectomy. However, much of the literature on ICH does not investigate which operation is the most efficacious surgical intervention, and may not even delineate the operation used, making it difficult to draw conclusions [19]. In the absence of high quality trials comparing surgical techniques in SDH, our institution utilizes both Burr holes and craniotomy or craniectomy for SDH depending on the patient's clinical status, as well as the anatomy and chronicity of the SDH.

Intraparenchymal Bleeding

Indications for surgical evacuation in patients with intraparenchymal bleeding are different from the indications with SHD and EDH. In patients with intraparenchymal bleeding, those without neurologic exam abnormalities, without

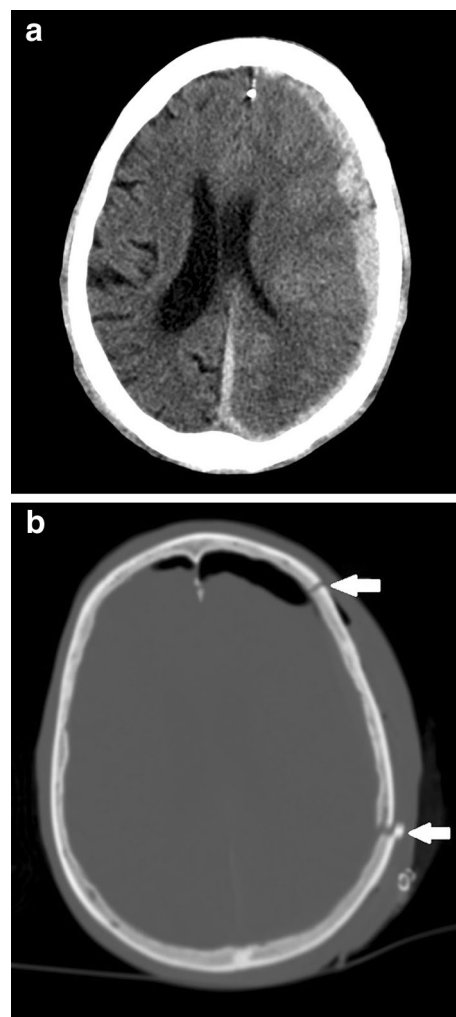


Fig. 4 CT images from a large subdural hematoma (SDH) with midline shift (a) and post-operative changes of a craniotomy with replacement of the bone flap on the left side of the skull (b). Margins of the craniotomy bone flap are delineated by the *white arrows*

compression on CT, and with non-elevated ICP can be observed. If any of these criteria are not met, the patient should undergo evacuation. Decompression may be indicated based on CT findings as well, as patients with intraparenchymal bleeding with a volume greater than 50 cc³ should undergo evacuation. Patients with intraparenchymal bleeding with a volume greater than 20 cc³, in conjunction with over 5 mm of midline shift or cistern compression on the CT, should undergo evacuation if the patient's GCS is <8 [20].

Craniotomy in the Posterior Fossa

Evacuation for posterior fossa hemorrhage is a more complicated proposition than for anterior lesions, for which a standard trauma DC can be performed. Clinically, posterior fossa ICH also brings certain unique difficulties when

compared with the anterior and middle fossae ICH. Posterior fossa ICH, given the small space of the posterior fossa compared to the anterior/middle fossae, can produce herniation with a smaller volume ICH. Additionally, measurement of posterior fossa pressure may be difficult as ICP monitors are placed into the ventricle in the anterior/middle fossae.

Unlike in SDH, EDH, and intraparenchymal bleeding where ICH size on CT plays a significant role in determining the need for DC, size of posterior fossa ICH is not a consideration in the evaluation for evacuation. After a posterior fossa bleed is diagnosed, direct, or indirect radiographic signs of mass effect are evaluated. These signs of mass effect include: compression of the basal cisterns, compression of the 4th ventricle, or hydrocephalus. Mass effect on CT imaging, with any GCS on clinical exam, is an indication for decompression. Patients who have a neurologic deficit, regardless of the presence or absence of mass effect on CT, should undergo evacuation. Patients without any neurologic abnormality or CT evidence of mass effect can be observed [21].

Use of DC for Intractable Intracranial Hypertension

Use of surgical decompression for elevated ICP refractory to medical management is a recently controversial topic. DC is a common practice in most high-volume trauma centers for patients with intractable elevated ICP despite maximal medical therapy, as good functional outcomes in a significant number of patients undergoing DC for refractory intracranial hypertension have been reported [17••]. However, the recently published DECRA trial reported worse mortality for patients undergoing DC for elevated ICP, and has brought the use of decompression into question for this specific diagnosis [22••]. Given the significant limitations of the DECRA trial, including the use of a bifrontal surgical approach, which is not the standard approach to a trauma decompression, as well as differences in lateralizing signs between the craniectomy and non-craniectomy groups, most major institutions continue to utilize surgical DC for refractory ICP until more definitive and generalizable data are published.

At our institution, we use DC for refractory intracranial hypertension in lieu of pentobarbital coma. DC for refractory ICP is used when patient positioning, hyperosmolar therapy, sedation, pain control, temperature control, blood pressure control, and ICP drainage have been maximized with a persistent ICP of >20 mmHg. When DC is used for intractable intracranial hypertension, the bone flap is left off at the completion of the decompression to allow the brain to swell outside the previous volume of the skull.

However, ICP is not just influenced by the intracranial contents, as the body is a series of interconnected compartments. Both intrathoracic pressure and intraabdominal pressure can elevate ICP. Care must be taken when selecting ventilator settings to limit pressure as possible. Intraabdominal pressure should be monitored, with decompressive laparotomy in patients with concurrent elevated ICP and intraabdominal hypertension [9].

Craniectomy Versus Craniotomy

At the completion of the decompression, a decision must be made about whether to replace the bone flap in a decompressive craniotomy, or to leave the bone flap off in a DC. Replacing the bone flap recreates the cranial vault, and makes the volume of the cranial vault the same as before the decompression. When the bone flap is replaced, there is no extra room for the brain to swell. In patients with intractable ICP, a craniectomy is performed allow the brain maximal room to swell. If the decompression is performed for an evacuable mass lesion, such as a SDH or an EDH, the decision to replace the bone flap is based on the amount of brain swelling seen in the operating room. If there is a large amount of swelling, then the bone flap is left off. Otherwise, if there is not significant brain swelling, the bone flap is replaced. When a craniectomy is performed, patients require a subsequent cranioplasty, which has its own complications [23•]. Craniectomy is thus avoided for evacuable mass lesions if possible.

Outcomes

Complications after DC are frequent, with an overall rate of 13.4 %. ICH is a common complication, with a rate of 12 %, highlighting the importance of a post-decompression CT scan. These ICHs can be at the operative site or remote to the operative site, and can require reintervention. Infectious complications occur in 6.9 %, and care is taken to preserve the superficial temporal artery to optimize wound healing. Infectious complications can also include abscess, empyema, meningitis, and ventriculitis. CSF flow can also be altered, and hydrocephalus, hygroma, or CSF leak occurs in 18 % [23•].

Individual outcomes can be excellent after DC for evacuable mass lesions, especially when considering the critical state of many patients with ICH. The rate of a good functional recovery is 35 % after decompression for ICH. While the mortality rate overall is 40 % in those undergoing decompression, and the rate of severe disability or vegetative state is 20 %, a significant percentage of patients achieve a good functional outcome [24].

At our institution, we have also seen excellent clinical results after DC for refractory ICP. The rate of a Glasgow outcome score (GOS) of 4–5 is 40 % in patients undergoing DC for elevated ICP. Good functional outcomes even occurred in patients with an initial post-resuscitation GCS ranging from 3 to 5. However, similarly to patients undergoing DC for evacuable mass lesions, there is a significant rate of mortality and severe disability after DC; the mortality rate is 28 %, and the rate of severe disability or vegetative state 32 % [17••]. The recent DECRA trial has brought the use of DC into question in elevated ICP, noting a worse GOS for DC patients compared with patients not undergoing DC. There was a similar mortality between DC and non-DC patients in the DECRA trial, and future research will delineate the optimal therapy for intractable elevated ICP [22••].

Craniectomy is avoided if possible given the significant complication rate associated with cranioplasty. ICH occurs in 3.6 %. Infectious complications occur in 6.0 %, and include both the superficial and deep complications seen with DC. CSF alterations occur in 5.4 % of cranioplasty patients. A unique complication to cranioplasty is bone resorption, which occurs in 9.5 % of patients [23•]. Bone flap resorption can require revision cranioplasty with a synthetic implant [25].

Post-Operative Care

Patients who undergo DC often have an ICP monitor placed intraoperatively depending on clinical exam and risk of cerebral edema. Despite removal of mass lesions and craniectomy, ICP can still remain elevated post-operatively. Further medical management of elevated ICP may be required after DC. In our institution, immediate post-operative CT scans are obtained after DC to establish a post-operative baseline and to evaluate for any delayed ICH or reaccumulation of the initial ICH. Persistent cerebral edema and intracranial hypertension can still remain significant problems after DC, and this cerebral edema should be treated according to the algorithms described above. Following craniectomy, at our institution, we usually allow for ICP up to 25 mmHg. Any significant change in neurologic exam should similarly prompt repeat CT imaging to evaluate for delayed ICH, as continued bleeding and further cerebral edema are always risks. Meningitis should be considered with any subsequent change in neurologic exam or systemic signs of infection. Hygromas and hydrocephalus are complications which present further in the hospital course [13]. Cranioplasty should also be considered in those patients who have undergone craniectomy.

Conclusions

Craniectomy is a valuable and potentially lifesaving operation for patients with evacuable mass lesions given the fixed volume of the skull, and may stave off herniation. Decompression improves both CBF and tissue oxygenation. Both anterior/middle fossae ICH and posterior fossa ICH can undergo DC. EDH, SDH, and intraparenchymal bleeding have separate indications for craniectomy utilization, incorporating CT, and neurologic exam findings. Whether craniectomy is done for evacuation of a mass lesion or for pure decompression, surgical therapy should be used in conjunction with medical management to optimize outcomes. Post-operative care must guard against a variety of early and late potential complications, cerebral edema, cerebral hypoperfusion, and further ICH. Future research should help to delineate the benefit of surgical decompression in ICP refractory to medical therapy, although currently most institutions continue to utilize DC for this indication.

Compliance with Ethics Guidelines

Conflict of Interest Drs. Lauerman and Stein declare no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. Faul M, Xu L, Wald M, Coronado V. Traumatic brain injury in the United States: emergency department visits, hospitalizations, and deaths, 2002–2006. 2010. http://www.cdc.gov/traumaticbraininjury/pdf/blue_book.pdf. Accessed 9 Mar 2015.
2. Coronado V, Xu L, Basavaraju S, McGuire L, Wald M, Faul M, et al. Surveillance for traumatic brain injury: related deaths—United States, 1997–2007. 2011. http://www.cdc.gov/mmwr/preview/mmwrhtml/ss6005a1.htm?s_cid=ss6005a1_w. Accessed 9 Mar 2015.
3. Juul N, Morris GF, Marshall SB, Marshall LF. Intracranial hypertension and cerebral perfusion pressure: influence on neurological deterioration and outcome in severe head injury. The Executive Committee of the International Selfotel Trial. *J Neurosurg*. 2000;92(1):1–6.
4. Maas AI, Stocchetti N, Bullock R. Moderate and severe traumatic brain injury in adults. *Lancet Neurol*. 2008;7(8):728–41.
5. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care, AANS/CNS, Bratton SL,

- Chestnut RM, et al. Guidelines for the management of severe traumatic brain injury VIII. Intracranial pressure thresholds. *J Neurotrauma*. 2007;24(1):S55–8.
6. Narayan RK, Greenberg RP, Miller JD, Enas GG, Choi SC, Kishore PR, et al. Improved confidence of outcome prediction in severe head injury. A comparative analysis of the clinical examination, multimodality evoked potentials, CT scanning, and intracranial pressure. *J Neurosurg*. 1981;54(6):751–62.
 7. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care, AANS/CNS, Bratton SL, Chestnut RM, et al. Guidelines for the management of severe traumatic brain injury II. Hyperosmolar therapy. *J Neurotrauma*. 2007;24(1):S14–20.
 8. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care, AANS/CNS, Bratton SL, Chestnut RM, et al. Guidelines for the management of severe traumatic brain injury. XI. Anesthetics, analgesics, and sedatives. *J Neurotrauma*. 2007;24(1):S71–6.
 9. Lauerma MH, Stein DM. Multicompartment management of patients with severe traumatic brain injury. *Curr Opin Anaesthesiol*. 2014;27(2):219–24.
 10. • Chestnut RM, Temkin N, Carney N, Dikmen S, Rondina C, Videtta W, et al. A trial of intracranial-pressure monitoring in traumatic brain injury. *N Engl J Med*. 2012;367(26):2471–81. *This article describes a trial examining outcomes with ICP monitor use, which did not show an improved mortality with invasive ICP monitor use.*
 11. Farahvar A, Gerber LM, Chiu YL, Carney N, Hartl R, Ghajar J. Increased mortality in patients with severe traumatic brain injury treated without intracranial pressure monitoring. *J Neurosurg*. 2012;117(4):729–34.
 12. Jallo J, Narayan R. General principals of craniocerebral trauma and traumatic hematomas. In: Sekhar L, Fessler R, editors. *Atlas of neurosurgical techniques: brain*. New York: Thieme Publishers; 2011. p. 899–901.
 13. Imhof H, Lenzlinger P. Traumatic brain injury. In: Oestern H, Trentz O, Uranues S, editors. *Head, thoracic, abdominal and vascular injuries*. Heidelberg: Springer Science & Business Media; 2011. p. 1–92.
 14. Heppner P, Ellegala DB, Durieux M, Jane JAS, Lindner JR. Contrast ultrasonographic assessment of cerebral perfusion in patients undergoing decompressive craniectomy for traumatic brain injury. *J Neurosurg*. 2006;104(5):738–45.
 15. Bor-Seng-Shu E, Hirsch R, Teixeira MJ, De Andrade AF, Marino R Jr. Cerebral hemodynamic changes gauged by transcranial Doppler ultrasonography in patients with posttraumatic brain swelling treated by surgical decompression. *J Neurosurg*. 2006;104(1):93–100.
 16. Stiefel MF, Heuer GG, Smith MJ, Bloom S, Maloney-Wilensky E, Gracias VH, et al. Cerebral oxygenation following decompressive hemicraniectomy for the treatment of refractory intracranial hypertension. *J Neurosurg*. 2004;101(2):241–7.
 17. •• Aarabi B, Hesdorffer DC, Ahn ES, Aresco C, Scalea TM, Eisenberg HM. Outcome following decompressive craniectomy for malignant swelling due to severe head injury. *J Neurosurg*. 2006;104(4):469–79. *This article describes a single institution study with many good functional outcomes after decompressive craniectomy.*
 18. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, et al. Surgical management of acute epidural hematomas. *Neurosurgery*. 2006;58(3):S7–15 (discussion Si–iv).
 19. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, et al. Surgical management of acute subdural hematomas. *Neurosurgery*. 2006;58(3):S16–24 (discussion Si–iv).
 20. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, et al. Surgical management of traumatic parenchymal lesions. *Neurosurgery*. 2006;58(3):S25–46 (discussion Si–iv).
 21. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, et al. Surgical management of posterior fossa mass lesions. *Neurosurgery*. 2006;58(3):S47–55 (discussion Si–iv).
 22. •• Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D’Urso P, et al. Decompressive craniectomy in diffuse traumatic brain injury. *N Engl J Med*. 2011;364(16):1493–502. *This article describes a randomized trial for DC with medically refractory ICP, showing decreased post-operative GOS scores in the DC patients.*
 23. • Kurland DB, Khaladj-Ghom A, Stokum JA, Carusillo B, Karimiy JK, Gerzanich V, et al. Complications associated with decompressive craniectomy: a systematic review. *Neurocrit Care* 2015;23(2):292–304. *Excellent review covering complications after DC and cranioplasty.*
 24. Aarabi B, Hesdorffer DC, Simard JM, Ahn ES, Aresco C, Eisenberg HM, et al. Comparative study of decompressive craniectomy after mass lesion evacuation in severe head injury. *Neurosurgery*. 2009;64(5):927–39 (discussion 939–940).
 25. Brommeland T, Rydning PN, Pripp AH, Helseth E. Cranioplasty complications and risk factors associated with bone flap resorption. *Scand J Trauma Resusc Emerg Med*. 2015;23(1):75.