Chapter 8
Decompressive Craniectomy

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Clinical Scenario

A man in his twenties is brought to the emergency room. It was reported that he was the unrestrained driver of a vehicle involved in a high-speed, roll-over collision. On arrival of the pre-hospital medical crew, the patient was unconscious; he had a patent airway but was hypoxic and hypotensive with high clinical suspicion of respiratory compromise due to chest trauma and pneumothorax. He received rapid sequence induction and was intubated at the scene while maintaining spinal precautions; bilateral thoracostomies were performed. Pre-intubation Glasgow Coma Score (GCS) was 5 (E1V1M3). Both pupils were small, with preserved pupillary light reflex on examination. He received supplemental oxygen and fluid resuscitation correcting the hypoxia and hypotension. There was suspicion of left femur fracture, and his left lower limb was immobilized. He was transported emergently to the nearest trauma center with full spinal precautions.

8.1 History and Neurologic Exam

Traumatic brain injury (TBI) is a significant cause of preventable morbidity and mortality across the world, with the most prevalent causes of TBI being falls, assaults, and motor vehicle accidents [1]. Prompt recognition and institution of TBI management improves outcomes [2].

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When attending to a trauma patient, the primary consideration is identification and management of immediately life-threatening injuries following the Advanced Trauma Life Support (ATLS) protocol, with multidisciplinary input [2]. Information from first responders and the pre-hospital medical team (who will often relay relevant information from bystanders) about the timing and circumstances of injury, followed by their clinical assessment of the patient at the scene and during transfer to hospital, will help the multidisciplinary trauma team to gauge the mechanism and potential impact and extent of injury, including the likelihood of TBI. Incorporating the information about the patient's level of consciousness and pupillary size and reactivity to light at the scene will then inform about the clinical severity of TBI.

8.1.1 History

TBI can be classified using different approaches, but the most frequently employed are mechanism of injury, clinical severity, and imaging findings. Obtaining information relevant to these elements informs the clinical picture at presentation and helps guide further interventions.

Different mechanisms of injury include closed, penetrating, crush, blast, and combination injuries—each resulting in characteristic pathological changes in the brain. Closed injuries involve cases where the cranium remains intact after injury. A major contribution of acceleration and deceleration forces would typically cause various degrees of diffuse axonal injury; this is often the case with road traffic accident. However, if there is an element of an impact in which the head hits a hard surface, either during a road traffic accident or as a consequence of a fall or a blow to the head, then this will typically result in focal contusional head injury and/or extra-axial hematoma [3, 4]. At the time of the impact, some of the dynamic forces are absorbed by the skull, which can result in skull fracture(s); depending on the magnitude of the impact, a varying degree of dynamic force will then be transferred onto the intracranial contents [3, 4]. In crush injuries, the dynamic forces are largely absorbed by the skull, causing skull injuries to be extensive and the TBI less severe. In penetrating head injuries, a projectile damages the brain tissue—on its way generating contusions and hematomas, with a high risk of vascular injury [3, 4].

The provider, therefore, should obtain as much information as possible about the circumstances of the injury. For a road traffic accident, the occurrence of blunt head trauma, the use of restraints, the rate of speed, the extent of damage to the vehicle(s), the deployment of airbags, and the severity of injury to other passengers may be relevant. Time to extrication is also important. For a fall, the height and point(s) of contact with the ground (or with other objects mid-fall) may be relevant.

Clinical severity of TBI is defined by patient's level of consciousness, which is assessed using the Glasgow Coma Scale (GCS) score [5]. Eye, verbal, and motor responses are recorded and added up to make the GCS score ranging from 3 to 15. Patients scoring 8 or less are classified as severe TBI, 9–12 as moderate, and 13–15 as mild [6]. The overall GCS score and the motor score are major predictors of

outcome in severe TBI [2]. It should be noted that GCS can change early after injury, following resuscitation or early recovery. Furthermore, it should be considered that GCS assessment can be confounded by prior alcohol or substance use, seizures (and post-ictal state), and hypoglycemia, as well as the use of sedation and paralysis in preparation for endotracheal intubation. It is thus paramount to record the GCS and its three components accurately before the patient is sedated and intubated.

Though not always feasible due to clinical condition at presentation and/or the absence of corroborating family members, information should be solicited regarding the patient's past medical and surgical history, as well as chronic medications—particular antiplatelet and/or anticoagulant agents—and social history.

8.1.2 Examination and Early Management

Primary survey of the trauma patient involves rapid identification of life-threatening extracranial injuries and rapid resuscitation. Adherence to Advanced Trauma Life Support (ATLS) guidelines ensures that assessment and treatment are provided thoroughly yet efficiently [2]. Hypotension and hypoxia, both pre-hospital and in-hospital, increase morbidity and mortality following severe TBI; therefore, the prevention or prompt correction of existing hypotension (aiming for a systolic blood pressure of at least 90 mmHg) and hypoxemia is of extreme importance [6]. However, if a lower blood pressure is required during the treatment of life-threatening extracranial hemorrhage, the duration of hypotension should be as short as possible, and other physiological parameters should be optimized to maximize cerebral oxygen delivery, such as avoiding hypoxia and hypocapnia [6].

Neurological examination will yield the patient's GCS score (unless already sedated), pupillary size, and reaction to light. Head-to-toe examination will reveal any external signs of head injury (lacerations with or without underlying skull fracture, abrasions, periorbital and soft tissue hematomas, mastoid bruising (i.e. Battle's sign), blood, or CSF otorrhea). In a comatose or sedated patient, pupillary size and reaction to light have significant diagnostic and prognostic weight. If there is unilateral or bilateral pupillary dilatation and loss of reaction to light, neuroprotective measures need to be adopted immediately—nursing the patient head up at 30° if possible, avoiding hypercapnia or even allowing a period of hyperventilation and hypocapnia, and using osmotic therapy as temporary measures until further diagnostic procedures have been completed and interventions can be performed [7].

In the current clinical scenario, the precipitating event is a road traffic accident, so the mechanism of injury is presumed to be a closed head injury. A GCS of 5 defines the TBI as severe. The motor score suggests a "best" response of flexion to stimulus. Pupils are small, equal, and reactive to light. Comorbid injuries are suspected. The presence of hypoxia and hypotension in the field raises concern for exacerbation of TBI. The patient's low GCS at presentation precludes interview to establish past medical history (Table 8.1).

Table 8.1 Key aspects of history and examination

History	– Injury mechanism (from bystanders/first responders) – Events at scene of trauma – Neurological status at scene (GCS, pupil size and reactivity, neurological deficits)
Examination	– ATLS protocol—identify immediate life-threatening injuries – Neurological status (GCS, pupil reactivity, pupil size, neurological deficits) – Head-to-toe survey for traumatic injuries – Avoid hypotension and hypoxia if possible

8.2 Differential Diagnosis

The patient in the current clinical scenario presents with a high-risk mechanism for traumatic brain injury. The presence of hypoxia and hypotension in the field suggests the likelihood of polytrauma. His presentation GCS of 5 defines him as *severe* TBI. A combination of intracranial traumatic pathology and the possibility of diffuse rather than focal injury should be presumed for patients presenting with a depressed level of consciousness. Neuroimaging will bring clarity to the array of possible intracranial findings, here summarized by involved anatomic compartment:

- *Extradural/epidural hematomas (EDHs)* occur in approximately 2% of all head injuries, are a result of direct impact, and usually present as isolated lesions without significant intraparenchymal swelling [8]. Typically, the source of bleeding is arterial, following a fracture in the region of the pterion with subsequent tearing of the middle meningeal artery and hematoma formation in the middle cranial fossa. Nevertheless, extradural hematomas may occur in other anatomical locations including in the frontal, occipital, and parafalcine regions—associated with injuries to the anterior ethmoidal artery, transverse or sigmoid sinuses, and superior sagittal sinus, respectively. EDHs originating from venous sources are thought to expand more slowly compared to their arterial counterparts (and, therefore, may present with a patient who "looks too good" for the size of the radiographic hematoma) [8, 9].
- *Acute subdural hematomas (ASDHs)* result from tearing of bridging veins that cross the subdural space to communicate with the venous sinuses or from disruption of superficial pial arteries on the brain surface. ASDHs can develop as a consequence of acceleration/deceleration injuries or direct impact or blow to the head. They are present in approximately a third of severe TBI patients and in two-thirds of patients undergoing surgery for TBI [10]. ASDHs are often associated with the presence of intraparenchymal contusions or hematomas and with a propensity for brain swelling [10–12].
- *Traumatic subarachnoid hemorrhage (tSAH)* is a frequent finding in closed head injuries, resulting from direct damage to cortical vessels. In patients with severe TBI, it is associated with other traumatic lesions and it may contribute to secondary injury (cerebral swelling and/or vasospasm) [4].

- *Intraventricular hemorrhage (IVH)* is found predominantly in severe TBI, in association with other extra-axial and intraparenchymal lesions. It results from damage to the septum pellucidum, choroid plexus, and subependymal veins in the fornix [4].
- *Cerebral contusions* result from forceful contact of the brain parenchyma with the internal bony prominences of the skull and occur in predictable locations, commonly on the antero-inferior aspect of the frontal lobes or at the temporal poles. Such injuries can be described as coup (same side of impact) or contrecoup (opposite side of impact). Initial CT imaging can underestimate their size, with ongoing bleeding occurring in the hours following the initial injury. Interval scanning can demonstrate blossoming of these injuries with hemorrhagic foci. They can contribute significantly to progressive brain swelling, intracranial hypertension, and secondary brain injury [4, 7].
- *Diffuse axonal injury (DAI)* results from shearing forces from rotational acceleration or deceleration that damages neuronal axons. Classically, DAI is defined as diffuse damage in the cerebral hemispheres, corpus callosum, brainstem, and cerebellum. Long-tract structures (axons and blood vessels) are especially at risk. It is more common with high energy injuries and often associated with other traumatic lesions such as a subdural hematoma. Signs of DAI may not be immediately visible on a CT in the acute phase. Brain MRI with diffusion weighted imaging and gradient echo sequences provides powerful tools to detect microbleeds and aid diagnosis of DAI [4].

8.3 Diagnostic Evaluation

Resuscitation and stabilization of the patient according to ATLS guidelines should succinctly be followed with diagnostic CT imaging. The dangerous injury mechanism and impaired consciousness are suggestive of intracranial pathology which necessitates neuroimaging [2].

Non-enhanced CT head remains the primary procedure for diagnostic imaging because of its sensitivity for detecting intracranial hematoma and the speed, availability, and safety of the examination [3]. It provides information about the morphology and extent of traumatic brain injury. In patients with moderate and, in particular, severe TBI, imaging is likely to show a combination of different lesions and diffuse rather than focal injury, as well as signs of increased intracranial pressure. In cases of penetrating head injury, cerebral angiography is recommended due to high risk of vascular injury. Computed tomography angiography (CTA) is an alternative, though interpretation may be limited by the presence of metallic streak artifact. There is no role for MRI in the initial clinical decision-making process, though MRI may play a role in further characterization of certain injuries (DAI, for example) once initial triage and acute management have been satisfied. MRI may not be feasible in the setting of retained metallic foreign bodies. CT imaging of the cervical spine, chest, and abdomen generally will be obtained by trauma staff in the course of evaluation for polytrauma.

In the current clinical scenario, CT head showed a thin left-sided acute subdural hematoma and left temporal intraparenchymal contusions, as well as contusions in both inferior frontal lobes; traumatic subarachnoid hemorrhage; and a small amount of intraventricular hemorrhage. There was left to right midline shift measuring 4 mm. There was no hydrocephalus (Fig. 8.1).

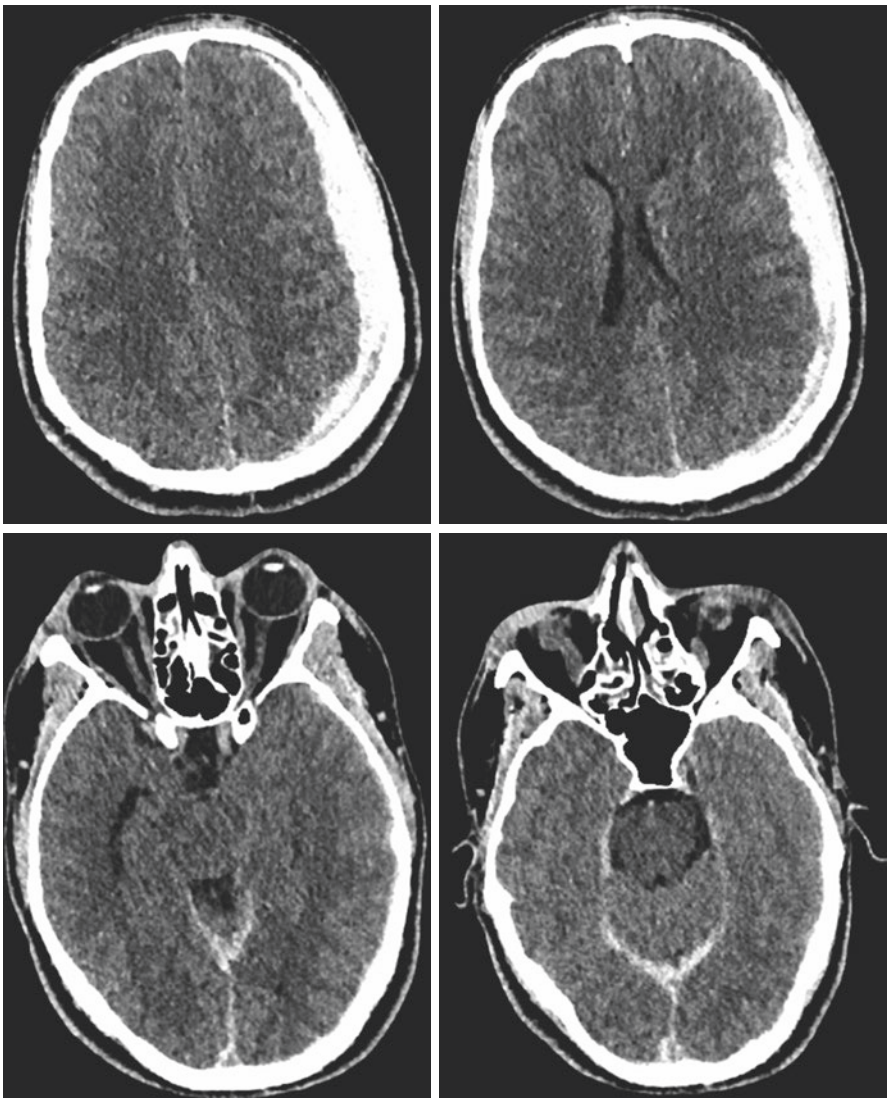

Fig. 8.1 CT head without contrast reveals a thin, left-sided acute subdural hematoma and left temporal intraparenchymal contusions, as well as contusions in both inferior frontal lobes; traumatic subarachnoid hemorrhage; and a small amount of intraventricular hemorrhage. There is left to right midline shift measuring 4 mm. There is no hydrocephalus

Basic laboratories—including BMP, CBC, PT/PTT, and a toxicology screen—should be performed coincident with initial clinical assessment. It is important to correct hypoglycemia, if present, as well as to identify and treat coagulopathy.

8.4 Clinical Decision-Making and Next Steps

The curious and disconcerting fact about TBI is that not all brain damage happens at the time of the traumatic event. Primary brain injury, which happens at the time of trauma, activates cellular and molecular cascades that mediate potentially reversible, secondary TBI in the ensuing hours and days. These events can lead to progressive brain swelling and increased intracranial pressure (ICP), thereby compromising cerebral perfusion pressure (CPP) and cerebral blood flow (CBF) and resulting in tissue ischemia, hypoxia, and cellular energy failure [7, 13, 14]. Management of TBI involves a combination of surgical procedures and medical measures. Clinical decision-making relies on the understanding of different TBI morphologies and their propensity to result in secondary brain injury and brain swelling and is helped by imaging and, where and when available, intracranial pressure monitoring.

Following initial assessment, a determination needs to be made as to whether the patient requires emergent cranial surgery. This decision must take into account the level of consciousness; pupillary size and reactivity; and review of imaging with attention to the presence and volume of extra-axial and/or intraparenchymal hematomas, as well as the degree of midline shift. Comorbid extracranial traumatic injuries—if deemed life-threatening and/or associated with hemodynamic instability—may take precedence in this setting. An ongoing dialogue with trauma staff (and possibly other subspecialist surgeons) may be necessary to coordinate the order of operations.

Surgical treatment would involve emergency craniotomy and evacuation of the hematoma, aiming to mitigate the injury caused by the space-occupying lesion and reduce intracranial pressure [15]. Decompressive craniectomy (DC) is a neurosurgical procedure that involves removal of a section of the skull ("bone flap") and opening of the underlying dura. From a physiological viewpoint, it provides additional space for the swollen brain to decompress, leading to reduction in ICP and maintained or improved cerebral compliance [15]. DC can be performed at the time of the initial craniotomy for removal of the traumatic extra-axial or intraparenchymal hematoma (**primary DC**), or as a treatment option for progressive and medically refractory secondary brain swelling (**secondary DC**) [15].

The decision whether to proceed to emergency surgery for a new TBI patient, as well as the choice of surgical technique (craniotomy versus primary decompressive craniectomy), will depend on the clinical severity of TBI, extent of injury, presence of a mass lesion amenable to evacuation, presence of brain swelling, degree of midline shift, and the propensity of the traumatic brain lesions for (further) swelling. Figure 8.2 reveals CT head findings for a patient with multifocal intracranial injury who underwent primary DC at the time of subdural hematoma evacuation. A decision about the need for post-procedural invasive intracranial pressure monitoring will also have to be made.

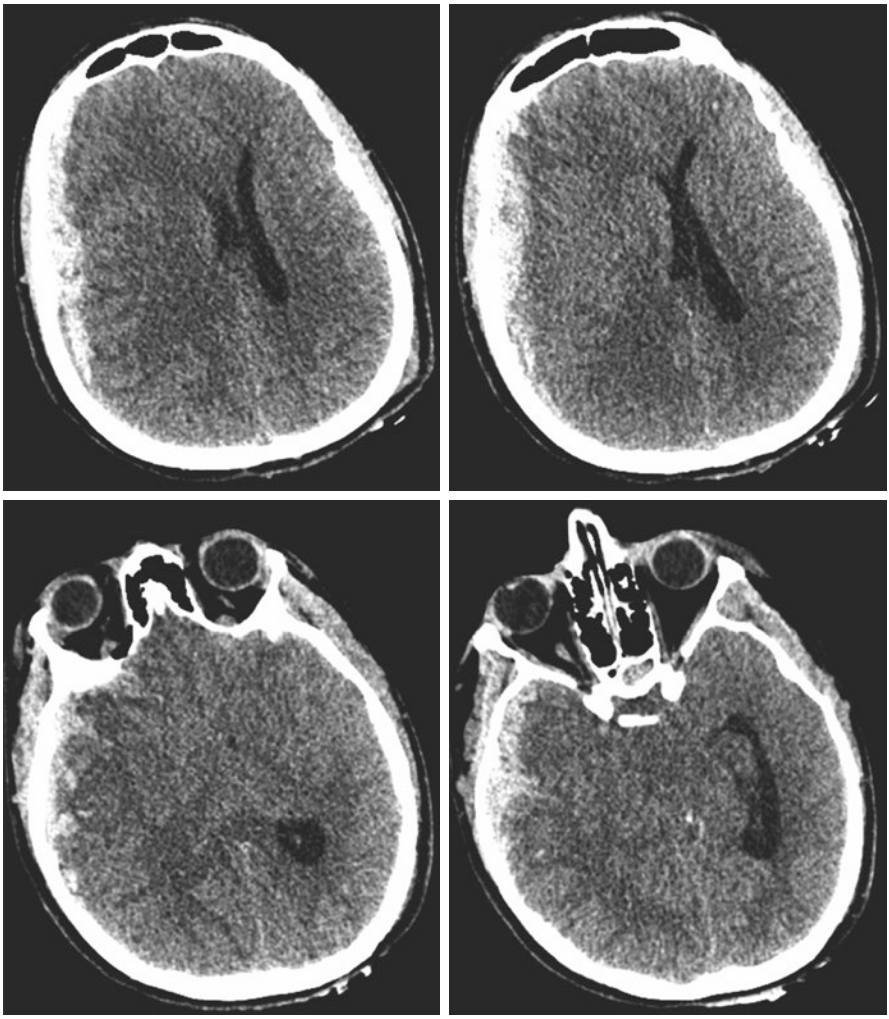

Fig. 8.2 CT head without contrast for a patient who underwent primary decompressive craniectomy. This 23-year-old female patient fell from a horse. Her GCS was 6 (E1V1M4) at the scene; the right pupil was dilated and the light reflex was lost. Imaging demonstrates a 13 mm right-sided acute subdural hematoma with multiple small contusions through the right frontal and temporal lobes, resulting in 9 mm midline shift and uncal herniation. The patient underwent emergency primary decompressive craniectomy and evacuation of subdural hematoma

If the imaging reveals an isolated extradural hematoma (EDH) , current guidelines recommend craniotomy and evacuation for all patients with an EDH volume of greater than 30 mL—regardless of GCS score—and in comatose patients (GCS 8 or less) with pupillary abnormalities [8]. Evidence on ICP trends following evacuation of isolated EDH shows that there is low risk of intracranial hypertension developing [16], suggesting that DC is not routinely required for treatment of isolated EDH [15, 16].

Acute subdural hematomas (ASDHs) , by contrast, are often accompanied by intraparenchymal contusions or hematomas and demonstrate a greater likelihood of secondary brain swelling [10–12]. Brain Trauma Foundation (BTF) guidelines recommend immediate operative intervention if ASDH thickness is more than 10 mm or midline shift is greater than 5 mm, regardless of the GCS score. Evacuation of ASDH is also recommended for severe TBI (sTBI) patients with hematoma thickness <10 mm and midline shift <5 mm if the GCS decreased by 2 points from injury to admission and/or if the patient presents with pupillary abnormalities and/or the ICP exceeds 20 mmHg [12].

There are variations in clinical practice around the world when it comes to ASDH evacuation, with some neurosurgeons performing primary DC more readily and more frequently than others. A recent consensus meeting on the role of DC in TBI recommends that primary DC should be performed following evacuation of the ASDH if the brain is bulging beyond the inner table of the skull intra-operatively; an ICP monitor may be placed, if available, for postoperative monitoring [15]. If the brain is relaxed following evacuation of ASDH and preoperative imaging is not in keeping with high risk of progressive brain swelling (such as for an elderly patient with involution brain changes and capacity to accommodate more brain swelling without a rise in ICP; or low energy mechanism of injury), the bone flap should be replaced [15]. Placement of an ICP wire intra-operatively for continuous ICP monitoring is recommended; in situations where continuous ICP monitoring is not available, serial CT imaging should be used to monitor progress [15]. For the intermediate category of ASDH patients (brain neither very relaxed nor bulging), surgeon judgment must be used to decide whether to leave the bone flap out or not. It is not clear if performing DC instead of replacing a bone flap in this clinical scenario provides any additional benefits for the patient; the results of the RESCUE-ASDH trial—a multicenter, pragmatic, parallel group randomized trial that aims to answer this question—are awaited [12, 15].

Intraparenchymal contusions and/or hematomas are often multiple and diffuse. The likelihood of perilesional cerebral edema is high. Current evidence and expert-based guidelines recommend operative intervention if hematoma volume is more than 50 mL, GCS score is 8 or less in a patient with a frontal or temporal hemorrhage more than 20 cm (>20 mL) with either midline shift of more than 5 mm and/or cisternal compression on CT scan [17]. The surgical approach may vary in this setting. We advocate craniotomy and evacuation of the hematoma or contusion(s). Others may elect to perform a decompression, without direct debridement of contusion. Primary DC is an option for comatose patients with diffuse contusions with signs of raised ICP on imaging if contusions are not being evacuated or, if following evacuation, the brain bulges beyond the inner table of the skull [15].

In cases of penetrating head injury, there are no clinical trials to date that specifically assess the role of DC. Practice is based on case series and has been driven by military experience. Brain swelling is often severe, and in these cases, intracranial hypertension can be relieved by a large DC [15].

The patient presented in our clinical scenario had severe TBI with a pre-intubation GCS score of 5. His pupils were small and reactive to light. His imaging showed diffuse head injury, but no lesions amenable to evacuation. There were no

indications for emergency cranial surgery; instead, an intracranial pressure monitor was inserted, and he embarked on a tiered intensive care TBI management (Table 8.2).

As mentioned earlier, secondary brain injury develops in the hours and days following primary brain injury and can lead to progressive and potentially dangerous brain swelling and intracranial hypertension. The burden of intracranial hypertension (the time spent with ICP above a defined threshold—usually 20–25 mmHg) is associated with excess mortality and worse functional outcomes [7, 13–15, 18].

ICP monitoring is performed ideally using an intraparenchymal microtransducer pressure probe inserted through a cranial access device or under direct vision at the time of craniotomy. Intraventricular catheters can be used; they allow therapeutic drainage of CSF, but are associated with a higher risk of complications, such as hematoma or infection, when compared to intraparenchymal probes [19]. In settings where invasive intracranial pressure monitoring is not available, non-invasive methods can be used according to available resources (for example, serial CT imaging) [15]. The goals of ICP control and preservation of CPP are pursued through the application of tier-based protocols employing neuroprotective measures such as sedation, controlled hyperventilation, therapeutic hypothermia, hyperosmolar therapies, barbiturate coma, and ventricular drainage [7]. Figure 8.3 illustrates the positioning of commonly employed invasive pressure monitoring devices.

Table 8.2 Key considerations in surgical decision-making

Factors necessitating consideration of emergency cranial surgery	– Neurological status (GCS, pupil size, and reactivity) – TBI morphology – Imaging findings (presence of lesions amenable to evacuation, midline shift, effacement of CSF spaces) – Presence and severity of extracranial injury
Factors influencing surgical approach (craniotomy vs primary decompressive craniectomy)	– TBI morphology—specifically, propensity for swelling of non-evacuated traumatic brain lesions – Intraoperative brain swelling

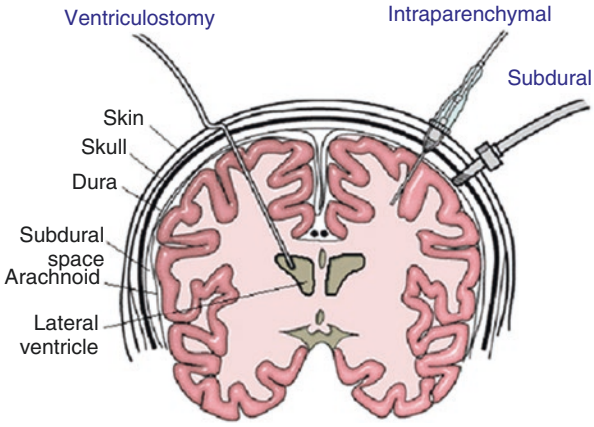

Fig. 8.3 ICP monitoring can be performed using subdural and intraparenchymal probes, as well external ventricular drains

A secondary DC can be undertaken as last-tier, life-saving therapy for patients with refractory intracranial hypertension (i.e., when all other measures have failed to reduce ICP) or as a second-tier therapy in patients with less pronounced elevation of ICP (i.e., as a neuroprotective measure) [7, 15]. DC is effective in reducing ICP and CPP, but the effects on functional outcomes are not straightforward [7, 15]. Two surgical techniques for DC are employed most commonly: bifrontal DC for diffuse injuries and unilateral frontotemporoparietal craniectomy (also termed hemi(spheric)-craniectomy or unilateral DC) for unilateral pathology with midline shift and swelling (e.g., ASDH with parenchymal injuries) [15].

Decompressive Craniectomy in Diffuse Brain Injury (DECRA)—an international, multicenter, randomized controlled trial—tested the utility of DC as an early neuroprotective measure [20]. Patients with severe, diffuse TBI were randomly assigned to either bifrontotemporoparietal DC or standard (medical) treatment if they developed intracranial hypertension—defined as ICP of more than 20 mmHg for more than 15 min in a 1-h period, refractory to first-tier therapies [20]. Patients in the DC group had better control of ICP and fewer days in the ICU. However, better ICP control did not translate into improved outcomes for the DC patients. Mortality was similar in the two treatment groups (19% in DC group and 18% in control group), and there was no improvement in functional outcomes in the DC group [20]. Therefore, current guidelines cannot recommend DC as an early neuroprotective measure. Rather, patients should be continued on the tiered intensive care TBI management [7, 15].

The Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intracranial Pressure (RESCUEicp) trial [21] aimed to examine the clinical and cost effectiveness of secondary DC (unilateral or bifrontal DC) as a last-tier therapy for severe TBI patients with refractory intracranial hypertension. Severe TBI patients with raised and refractory ICP (threshold 25 mmHg >1–12 h despite standard medical therapy) were randomized to ongoing medical therapy or secondary decompressive craniectomy. The results showed that decompressive craniectomy resulted in a marked reduction in mortality, with a concomitant increase in vegetative state, an increase in lower (dependent) and upper (independent at home for at least 8 h) severe disability, and similar rates of moderate disability and good recovery. Outcome improved between 6 and 12 months, with a significant proportion of patients in the surgical arm being upper severe disability or better [21].

A recent consensus meeting on the role of DC in the management of TBI has agreed that while secondary DC is a potentially useful operation, it should be applied selectively as there is uncertainty as to which severe TBI subgroups will truly benefit. It may decrease mortality but is associated with significant risk of complications and survival with severe disability; thus, frank discussions with family members/surrogates regarding the risks, benefits, alternatives, and potential prognosis are needed preoperatively. Both bifrontal and unilateral DC are options in the surgical treatment of diffuse TBI. The consensus group recommended a large DC (at least 12 × 15 cm) with durotomy to effectively reduce ICP and reduce incidence of secondary cortical injury from reduced venous drainage [15].

8.5 Clinical Pearls

- Primary DC involves removal of the bone flap at the time of initial evacuation of a mass lesion; the decision to leave the bone out depends upon intraoperative assessment of clinical findings and projected risk of intracranial hypertension.
- Secondary DC is usually employed as an end-stage measure when maximal medical management fails to control the ICP.
- DC is rarely indicated in the setting of EDH evacuation.
- DC carries a significant risk for morbidity.
- Clinical decision-making depends on the synthesis of several parameters (pathology, clinical examination, radiology findings, and ICP trends).

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