Surgical Treatment of Elevated Intracranial Pressure

Decompressive Craniectomy and Intracranial Pressure Monitoring

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KEYWORDS

• Elevated intracranial pressure • Decompressive craniectomy • Intraventricular catheter
• Intracranial pressure monitoring • Intracranial mass • Intracranial hypertension hemicraniectomy

KEY POINTS

- Elevated intracranial pressure (ICP) is a relatively common complication of severe traumatic brain injury (TBI), intracranial hemorrhage, malignant middle cerebral artery (MCA) infarction, and high-grade subarachnoid hemorrhage (SAH). It is associated with high mortality and morbidity.
- Surgical interventions such as (1) insertion of intraventricular catheter, (2) removal of a space-occupying lesion, and (3) decompressive craniectomy can be life-saving in cases of refractory elevated ICP.
- Indications for ICP monitoring are summarized herein.
- Level I evidence obtained from 3 major randomized controlled trials (RCTs) (DECIMAL, DESTINY, and HAMLET) that supports decompressive craniectomy in young patients ($\leq$60 years) with malignant MCA infarction is currently available.
- Recommendations for decompressive craniectomy in patients with severe TBI are currently being clarified. Data from the DECRA trial have shown increased unfavorable outcome in association with decompressive craniectomy. The RESCUEicp is an ongoing trial currently assessing the outcome of decompressive craniectomy in this setting.
- Recommendations for decompressive craniectomy in the setting of aneurysmal SAH are less clear. No RCTs are currently available.
- Although protocols are important, an individualized approach to all patients is crucial in the management of elevated ICP. It is important to balance the risks and potential outcomes of elevated ICP against those associated with any surgical intervention. Future RCTs should focus on functional outcome and quality of life of patients after surgery.

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INTRODUCTION

Elevated intracranial pressure (ICP) is a relatively common and potentially devastating complication of a variety of cerebral pathologic conditions.[1] It occurs mainly as a result of large ischemic or hemorrhagic stroke, or following severe traumatic brain injury (TBI).[2,3] Current advances in technology, including modern neuroimaging and neuromonitoring techniques, allow early anticipation and detection of elevated ICP and hence more expeditious initiation of therapy.[4] First-line treatment for patients presenting with elevated ICP mainly involves noninvasive measures such as midline positioning and elevation of the head, hyperosmolar therapy, and, occasionally, hyperventilation and sedation in intubated patients.[5] In certain instances, however, the clinical impact of the primary or secondary brain injury can be severe, and patients may not respond well to conventional medical approaches. In such cases, more invasive techniques need to be implemented urgently to help immediately reduce elevated ICP. These techniques include (1) insertion of intraventricular catheter and cerebrospinal fluid (CSF) drainage, (2) removal of an intracranial space-occupying lesion (eg, hematoma), and (3) undergoing a decompressive craniectomy.[6] This review discusses the role of surgery in the management of elevated ICP, with special focus on the placement of intraventricular catheter and decompressive craniectomy. The authors describe the techniques and potential complications of each procedure, and review the existing evidence regarding the impact of these procedures on patient outcome. Surgical management of mass lesions and ischemic or hemorrhagic stroke occurring in the posterior fossa is not discussed herein.

NORMAL CEREBRAL DYNAMICS (MONRO-KELLIE DOCTRINE)

In a normal adult, the cranial vault can accommodate an average volume of approximately 1500 mL.[7] This volume is distributed among intracranial components as follows: brain tissue (≈88%), blood (≈7.5%), and CSF (≈4.5%).[4,7] The cranium itself, however, is a closed container inside which the pressure depends on the sum of volumes of these components ($V_{Intracranial\ space} = V_{Brain} + V_{Blood} + V_{CSF}$). The normal ICP ranges between 10 and 15 mm Hg in an adult. For a normal ICP to be maintained, any increase in the volume of one of the intracranial components should be met with an equivalent decrease in one or both other components. This phenomenon is known as the Monro-Kellie doctrine.[8] Accordingly, in cases of severe cerebral edema, brain swelling can lead to an uncompensated increase in the intracranial volume, resulting in elevated ICP and potential subsequent brain herniation. Elevated ICP can also lead to a decrease in cerebral blood flow (CBF) and a subsequent decrease in cerebral perfusion pressure (CPP).[9] In turn this can result in diffuse cerebral ischemia, which is one of the most important causes of secondary brain injury.[9]

CAUSES OF ELEVATED ICP

Based on the Monro-Kellie doctrine, any abnormality that leads to an uncompensated increase in intracranial volume can cause elevated ICP. One common cause of elevated ICP is severe TBI, which can result in primary or secondary brain edema.[10] Approximately 40% of patients with severe TBI develop brain edema and subsequent elevated ICP.[11] Other contributors to elevated ICP after severe TBI include: hyperemia (loss of vasomotor autoregulation); intracranial bleeding (eg, epidural hematoma, subdural hematoma, and intraparenchymal hemorrhage); hydrocephalus (eg, intraventricular hemorrhage and obstruction of CSF flow); and/or posttraumatic seizures (status epilepticus).[5] Large ischemic stroke, namely malignant middle cerebral artery (MCA) infarction, is another common cause of elevated ICP.[3,12] This type of stroke can lead to severe cytotoxic edema and reduction of regional CBF, and is associated with a mortality rate of approximately 80%.[13,14] Other clinical conditions that can lead to elevated ICP include brain tumors, abscesses, meningitis, hydrocephalus, aneurysmal subarachnoid hemorrhage (SAH), idiopathic intracranial hypertension, and venous sinus thrombosis.[4,5]

CLINICAL PRESENTATION

The clinical presentation of patients with elevated ICP can vary according to the nature of the underlying disorder as well as the severity and acuteness of symptoms. For example, patients who experience a severe TBI can present with an acute onset of elevated ICP caused by a rapidly expanding epidural hematoma. On the other hand, patients with slowly growing brain tumors may tolerate elevated ICP without showing any clinical manifestation. The classic signs and symptoms of elevated ICP include headache, vomiting, and papilledema.[1] Papilledema is a very reliable and specific sign of elevated ICP; however, it may not be evident in a large number of affected patients and is known to be observer dependent.[15] The Cushing triad, including hypertension, bradycardia, and

respiratory irregularity (Cheyne-Stokes respiration), is a well-described phenomenon related to brainstem distortion or ischemia caused by elevated ICP.[16] The full triad, however, is only found in around 33% of cases. Cranial nerve palsies may also manifest in patients with elevated ICP.[17,18] Brain herniation syndromes, such as central and uncal herniation syndromes, can also occur as a result of elevated ICP, leading to progressive deterioration in the level of consciousness.

EVALUATION AND DIAGNOSIS

A high index of suspicion is crucial in the initial evaluation of patients at high risk of developing elevated ICP. Regardless of the underlying cause, assessment of the airway, breathing, and circulation (ABCs) should first take place in an acute setting. A full neurologic examination, particularly assessment of the Glasgow Coma Scale (GCS) score and bilateral pupils, should also be performed.[19] This neurologic assessment should be repeated frequently to detect early clinical deterioration. Any decrease in the patient's GCS score over the clinical course of the disease and/or development of pupillary asymmetry should be considered as a warning sign for an increasing ICP.[6] Endotracheal intubation should be considered in patients presenting with low levels of consciousness (GCS $\leq$8). In such scenarios, further clinical assessment of the patients is limited, and the use of radiologic studies, as well as direct ICP monitoring, becomes warranted. Unenhanced computed tomography (CT) of the brain can help support the diagnosis by detecting abnormalities that may be predictive of elevated ICP (eg, compression of basal cisterns and ventricles, effacement of sulci, and midline shift).[6,20,21] However, patients with no abnormal findings on initial CT scans may still have a 10% to 15% chance of developing elevated ICP.[22] Unenhanced CT of the brain can also help detect the underlying pathologic condition (eg, epidural hematoma). Magnetic resonance imaging (MRI), on the other hand, can help determine and calculate the stroke volume in patients presenting with large MCA infarcts.

MONITORING OF ICP

Methods

Two main methods of ICP monitoring are commonly used in clinical practice: (1) intraventricular catheters and (2) intraparenchymal microtransducer sensors.[23,24] Intraventricular catheters are considered the gold standard for ICP monitoring.[25] These catheters allow accurate measurement of the global ICP but also carry the advantage of therapeutic drainage of CSF.[24] The technique of intraventricular catheter insertion is described in the section on surgical management.

Indications for ICP Monitoring

Indications for ICP monitoring may include any clinical condition that carries an impending risk of elevated ICP (**Box 1**).[26] In 2007, the Brain Trauma Foundation guidelines recommended the use of ICP monitors in the management of patients with severe TBI.[10] These guidelines described level II evidence for ICP monitoring in all salvageable patients presenting with severe TBI (GCS 3–8) and an abnormal CT scan (defined as a scan that reveals an intracranial hematoma, contusion, swelling, herniation, or compressed basal cisterns).[10] In addition, level III evidence for ICP monitoring was described in patients presenting with severe TBI and a normal CT scan, but with 2 or more of the following: (1) age older than 40 years; (2) unilateral or bilateral motor posturing; or (3) systolic blood

Box 1
Potential indications for ICP monitoring

- According to Brain Trauma Foundation guidelines: All salvageable patients with Severe TBI (GCS 3–8) and
  - Abnormal CT scan (defined as scan that reveals intracranial hematoma, contusion, swelling, herniation, or compressed basal cistern) (level II evidence)
  - Normal CT scan, but with 2 or more of the following: (1) age older than 40 years, (2) unilateral or bilateral motor posturing, or (3) systolic blood pressure less than 90 mm Hg (level III evidence)
- Acute ischemic stroke (if surgical intervention is not feasible):
  - If patient is deteriorating clinically
  - If CT scan suggestive of mass effect
- Acute symptomatic hydrocephalus after SAH (level II evidence) and possibly in high-grade aneurysmal SAH
- Intubated patients in whom clinical assessment is not feasible
- After surgical removal of intracranial mass
- Central nervous system infections such as meningitis and encephalitis

Abbreviations: CT, computed tomography; GCS, Glasgow Coma Scale; ICP, intracranial pressure; SAH, subarachnoid hemorrhage; TBI, traumatic brain injury.

pressure less than 90 mm Hg.[10] Despite these recommendations, there has been significant variability in practice of ICP monitoring among practitioners worldwide.[27] In the authors' practice, the need for ICP monitoring is dictated by the clinical presentation of the patient and imaging findings, even in patients younger than 40 years. A case-by-case approach to all patients is therefore critical in the ICP management issues related to TBI. In a recently published multicenter controlled trial, Chesnut and colleagues[28] noted no significant improvement in outcomes in patients who presented with severe TBI and were managed according to the ICP monitoring guidelines, in comparison with those who were managed based on follow-up imaging and clinical examination (P = .49). This observation raises intriguing and counterintuitive questions about the true role of ICP monitoring in TBI.

Indications for ICP monitoring in other clinical settings are not well defined, and vary depending on individual and institutional protocols. According to the 2012 guidelines for management of aneurysmal SAH, insertion of an intraventricular catheter and diversion of CSF are recommended in patients presenting with acute symptomatic hydrocephalus after SAH (level II evidence).[29] In addition, significant support exists in the literature for ICP monitoring in patients presenting with high-grade aneurysmal SAH (Hunt and Hess grades III–V),[30] especially because elevated ICP in such patients is associated with poor outcome.[24,26,31,32] ICP monitoring has also been suggested in SAH cases where serial clinical assessment of patients is not feasible.[26] In the setting of acute ischemic stroke, it may be appropriate to monitor ICP in patients who are deteriorating clinically and in those with CT scans suggestive of mass effect.[4] However, it remains unclear whether ICP monitoring can improve clinical outcome in this particular setting.[24,33] Other indications of ICP monitoring may include central nervous system infections such as meningitis and encephalitis.[24]

Multimodal Neuromonitoring

A patient-specific multimodal intracranial monitoring approach has been suggested in the management of patients at increased risk of elevated ICP.[4,23,24,34–37] This approach includes: (1) monitoring of ICP and CPP; (2) monitoring of cerebral oxygenation (jugular bulb venous oximetry, or cerebral oxygen partial pressure); (3) monitoring of the metabolic status (cerebral microdialysis); and (4) monitoring of electrophysiologic brain activity (electroencephalography and somatosensory evoked potentials). It is thought that ICP monitoring alone may not reflect the full neurologic picture of patients at increased risk of elevated ICP. Consequently, ICP-driven therapy alone may not be able to address all the underlying pathophysiologic processes that can cause brain injury.[23,38] Furthermore, in their review of 70 patients who presented with severe TBI (GCS <8), Spiotta and colleagues[36] reported a significant decrease in the mortality rate among patients who received cerebral oxygen–driven therapy (25.7%) compared with those who received ICP-CPP–driven therapy alone (45.3%, P<.05). A similar decrease in the mortality rate was noted in several other studies comparing cerebral oxygen–driven therapy with ICP-CPP–driven therapy alone.[35,37] The studies suggested implementation of oxygen-driven therapy into the management of elevated ICP. Nevertheless, given the current lack of randomized controlled trials (RCTs) addressing this issue, definitive conclusions based on single-center experiences cannot be drawn.

Monitoring of the brain metabolic status using cerebral microdialysis techniques has also been suggested in the multimodal monitoring of patients at increased risk of elevated ICP.[39,40] Microdialysis catheters can detect intraparenchymal glucose and lactate concentrations in the brain, and are thought to reflect brain metabolic status. However, the role of microdialysis in the management of patients with elevated ICP remains to be defined.[41] Monitoring of the electrophysiologic activity of the brain can also be used in the management of patients at increased risk of elevated ICP.[4,34,42]

MANAGEMENT OF ELEVATED ICP

The main goal in the management of elevated ICP is to recognize and address the underlying pathology while at the same time applying measures to rapidly reduce ICP. In cases where the underlying pathology requires urgent surgery, for example in a patient presenting with a large epidural hematoma, it is important to move the patient to the operating room in a timely fashion. Meanwhile, noninvasive measures such as elevation of the head, mannitol administration, and even hyperventilation may be implemented to reduce ICP. In such cases, however, surgical evacuation of the hematoma may be the only definitive treatment for elevated ICP. In other cases where the underlying pathology is essentially nonsurgical, for example in a patient presenting with a mild to moderate cerebral edema from TBI or stroke, conservative (medical) therapy should be the primary treatment of choice, until further intervention becomes indicated.

Threshold Values for Treatment

According to the Brain Trauma Foundation guidelines, treatment of patients with elevated ICP caused by TBI should be initiated when ICP measurement exceeds 20 mm Hg (level II evidence).[10] Data from the literature have shown that patients with a mean ICP greater than 20 mm Hg have a significantly higher mortality rate than those with lower ICP values.[10,43,44] The mortality rate has also been shown to further increase in patients with prolonged, refractory elevated ICP.[45] Nonetheless, in the setting of less acute conditions, such as brain tumors, it has been suggested that patients may be able to tolerate higher ICP values, for longer periods of time, without significant complications.[6,11] Unfortunately, there are currently no well-defined thresholds for the treatment of elevated ICP caused by clinical conditions other than severe TBI. Most medical centers, however, initiate therapy when the ICP measurement exceeds 20 to 25 mm Hg.[5,6] The Brain Trauma Foundation guidelines also recommend treatment of patients with CPP values lower than 50 mm Hg (level III evidence).[10] Fluid resuscitation and vasopressors are typically used in such cases to induce hypertension, and therefore prevent cerebral ischemia that can result from low CBF and CPP.[24,46] It has been suggested, however, according to the same guidelines, to maintain a CPP value lower than 70 mm Hg to prevent the advent of life-threatening conditions such as secondary brain edema and/or adult respiratory distress syndrome, which can be induced by the excessive administration of vasopressors and fluids to raise CPP.[10,47,48]

Medical Management

When a patient is suspected clinically to have an elevated ICP confirmed by imaging, the treatment dictated by the clinical scenario should be initiated. Ordinarily, the head is elevated greater than 45° and sometimes up to 90° off the bed. The head is also maintained in midline and, if the patient has a neck collar, it is important to ensure that it is not compressing the jugular venous system in the neck area. The goal of these measures is to optimize the venous drainage of the brain and thus help decrease intracranial blood volume and ICP. Hyperosmolar therapy can then be implemented using diuretics, mannitol, and/or hypertonic saline. Mannitol is given at a bolus dose of 1 g/kg body weight followed by a maintenance dose of 0.5 g/kg every 6 hours, weaned over 2 to 3 days.[10] Serum osmolality is usually checked before every dose to maintain an osmolality of 310 to 320 mOsm/kg. Occasionally, a diuretic (eg, furosemide) may be used as needed to maintain a negative total daily balance. Meanwhile, mean arterial pressure is monitored to maintain CPP at 50 mm Hg or greater. A hypertonic saline (1.5% or 3% NaCl) drip may also be used, targeting a sodium level of 145 to 155 mEq/L. Serum sodium level should be monitored closely every 4 to 6 hours to ensure that the change in serum sodium level does not exceed 12 mEq/L/d. In the case of acute herniation, a sodium bullet (23.4%) may be used.

In the setting of an intracranial tumor with concern of associated elevated ICP, steroids may help decrease vasogenic edema by decreasing brain volume. On the other hand, if a patient has presented with a low GCS score that required intubation, the insertion of an ICP monitor may be warranted. In such cases, additional maneuvers such as short-term hyperventilation can be used to treat spikes of elevated ICP (target $Paco_2$: 30–35 mm Hg or even lower).[49] In patients with an intraventricular catheter, CSF drainage may be used as well. In cases where medical management fails and surgical intervention is not feasible, barbiturates can be used to induce a pharmacologic coma, which usually controls ICP by decreasing the metabolic demand and sympathetic response.[10]

Surgical Management

Although medical treatment may in certain instances reduce elevated ICP, medical management alone may not be sufficient to affect a normal ICP. Clinical conditions including severe TBI, large ischemic stroke, and high-grade SAH, as well as large subdural, epidural, and intraparenchymal hematomas, can result in a propagating a vicious cycle of brain edema, elevated ICP, reduced CBF, hypoxemia/ischemia, and further edema, eventually leading to severe brain injury.[50] Breaking this vicious cycle may require an urgent surgical intervention that allows complete removal of the underlying pathology or provides more room for brain swelling. Surgical management of elevated ICP includes: (1) insertion of intraventricular catheter and CSF drainage; (2) removal of an intracranial space-occupying lesion; and (3) undergoing a decompressive craniectomy.

Insertion of intraventricular catheter and CSF drainage

The earliest report on surgical cannulation of the lateral ventricle dates back to the late 1800s, when Keen first described the anatomic landmarks used in the procedure to treat hydrocephalus.[51,52] In 1908, Anton and Von Bramann described the first corpus callosum puncture technique, which aimed at connecting the lateral ventricle to the

subarachnoid space.[53,54] Ten years later, Dandy described his technique of intraventricular catheterization of the lateral ventricle, which involved anterior and occipital ventricular horn punctures to obtain an air ventriculography.[55,56] Guillaume and Janny[57,58] eventually introduced the concept of ICP monitoring using intraventricular catheters in the early 1950s. Currently, with proper training and experience, placement of external ventricular drains in most patients can be achieved safely and quickly at the bedside.[59] In addition, intraventricular catheters are now considered the gold standard for measurement and monitoring of elevated ICP, and are also an important component of its management.

Indications In addition to their previously described role in ICP monitoring, intraventricular catheters are used in the treatment of various clinical conditions associated with elevated ICP. These catheters allow temporary drainage of CSF from the ventricular system, resulting in an immediate reduction of the intracranial volume and ICP. Indications for insertion of an intraventricular catheter and CSF drainage include elevated ICP attributable to a variety of causes including acute or subacute hydrocephalus, severe TBI, subarachnoid hemorrhage (Hunt and Hess grades 3–5), intraventricular hemorrhage, and meningitis.[6] Intraventricular catheters can further serve as a means for direct injection of tissue plasminogen activator (tPA) into the ventricular system in cases of severe hemorrhage (casted ventricles). Studies on intraventricular injection of tPA have shown promising results in terms of clot resolution and reduction in mortality rate.[60–63] In the recently published CLEAR-IVH trial (Clot Lysing: Evaluating Accelerated Resolution of Intraventricular Hemorrhage), tPA was found to significantly accelerate the resolution of intraventricular hemorrhage in a dose-dependent manner ($P<.0001$).[64] Drainage of CSF using an intraventricular catheter can provide brain relaxation, and may therefore be beneficial during aneurysm surgery. Indications for the insertion of an intraventricular catheter and ICP monitoring in TBI are summarized in **Box 1**.

Surgical technique Before the insertion of an intraventricular catheter, it is important to assess the size of the lateral ventricles to determine whether an adequate volume of CSF is available. Placement of a catheter into severely compressed ventricles can be technically challenging, and may lead to devastating complications such as hemorrhage.[6] Neurosurgical navigation can be helpful in cases of slit ventricles or shifted ventricles caused by mass effect.[6,52] It is also important to study intracranial abnormality before inserting an intraventricular catheter, to avoid passing through lesions lying in the trajectory of the catheter (eg, arteriovenous malformation).[52] Intraventricular catheters are usually inserted into the frontal horn of the lateral ventricle through a burr hole made at the Kocher point (usually the nondominant side), which is located 11 cm posterior to the nasion and 2 to 3 cm from the midline (approximately at the midpupillary line). After defining the anatomic landmarks, proper prepping and draping of the surgical site should be performed. A 1-cm linear skin incision is made, and a self-retaining retractor is used to hold the skin edges. A twist drill is then used to access the dura. After the drilling is completed, bone-wax may be placed on bone edges to stop the bleeding. A small incision in the dura is made. The catheter is then inserted perpendicular to the brain surface for a depth of 5 to 6 cm (from the brain surface) aiming at the ipsilateral medial epicanthus in the coronal plane and external auditory meatus in the sagittal plane, until CSF is obtained.[56] Deeper insertion of the catheter ($\geq$8 cm from the brain surface) often results in undesirable positioning and should therefore be avoided. This procedure can be performed free-handedly or using stereotactic guidance. The catheter is tunneled subcutaneously around 3 to 5 cm away from the burr hole, and is attached to an external pressure transducer with a 3-way stopcock and a draining bag. In the case of a ruptured aneurysm, care should be taken to avoid any rapid drainage. The authors open the drain at 20 cm H_2O in such cases. For most other indications the drainage threshold is typically set at 10 cm H_2O. The patient's head is elevated at an angle of approximately 30° and the zero-line, located at the ICP monitor scale, is placed at the level of the tragus (equivalent to foramen of Monroe). The setting for CSF drainage can be adjusted based on clinical and imaging response. In cases of trauma-induced elevated ICP, once a normal ICP is maintained for 48 to 72 hours the draining tube is clamped and the ICP monitored, with serial neurologic examinations being performed. If the patient does not show any signs or symptoms of elevated ICP for 12 to 24 hours, the catheter may be removed.

Outcomes In terms of clinical outcomes, external ventricular drainage has been shown to result in immediate reduction in ICP and has been deemed to be a highly effective measure for controlling ICP in multiple clinical settings.[65–68] Evidence of improved neurologic outcomes at 6 months has been reported following ICP control with

intraventricular catheters in patients presenting with TBI.[6,65] Improvement in the neurologic status of patients presenting with aneurysmal SAH has also been noted after insertion of an intraventricular catheter.[68]

Complications Three main complications are associated with the insertion of intraventricular catheters: (1) intracranial bleeding, (2) infection, and (3) obstruction.[6] In their meta-analysis of hemorrhagic complications associated with insertion of intraventricular catheters, Binz and colleagues[69] reported an overall hemorrhagic risk of 5.7% and a clinically significant hemorrhagic risk of less than 1%. The risk of intracranial bleeding is found to increase in patients with coagulation disorders, and may be influenced by technique. Subdural hematoma is another potential complication.[70,71] Rebleeding from a ruptured intracranial aneurysm can occur following the insertion of an intraventricular catheter, possibly due to changes in the pressure gradients across the aneurysm wall.[72] Infection related to insertion of intraventricular catheters is another potential complication that may result in ventriculitis, meningitis, formation of brain abscess, and empyema. The rate of infection associated with intraventricular catheters ranges between 0% and 22%.[52,73,74] Catheter infection can be minimized by ensuring a sterile insertion technique with appropriate catheter tunneling and antibiotic administration.[75] In addition, proper wound care and minimum interruption of the closed system (eg, frequent flushing) may help reduce infection rates.[52,75] The use of antibiotic-impregnated catheters has been suggested as a way to reduce the risk of infection.[76,77] In their review of the literature, Gutierrez-Gonzalez and Boto[76] reported a decrease in device-related infection rate and hospital costs in patients with antibiotic-impregnated catheters, compared with those with standard catheters. The review included 3 retrospective cohorts, 1 retrospective review of a prospective database, and 1 prospective RCT.[76] However, in a recently published prospective RCT, these catheters did not a show significant reduction in infection risk when compared with standard catheters.[78] Silver-impregnated catheters have been shown to significantly reduce the infection rate in comparison with standard catheters in a double-blind RCT.[79] Obstruction of CSF drainage is a commonly encountered complication of intraventricular catheters, which can be caused by a blood clot obstructing the catheter.[6] Flushing the catheter with normal saline or tPA, in cases of hemorrhage, may clear the pathway and retain function. If patency cannot be established with flushing, the catheter may be replaced by a new catheter, typically with a "soft-pass technique."

Removal of intracranial space-occupying lesions

The most effective and definitive treatment for elevated ICP caused by a space-occupying lesion is surgical removal. Of note, the insertion of an intraventricular catheter can also be helpful in detecting secondary increases in ICP and can be an important component of the surgical management.[6,10,80] Intracranial hematomas are by far the most common cause of elevated ICP in patients presenting with severe TBI, complicating around 25% to 45% of cases.[81] According to the Brain Trauma Foundation guidelines, surgical evacuation of acute epidural hematomas is indicated if the hematoma is greater than 30 cm^3, regardless of the patient's GCS.[82] However, nonsurgical measures and serial CT scans may be implemented in the management of epidural hematomas that are smaller than 30 cm^3 and have the following features: (1) a thickness less than 15 mm, (2) a midline shift less than 5 mm, and (3) a GCS greater than 8.[82] Evacuation of subdural hematomas is also indicated in cases where a hematoma thickness greater than 10 mm or a midline shift greater than 5 mm is noted on CT scan.[80] Comatose patients with smaller hematomas may also need surgical evacuation if they deteriorate clinically between the time of injury and hospital admission: (1) GCS decrease by 2 or more points, (2) and/or asymmetric or fixed, dilated pupils, (3) and/or ICP increase more than 20 mm Hg.[80]

In contrast to epidural and subdural hematomas, for which clear evidence supporting surgical evacuation is available, surgical management of traumatic intraparenchymal mass lesions remains a subject of controversy.[6,83–85] The Surgical Trial in Traumatic Intracerebral Hemorrhage (STITCH) is a multicenter RCT that is currently comparing outcomes of early surgical treatment of patients with traumatic intracerebral hemorrhage with those of conservative (medical) therapy.[83] Until prospective data are obtained from this trial, evidence regarding the management of patients with traumatic intracerebral hemorrhage remains dependent on single-center retrospective studies.[6,86,87] Based on the available studies, the Brain Trauma Foundation guidelines recommended surgical evacuation of intraparenchymal hematomas in clinically deteriorating patients, and in patients who are not responding to medical therapy or who have a clear evidence of mass effect on imaging.[88] Patients with frontal or temporal contusions greater than 50 cm^3 should also be treated surgically.[88] Similarly, patients with

contusions greater than 20 cm^3, who have a GCS score of 6 to 8 and an additional midline shift of at least 5 mm and/or cisternal compression, may be considered for surgical treatment.[88]

Indications for surgical evacuation of spontaneous intracerebral hematomas remain controversial. In the Surgical Trial in Intracerebral Hemorrhage (STICH), 1033 patients presenting with spontaneous intracerebral hemorrhage were randomized to one of two treatment arms: (1) early surgery or (2) initial conservative therapy.[84] The results of the trial showed no overall benefit from early surgery (468 patients) in comparison with conservative treatment (496 patients) (P = .414).[84] The mortality rate at 6 months for surgically treated patients was 36%, compared with 37% for conservatively treated patients. A trend toward favorable outcome from early surgery was noted in patients with superficial (cortical) intracerebral hematomas 1 cm or less from the cortical surface in comparison with deeper hematomas.[84] In current practice, whether an intracerebral hematoma should be surgically evacuated depends largely on the clinical condition of each patient and the treatment protocol at each institution. Surgical management of patients with spontaneous intracerebral hematomas may include the insertion of an intraventricular catheter in the contralateral lateral ventricle to help monitor and control ICP. This action can be of particular importance in patients who present with an intraventricular extension of the bleed.

Indications for surgical removal of other space-occupying lesions, such as brain tumors or abscesses, in the setting of elevated ICP are not well defined and may therefore vary according to the inherent features of the lesion itself, the clinical condition of the patient, and the experience at each center. In general, any abnormal intracranial mass resulting in elevated ICP should be considered for surgery, especially after failure of medical therapy.

Decompressive craniectomy

The practice of craniectomy dates back to the prehistoric age.[89] In an interesting observation, human skulls from the Paleolithic Period were found to have bony defects suggestive of human interventions.[89] These defects were recognized by Broca, in the late 1800s, to have been created in subjects who subsequently survived.[90,91] It remains unclear, however, whether these procedures were performed as part of old rituals or in attempts to treat patients with disorders. The modern description of craniectomy goes back to the time of 2 famous neurosurgeons, Theodor Kocher (1901) and Harvey Cushing (1905), who were the first to report surgical decompression techniques as a measure to effectively reduce elevated ICP.[92] It was suggested that surgical removal of part of the skull provides room for the brain to swell and therefore reduces ICP. Over the past century, however, management of elevated ICP with decompressive craniectomy has been a subject of extensive scrutiny and controversy. It was not until 1999, after Guerra and colleagues[93] published their experience of 20 years, that decompressive craniectomy regained wide acceptance. At present, decompressive craniectomy is being implemented in various treatment protocols for the management of elevated ICP and has been part of several RCTs.[12,93,94]

Indications Three main indications, of variable levels of evidence, exist for the use of decompressive craniectomy in the management of elevated ICP: (1) severe TBI, (2) malignant MCA infarction, and (3) aneurysmal SAH.[50,92] According to the Brain Trauma Foundation guidelines, bifrontal decompressive craniectomy is indicated within 48 hours of injury for patients with diffuse, posttraumatic cerebral edema and medically refractory elevated ICP.[88] In addition, according to the same guidelines, subtemporal decompression, temporal lobectomy, and hemispheric decompressive craniectomy can be considered as treatment options for patients who present with diffuse parenchymal injury and refractory elevated ICP who also have clinical and radiographic evidence for impending transtentorial brain herniation.[88] Despite these guidelines, there has been a continuous debate regarding the optimal timing of the procedure, and its effect on patient outcome and quality of life.[6] Many argue that the obtained results may not justify the treatment, especially if the reduction in the mortality rate is to be associated with an increase in the rate of severe disability.[50,92] Furthermore, long-term functional outcomes of patients undergoing decompressive craniectomy after TBI remain to be determined.

A higher level of evidence is currently available for the use of decompressive craniectomy in patients with malignant MCA infarction.[95] This evidence is based on data obtained from 3 major RCTs: DECIMAL,[96] DESTINY,[97] and HAMLET (**Table 1**).[98] Indications for decompressive craniectomy in patients with malignant MCA infarction are well described in the 2008 guidelines of the United Kingdom National Institute for Health and Clinical Excellence.[99] Decompressive craniectomy is recommended in patients who present within 24 hours of the onset of symptoms and meet all of the following criteria: (1) age younger than 60 years, (2) National Institute of Health Stroke Scale (NIHSS) score higher than 15, and (3) infarct of at least 50% of the MCA territory or infarct volume greater than

Table 1
Randomized controlled trials conducted on decompressive craniectomy in the management of malignant MCA infarction

Study	Study Type	No. of Patients	Time Interval from Symptom Onset to Surgery (h)	Follow-Up	Treatment Arms	Primary Outcome (mRS <4) (%)	Severe Disability (mRS 4–5) (%)	Mortality (mRS 6) (%)
HeADDFIRST (Frank et al)[133]	Multicenter RCT	26	96	180 d	DC Conservative	Awaiting publication	—	35.3 40.0
HeMMI (Jamora et al)[134]	Single-center RCT	—	72	6 mo	DC Conservative	Recruitment started in 2004, awaiting publication	—	—
DECIMAL (Vahedi et al,[96] 2007)	Multicenter RCT	38	24	12 mo (stopped)	DC (n = 20) Conservative (n = 18)	50.0 22.2	25.0 0	25.0 77.8
DESTINY (Juttler et al)[97]	Multicenter RCT	32	12–36	12 mo (stopped)	DC (n = 17) Conservative (n = 15)	47.1 26.7	35.3 20.0	17.6 53.0
HAMLET (Hofmeijer et al,[98] 2006)	Multicenter RCT	64	96	12 mo (stopped)	DC (n = 32) Conservative (n = 32)	25.0	53.1 15.6	21.9 59.4
POOLED ANALYSIS (Vahedi et al,[95] 2007)	Multicenter RCT: enrollment in DECIMAL, DESTINY, or HAMLET	93	48	12 mo	DC (n = 51) Conservative (n = 42)	43.1 21.4	35.3 7.2	21.6 71.4

Abbreviations: DC, decompressive craniectomy; MCA, middle cerebral artery; mRS, modified Rankin Scale; RCT, randomized controlled trial.

145 cm^3 as shown on diffusion-weighted MRI.[99] The procedure is likely best performed within a maximum of 48 hours after the onset of symptoms.[99] In a pooled analysis combining data from the 3 RCTs, decompressive craniectomy done within 48 hours of stroke onset was associated with lower mortality and more favorable outcomes (see **Table 1**).[95] Whether the outcome scales used in clinical trials truly reflect the quality of life of patients after surgery remains a subject of controversy. Decompressive craniectomy may still be considered in patients older than 60 years; however, the likelihood of a good outcome is less than in younger individuals. In their literature review on decompressive craniectomy performed in patients with malignant MCA infarction, Arac and colleagues[13] reported significantly higher rates of poor outcomes in the patient group older than 60 years (81.8%) compared with patients 60 years or younger (33.1%) ($P<.0001$).

Evidence for the use of decompressive craniectomy in the setting of aneurysmal SAH is limited to single-center experiences.[32,94,100–102] In one study by Buschmann and colleagues,[102] 38 patients who presented with aneurysmal SAH were treated with surgical clipping of the aneurysm and concomitant decompressive hemicraniectomy. Of the 38 patients, 31 presented with a high-grade SAH (Hunt and Hess grade 4–5). Indications for the hemicraniectomy included: (1) intraoperative signs of brain edema (group 1); (2) elevated ICP with epidural, subdural, or intracerebral hematoma after surgery (group 2); (3) elevated ICP and cerebral edema without radiologic signs of infarction (group 3); and (4) elevated ICP and cerebral edema with radiologic signs of infarction (group 4).[102] At 12-month follow-up, a good functional outcome was noted in 52.4% of patients in group 1, 60% in group 2, 83.3% in group 3, and 16.7% in group 4.[102] The study concluded that more than half of the patients with aneurysmal SAH can benefit from decompressive craniectomy, and recommended early surgical intervention in patients who present with aneurysmal SAH and refractory elevated ICP.[102] Another study by Smith and colleagues[32] reported good outcomes in patients who presented with a high-grade SAH (Hunt and Hess grade 4–5) and large Sylvian fissure hematoma, and who were treated with decompressive hemicraniectomy and surgical evacuation of the hematoma. The study suggested that a decompressive hemicraniectomy may be of benefit in a carefully selected group of patients with aneurysmal SAH.[32]

Other indications for decompressive craniectomy may include intracerebral hemorrhage,[103,104] subdural empyema,[105] meningitis,[106] encephalitis,[107] toxoplasmosis,[108] encephalopathy caused by Reye syndrome,[109] and severe cerebral venous and dural sinus thrombosis.[110]

Surgical technique Two main types of decompressive craniectomy are currently used in clinical practice: (1) unilateral craniectomy and (2) bilateral craniectomy.[50,92,111–114] Unilateral craniectomy, or decompressive hemicraniectomy, is usually performed in patients presenting with unilateral brain edema and midline shift caused by TBI, malignant MCA infarction, or SAH.[6,12,102] The presence of midline shift is an important factor that dictates the side on which the craniectomy should be performed. A thorough evaluation of the patient should be conducted prior to the procedure. Once in the operating room, the patient is placed in the supine position after induction of general anesthesia. The head is elevated and turned laterally, such that the craniectomy side faces upward, and is then fixed with a head frame. If needed, a shoulder roll may be used to elevate the ipsilateral shoulder and assist positioning. It is important to avoid excessive rotation of the head and neck so as not to compress the jugular venous system. After proper shaving, a marker is used to align the surface anatomy of important structure, such as the sagittal and transverse sinuses, and to draw the incision line. The incision line is "question mark" in shape, starting approximately 1 cm anterior to the tragus and moving up toward the area just above the ear pinna, then turning posteriorly toward the external occipital protuberance (**Fig. 1**). The line is then curved superiorly and anteriorly to end just behind the hairline. It is important to ensure that the curvature extends posterior enough to allow for a minimum craniectomy size of 12 cm.[12,115] Local anesthesia is injected along the incision line, and appropriate prepping and draping is performed. The incision is made, and the skin flap and underlying temporalis muscle are reflected anteriorly. Five burr holes are made in the following locations: (1) temporal squamous bone superior to the zygomatic process inferiorly, (2) keyhole area behind the zygomatic arch anteriorly, (3) along the superior temporal line posteroinferiorly, and in the (4) parietal and (5) frontal parasagittal areas (see **Fig. 1**). The burr holes are then connected with a footplated bit. Careful attention should be given to avoid laceration of the superior sagittal sinus. The bone flap can then be removed and stored either in the subcutaneous tissue of the abdominal wall or in a freezer (-20° to -70°C).[92,116] Further removal of bone from the sphenoid wing of the temporal bone can help provide access to the temporal lobe. A stellate-shaped durotomy is then made slowly and carefully to avoid injury to

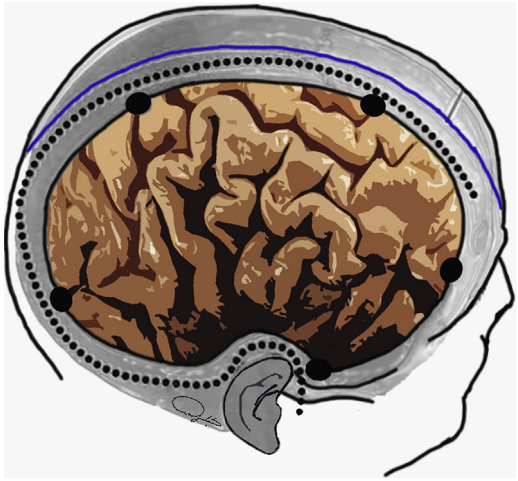

Fig. 1. Unilateral craniectomy or decompressive hemicraniectomy. Question mark–shaped skin incision (*dotted line*). Five burr holes made in the temporal squamous bone superior to the zygomatic process inferiorly, keyhole area behind the zygomatic arch anteriorly, along the superior temporal line posteroinferiorly, and in the parietal and frontal parasagittal areas.

the underlying brain tissue. It is important to extend the durotomy inferiorly to the skull base, to decompress the middle cranial fossa.[111] After the durotomy is completed, a drain may be placed if indicated. A duraplasty is performed, and a layer of Surgicel (Johnson and Johnson Medical, Inc., Arlington, TX, USA) left on the brain surface. The temporalis muscle and fascia are left unsutured to allow expansion of the brain, whereas the galea and skin are sutured in layers. Reconstruction with autologous or synthetic bone is often performed 5 to 8 weeks after the procedure.[92]

Bilateral craniectomy is usually indicated in patients who present with diffuse brain edema without a focal lesion (eg, contusions due to severe TBI).[6,117] This procedure can be performed by either decompressive hemicraniectomies on both sides, or a bifrontal craniectomy.[92,117] The same surgical principles, previously described for the unilateral craniectomy, apply for the bifrontal craniectomy. However, in the latter the head should be fixed in the neutral position, and the incision should extend from the area anterior to the tragus on both sides. The curve should be made parallel and 2 to 3 cm posterior to the coronal suture. Burr holes are made in keyhole areas on both sides, in both squamous temporal bones, and in the parietal area just behind the coronal suture and 1 cm from the midline.[111] The burr holes are connected, and the bone flap carefully removed. The area overlying the superior sagittal sinus should be manipulated carefully, as the dura may be adherent to the scalp. A rim of bone may be preserved over the sinus to avoid bleeding complications.[93] The durotomy may be performed in a bilateral stellate, U-shaped, or "fish-mouth" fashion.[50,111,116]

Outcomes Over the past few years, several single-center studies have been conducted to assess the outcome of decompressive craniectomy in the setting of TBI.[118–120] However, variations in patient selection, outcome scoring, and the timing and techniques of surgery have made it difficult to reach conclusions. In one of the largest studies, Williams and colleagues[118] retrospectively reviewed 171 patients who presented with severe TBI and elevated ICP and underwent decompressive craniectomy. The study used the Glasgow Outcome Scale Extended (GOSE), and reported good outcomes (GOSE 5–8) in 56% of the patients. This outcome was noted in younger individuals and in patients who achieved higher ICP reduction.[118] More recently, the outcome of decompressive craniectomy after TBI has been assessed in RCTs (**Table 2**). In the DEcompressive CRAniectomy (DECRA) trial, published in 2011, 155 adult patients presenting with severe TBI and refractory elevated ICP were randomized to one of two treatment arms: (1) early bifrontotemporoparietal decompressive craniectomy (within 72 hours) and standard care; or (2) standard care alone.[117] The study showed that early decompressive craniectomy is associated with more unfavorable outcomes at 6 months than is standard care ($P = .02$).[117] However, decompressive craniectomy was found to significantly decrease ICP and the length of stay in the intensive care unit. Mortality rates were similar in both treatment arms (19% in craniectomy group vs 18% in standard-care group).[117] At present, another ongoing RCT (RESCUEicp: Randomized Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of intracranial pressure) is evaluating the role of decompressive craniectomy in the management of patients with severe TBI and refractory elevated ICP.[121] This study is expected to provide additional insight regarding the long-term outcome of decompressive craniectomy in the setting of severe TBI. Other issues, such as the timing of surgery and the optimal technique to be used, also need to be clarified.

The effect of decompressive craniectomy on outcome of patients with malignant MCA infarction has been assessed in multiple retrospective and nonrandomized prospective studies.[122–125] Most studies showed a significant decrease in mortality when compared with medical management. However, some uncertainty remains regarding the impact on functional outcome and quality of life.

Table 2
Randomized controlled trials conducted on decompressive craniectomy in the management of traumatic brain injury

Study	Study Type	Inclusion Criteria	Treatment Arms	Follow-Up	Outcome
DECRA trial (Cooper et al,[117] 2011)	Multicenter RCT	Severe diffuse TBI and intracranial hypertension refractory to first-tier therapies	Two arms: 1. DC + standard care (n = 73) vs 2. Standard care alone (n = 82)	6 mo	DC group had less time in ICU and less time with high ICP. On Extended Glasgow Outcome Scale DC patients performed worse than patients who received standard care (OR 1.84) and had a greater risk of unfavorable outcome (OR 2.21). Death rate in DC group (19%) and in standard-care group (18%) was comparable
RESCUEicp (Hutchinson et al,[121] 2006)	Multicenter RCT	Severe TBI; ICP refractory to optimal, protocol-driven conservative therapy	Two arms: 1. DC vs 2. Medical management	—	Ongoing

Abbreviations: DC, decompressive craniectomy; ICP, intracranial pressure; ICU, intensive care unit; OR, odds ratio; RCT, randomized controlled trial; TBI, traumatic brain injury.

In an attempt to better define the role of decompressive craniectomy in the management of malignant MCA infarction, 3 RCTs were conducted: DECIMAL,[96] DESTINY,[97] and HAMLET (see **Table 1**).[98] In their pooled analysis of the 3 trials, Vahedi and colleagues[95] reported on 93 patients, of whom 51 were randomized to decompressive craniectomy and 42 to medical treatment. The study noted a higher number of patients in the surgical arm who had a modified Rankin Scale (mRS) score of 4 or less (75% vs 24% in the medical arm), mRS score of 3 or less (43% vs 21% in the medical arm), and who survived (78% vs 29% in the medical arm).[95] Data obtained from the 3 randomized trials and the pooled analysis suggests that decompressive craniectomy can significantly decrease mortality and increase the number of patients with a favorable outcome. However, it must be noted that the number of patients with moderately severe disability (mRS $\leq$4) is also increased.[95] In addition, the long-term functional and psychosocial outcome, as well as the effect of decompressive craniectomy on elderly patients, remained undefined. Recently, in their systematic review of the literature, Rahme and colleagues[126] assessed functional outcome, depression, quality of life, and mortality rates in patients who underwent decompressive craniectomy in the treatment of malignant MCA infarction. Their study included 3 major RCTs, 3 prospective cohorts, and 10 retrospective studies. The mortality rate was 24.3% among the 382 patients reviewed. Favorable functional outcome was reported in 41% of the 156 survivors (mRS score $\leq$3); however, 47% of the patients experienced moderately severe disability (mRS score $\leq$4).[126] The quality of life was also assessed despite significant variations in assessment tools and reporting among studies. The mean overall reduction in quality of life after decompressive craniectomy was 45.2% of the 157 survivors with available quality-of-life data. The mean physical and psychosocial reductions in quality of life were 66.9% and 36.8%, respectively.[126] In terms of depression outcomes, 25% of the 80 survivors with available data were diagnosed with moderate or severe depression. It is interesting that despite the high number of patients with moderate disability, 76.6% of the 209 survivors with available patient feedback after surgery were satisfied with their lives and announced they would again give their consent for decompressive craniectomy.[126]

The outcome of decompressive craniectomy in patients presenting with SAH and refractory elevated ICP has been assessed in several studies.[94,101,102] In their review of 16 patients with aneurysmal SAH who underwent decompressive craniectomy, Schirmer and colleagues[94] reported good functional outcomes in 7 patients (64%; mRS $\leq$3). Eleven patients in this study had a Hunt and Hess score of 4 to 5. The median follow-up duration was approximately 15 months.[94] Decompressive craniectomy performed within 48 hours after SAH was the only factor significantly associated with better outcomes (P<.01). The study suggested early surgery in patients with high-grade aneurysmal SAH.[94] Ziai and colleagues,[101] on the other hand, reported poor outcome in 3 of 4 patients with aneurysmal SAH who underwent decompressive craniectomy, and death in 1 patient. Because of the lack of clear guidelines recommending decompressive craniectomy in this particular setting, a careful case-by-case approach to all patients with aneurysmal SAH is advised. The pros and cons of surgical intervention and the possibility of a poor prognosis should be discussed with the family.

Complications The most common complication following decompressive craniectomy is the formation of a subdural fluid collection or hygroma.[93,127–129] In their review of 57 patients who underwent decompressive craniectomy after TBI, Guerra and colleagues[93] reported the occurrence of postoperative hygromas in 15 patients (26%). Kilincer and colleagues[128] also reported contralateral subdural effusion in a patient who presented with aneurysmal SAH and underwent surgical clipping and decompressive craniectomy. Brain herniation through the cranial defect is another potential complication that can occur as a result of inadequate decompression.[115,130] In their review of 60 patients who presented with right MCA infarctions and underwent decompressive hemicraniectomy, Wagner and colleagues[115] reported a higher rate of bleeding complications and mortality in association with small-sized craniectomies, and recommended a craniectomy size of at least 12 cm to achieve an adequate decompressive volume and reduction in ICP.[115] Other complications may include infections such as meningitis or osteomyelitis of the bone flap,[129,131] postoperative seizures,[132] and hydrocephalus.

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