Review Article

Anesthetic considerations for extracranial injuries in patients with associated brain trauma

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Abstract

Patients with severe traumatic brain injury often presents with extracranial injuries, which may contribute to fatal outcome. Anesthetic management of such polytrauma patients is extremely challenging that includes prioritizing the organ system to be dealt first, reducing on-going injury, and preventing secondary injuries. Neuroprotective and neurorescue measures should be instituted simultaneously during extracranial surgeries. Selection of anesthetic drugs that minimally interferes with cerebral dynamics, maintenance of hemodynamics and cerebral perfusion pressure, optimal utilization of multimodal monitoring techniques, and aggressive rehabilitation approach are the key factors for improving overall patient outcome.

Keywords: Anesthesia, extracranial injury, intracranial pressure, traumatic brain injury

Introduction

Traumatic brain injury (TBI) is one of the most serious forms of trauma-induced injuries and is a leading cause of morbidity and mortality. Nearly, 80% of TBI are classified as mild with Glasgow Coma Scale (GCS) score of 13–15, 10% as moderate (GCS 8–12), and 10% as severe (GCS 3–7).[1] Patients with severe TBI often presents with extracranial injuries (23%–41%), which may result in poor outcome.[2-5] Various theories (e.g., second hit hypothesis) have elucidated that concomitant injuries may increase both peripheral and central inflammatory responses and exacerbate structural and functional deficits associated with TBI.[6,7] In fact, low GCS is also linked to severity of extracranial injuries.[7] The effects of concomitant injuries are heterogeneous and have been shown to vary with injury site and severity in adult trauma patients.[8] Whereas the vast majority of patients with mild TBI make a complete and uneventful recovery, a small proportion might deteriorate due to the development of potentially lethal intracranial bleeding and/or brain swelling, diagnosis and management of which may be delayed by anesthesia and surgery for extracranial procedures. Further, secondary insults such as hypotension, hypoxemia, hypercarbia, hypocarbia, hyperglycemia, hypoglycaemia, and hyperthermia during resuscitation and perioperative period might exacerbate the primary brain injury. As a result, it becomes prudent to institute simultaneous neuroprotective and neurorescue measures for optimal neurological outcome.

The primary goal in case of polytrauma is to transfer the patient to the nearest emergency department (ED) as early as possible. If required, the patient should be transferred to multispecialty trauma center after initial resuscitation and stabilization in the ED. Subsequently, the priorities of management must be identified and executed in an orderly fashion. It is clear, therefore, that only urgent surgery should be undertaken at this time. Thus, certain important questions

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need to be answered: (i) which surgery is urgent? (ii) which anesthetic techniques should be used? and (iii) for how long is the patient at risk? For patients with more serious head injuries, the problems are those of establishing priorities for a number of procedures which should seemingly all be carried out immediately but frequently, for practical reasons, have to be carried sequentially, in different departments or even in different hospitals. For such patients, what are the priorities? How should the patient be managed during and between the procedures? Availability of innumerable scoring systems for the assessment of the injured patient makes it imperative that the choice of the severity score accurately match the application. The commonly used scores in a polytrauma patient are abbreviated injury scale, injury severity score, and revised trauma score in addition to GCS. In this article, we have highlighted the management strategies of such issues based on recent advances and guidelines.

Resuscitation and Preoperative Preparation

The traditional approach to complete preanesthetic check-up is clearly not feasible in a patient with massive trauma. A brief history outlined by the mnemonic SAMPLE (Signs and symptoms, Allergies, Medications, Past medical/surgical history, Last oral intake, Events related to injury) along with relevant clinical examination (airway, breathing, circulation, neurological examination, and extracranial injuries) may be feasible and should suffice the necessary medical information.[9] Signs and symptoms of intracranial hypertension (ICH) or impending herniation, such as altered level of consciousness, pupillary dysfunction, lateralizing signs, extremity weakness, or Cushing's triad (hypertension, bradycardia, and irregular respirations) should alert the need for urgent interventions to control intracranial pressure (ICP). Of note, mild head injury or spinal injury is not associated with immediate life-threatening consequence. On the contrary, a ruptured liver or spleen requires urgent abdominal exploration.

Based on the high probability of progressive cerebral edema, TBI patients with GCS ≤8 will require urgent endotracheal intubation.[10] Moreover, due to impaired neurological status and depressed airway reflexes, tracheal intubation is deemed necessary to prevent aspiration. All TBI patients requiring intubation should ideally undergo rapid sequence induction (RSI). In addition, all TBI patients should be suspected to have coexisting cervical spine injury (CSI) unless excluded. The reported incidence of cervical spine trauma in head-injured patients has generally ranged from 4% to 8%.[11] Extreme caution should be exercised to prevent excessive neck manipulation during intubation. Established techniques such as manual in-line stabilization alone or in combination with video-laryngoscopes are often used. Flexible fibreoptic laryngoscopy does not seem to be feasible during emergency situations; however, it can be a useful resort in expert hands. Again, tracheal intubation should be smooth and gentle to prevent hemodynamic responses and an increase in ICP. Nasotracheal intubation is contraindicated in cases with basilar skull fractures.[12] Hypoxia and hypotension are independent predictors of morbidity and mortality and should be corrected promptly.[13] Blood loss from the ruptured viscus and diuresis resulting from mannitol administered to decrease ICP may contribute to hypovolemia. In such situation, hypertonic saline (HTS) is recommended to reduce ICP. The pertinent advantages of HTS include hemodynamic stability, attenuation of inflammatory cascade, and lesser/no rebound cerebral edema as compared with mannitol.[14] Furthermore, multisystem injuries without hemorrhage results in the release of vasodilating mediators causing relative hypovolemia and hypotension.[15] The role of anesthesiologist along with team of treating trauma surgeon and neurosurgeon is to take a concerted approach to management while continuing resuscitation.

The optimal time for surgical intervention is unclear and must be individualized according to the status of ICP, presence of intracranial mass, stability/instability of neurological state, and severity of associated injuries. The decision to move the patient urgently for surgery may be for systemic injury, head injury, or both. As part of emergency treatment protocol, ultrasound-based extended FAST (E-FAST) examination can rapidly rule out pneumothorax, hemothorax, peritoneal, and pelvic bleeding/hematoma without any risk of radiation.[16] Computed tomographic (CT) scan of the head and abdomen is useful in diagnosis; it helps in deciding about the lesion to be dealt first. Radiological examination of neck should also be performed as patients with substantial head injury might have associated CSI. Laboratory values especially hematocrit, blood group and Rh typing, coagulation profile, blood glucose, and arterial blood gas (ABG) analysis should be done on urgent basis. Thromboelastography (TEG) or rotational thromboelastometry (ROTEM) as a point of care test may be appropriate; the standard laboratory tests in the setting of trauma are of poor sensitivity and associated with a lag period. By the time investigation reports are available, concerned anesthesiologist should ensure adequate ventilation, optimal perfusion and systemic blood pressure (BP), normal blood sugar, electrolytes, and acid-base parameters. There are instances when enough time to obtain a CT scan due to severity of extracranial injury (splenic rupture/liver laceration) is not available. Under such scenario, rapid bedside, noninvasive estimation of ICP with the use of ultrasonography [e.g., optic

nerve sheath diameter, transcranial Doppler (TCD)] and near-infrared spectroscopy (NIRS) gives a fair idea of the coexisting raised ICP.

Neuroanesthetic Principles in Extracranial Surgeries

Anesthetic drugs

Intravenous (IV) and inhalational agents exert diverse effects on cerebral blood flow (CBF), cerebral blood volume (CBV), and $CMRO_2$. The IV agents including thiopental, propofol, and etomidate reduce $CMRO_2$, which results in cerebral vasoconstriction and a decrease in CBF, CBV, and ICP. However, thiopental and propofol may result in significant hypotension and a reduction in cerebral perfusion pressure (CPP).[17,18] Etomidate has limited effect on mean arterial pressure (MAP). Besides, it decreases CMR and hence ICP, with a net increase in CPP.[19] While there are no data examining ketamine as an induction agent in TBI, it has been shown to decrease ICP in painful interventions in the intensive care unit (ICU).[20,21] All the volatile anesthetic agents such as halothane, isoflurane, sevoflurane, and desflurane decrease $CMRO_2$ but at the same time also cause cerebral vasodilatation, thereby increasing CBF, CBV, and ICP. Halothane dilates cerebral vessels the most and thus is almost obsolete in neurosurgical practice. Other agents may be used with a minimum alveolar concentration of <1.[22] Furthermore, nitrous oxide (N_2O) increases CBF, $CMRO_2$, and ICP and should be ideally avoided in severe TBI.[23] N_2O should not be used prior to dural opening if there is suspicion of pre-existing intracranial air. There are no data demonstrating improved TBI outcomes based on the type of anesthetic agents; a balanced anesthetic technique is, thus, acceptable.

Either succinylcholine or rocuronium can be used to facilitate RSI. Although succinylcholine may increase ICP secondary to increased carbon dioxide production and afferent muscle activity, this effect is transient and may be offset by short-term hyperventilation. It should be borne in mind that the inability to secure a definitive airway poses far more risk than the transient increase in ICP.[24] Rocuronium (1.2 mg/kg) can also achieve rapid intubating conditions similar to succinylcholine, but it has longer duration of action. Although neuromuscular blocking agents decrease oxygen consumption and may transiently decrease ICP as they eliminate thoracic skeletal tone and thereby increasing venous drainage; their use during the postoperative period is generally not recommended.[25] Narcotics such as fentanyl or sufentanil can be used for supplemental analgesia but they may cause hypotension, which may result in compensatory vasodilatation and increase in ICP. Remifentanil, an ultra-short acting narcotic, should be used cautiously due to the frequent occurrence of profound reduction in BP and heart rate (HR). Dexmedetomidine, an alpha-2-adrenergic agonist, has favorable properties of conscious sedation, sympatholysis, analgesia, and lack of respiratory depression. Moreover, it has been shown to decrease ICP secondary to decrease in CBF and CMR, in addition to decreasing anesthetic and opioid requirements.[26,27]

Oxygenation and ventilation

Hypoxemia (PaO_2 <60 mmHg) linearly increases CBF, CBV, and ICP, and thus should be avoided. Addition of positive end-expiratory pressure (PEEP) improves oxygenation by recruiting collapsed alveoli[28]; however, high PEEP (>15 cmH_2O) therapy has been shown to increase ICP and compromise CPP in adults.[29] The tenet of perioperative ventilation in head injury patient is to maintain normocapnia. Both hypercapnia and hypocapnia have been shown to worsen perioperative outcome.[30] Although hypocapnia decreases CBF by causing vasoconstriction, concomitant reduction in $CMRO_2$ is not ensured and thus predisposes to cerebral ischemia. Hyperventilation with $PaCO_2$ not below 25 mmHg should be used judiciously during intraoperative period only for short-term control of ICP in patients at risk of developing herniation.

CPP and hemodynamic targets

Intraoperative hypotension (IH) during craniotomy for TBI is not common. Sharma *et al.* suggested the incidence of IH in adult patients during craniotomy to be as high as 65%. Independent risk factors for IH were multiple CT lesions, subdural hematoma, maximum CT lesion thickness, and anesthesia duration. IH was not affected by the choice of anesthetic agent.[31] There is widespread ambiguity regarding optimal CPP in TBI patients. Initially, the recommendation was to keep CPP >70 mmHg with vasopressors. However, a subsequent study suggested that outcomes were better with a lower CPP, possibly because of a reduced incidence of acute respiratory distress syndrome (ARDS) secondary to reduced vasopressor usage.[32] Vasopressors are commonly used to augment CPP in the setting of TBI, although data comparing these drugs are limited. Norepinephrine has more predictable and consistent effect on augmentation of CPP as compared with dopamine.[33] Moreover, norepinephrine increases the level of brain tissue oxygen and also significantly reduces the regional oxygen extraction fraction.[34] One single-center retrospective study of patients with severe TBI who received phenylephrine, norepinephrine, or dopamine reported that phenylephrine resulted in the maximum increase in MAP and CPP from baseline.[35] The 4th edition of the Brain Trauma Foundation (BTF) guidelines recommends maintenance of SBP at ≥100 mmHg for patients 50- to 69-year old and at ≥110 mmHg for patients 15- to 49- or >70-year old to

decrease mortality and improve outcomes (Level III). CPP should be maintained between 60 and 70 mmHg.[36]

Fluid and blood component therapy

Concerns regarding intraoperative fluid management depend on the type of fluid (crystalloids versus colloids), osmolarity of the fluid, and restrictive versus liberal approach fluid therapy. Vigorous fluid resuscitation is often necessary but may aggravate cerebral edema. Thus, fluid therapy should be targeted to maintain euvolemic and isotonic or mild hypertonic state and should be ideally goal-directed based on dynamic indicators of fluid responsiveness. Hyperglycemia, which worsens the consequences of cerebral ischemia, and hypo-osmolality, which can increase the brain edema, should be avoided. Thus, hypo-osmolar glucose containing solutions are avoided. Iso-osmolar crystalloids such as 0.9% normal saline and Plasmalyte A are recommended as the fluids of choice. Lactated Ringers' (LR) solution is relatively hypotonic to plasma (calculated osmolarity of 275 mosm/L but a measured osmolality of 254 mosm/L due to incomplete dissociation). As such, large volume of LR solution should be avoided so as to prevent increase in cerebral edema. In large volumes' resuscitation, a combination of isotonic crystalloids, colloids, and blood transfusion may be the best choice. Although anemia is consistently associated with worse outcomes among patients with TBI, transfusion of red blood cells to correct anemia is also associated with worse outcomes. Currently, there is insufficient evidence regarding the optimal hemoglobin (Hb) threshold for transfusion after TBI. Current clinical practice guidelines from trauma and critical care specialties recommend a target Hb of 7–9 g/dL.[37] The British Committee for Standards in Haematology similarly recommends a target threshold of 7–9 g/dL for patients with TBI, but for patients with evidence of cerebral ischemia, the Hb target increases to >9 g/dL.[38] Severe TBI has been associated with increased activation of various inflammatory mediators, which, in turn, leads to consumption of existing coagulation factors and activation of anticoagulation pathways. As such, early monitoring of the coagulation profile is warranted. In the setting of polytrauma, this resulting TBI coagulopathy can contribute to ongoing acute hemorrhage both within the brain and at other sites, thereby increasing blood product requirements necessary for adequate resuscitation. Depending on the coagulopathy, fresh frozen plasma, platelet-rich concentrates, cryoprecipitate, or isolated coagulation factors can be transfused. Recombinant factor VIIa (rFVIIa) has been used off-label as an adjunct in the reversal of warfarin therapy and management of hemorrhage after trauma. However, some studies have failed to show a clinical benefit of rFVIIa in early management of TBI.[39] In addition, it has been shown to negatively impact recovery and functional status at discharge in the severely injured patient with polytrauma.[40] Intra-abdominal packing to control hemorrhage may result in a rise in ICP and should be avoided in these patients. Tranexamic acid is an antifibrinolytic drug and has been shown to decrease mortality in adult trauma patients (The Clinical Randomization of Antifibrinolytics in Significant Hemorrhage [CRASH-2] trial) and currently is being investigated in patients with TBI (CRASH 3 trial).[41] Erythropoietin (EPO) is generally effective in treating anemia and is relatively more effective in treating end-stage renal disease or hematological diseases. In addition, it has neurocytoprotective, anti-inflammatory, and anti-apoptotic actions.[42] A recent *post hoc* analysis of the EPO-TBI trial revealed that EPO administration was associated with a potential differential improvement in 6-month mortality in TBI patients with more severe extracranial injury.[43] In a recent meta-analysis (6 RCTs, 1,041 patients), the authors observed that EPO significantly reduced the occurrence of mortality but did not significantly reduce poor functional outcome. There were no significant differences in the occurrence of complications, such as deep vein thrombosis, between the treatment groups.[42]

Glycemic control

Hyperglycemia worsens neurological outcome after TBI.[44,45] Age ≥65 years, severe head injury (GCS <9), preoperative hyperglycemia, and acute subdural hematoma are independent predictors of intraoperative hyperglycemia.[46,47] Causes of hyperglycemia after TBI include an increase in gluconeogenesis and glycogenolysis from catecholamine response, cortisol release, and glucose intolerance. As such, dextrose containing fluids should be avoided except in cases of established hypoglycaemia. On the contrary, aggressive treatment of hyperglycemia should also be done with caution due to the potential for an increased risk of hypoglycemia and its potentially devastating neurological consequences if unrecognized and untreated.[48] Given the potential impact of both hyperglycemia and hypoglycemia, intermittent monitoring of blood glucose concentrations during intraoperative care is suggested.

Management of ICH

Updated BTF guidelines state that ICP >22 mmHg should not be left untreated. The management of increased ICP includes a stepwise approach that comprises of preventing excessive neck flexion or rotation, maintaining slight head elevation, ensuring appropriate sedation and analgesia, avoiding hypercapnia, allowing intermittent CSF drainage through external ventricular drain (EVD), administering osmotherapy with mannitol or hypertonic saline, and instituting mild hyperventilation for short-term control of ICP. Refractory ICH might require decompressive craniectomy (DC), therapeutic hypothermia, or barbiturate coma. Latest trials

have shown that though DC decreases ICP and the length of stay in the ICU but is associated with more unfavorable outcomes on extended Glasgow Outcome Scale (GOS-E) at 6 months.[49,50] During transportation of patients to and from the OR, it is important to individualize decision to travel with EVD open versus closed to CSF drainage. If traveling with EVD clamp, ensure clamping at both proximal port on EVD and distal port on CSF collecting system.

Positioning

A slight head up (30°) position is advocated if it does not interfere with the surgical procedure and is hemodynamically tolerated by the patient. However, elevated head position may compromise the surgical management of intra-abdominal injuries. Thus, the optimal head position varies from patient to patient, and even from time to time in an individual. Care should be taken to avoid hyperflexion, hyperextension, extreme lateral flexion, and rotation of the head to prevent obstruction of cerebral lymphatic and venous outflow. Impairment of cerebral venous outflow can cause intraoperative cerebral edema, increased ICP, ischemia, and cerebral infarction.[51] Other complications related to positioning includes pressure sores and injuries to peripheral nerves and major nerve plexus. Proper eye-padding is important to avoid exposure keratitis. All the bony prominences should be properly padded to prevent pressure sores.

Temperature management

Induced hypothermia causes reduction in $CMRO_2$, CBF, and ICP. However, therapeutic advantage of hypothermia is offset by complications, such as hypotension/hypertension, bradycardia, arrhythmias, hyperglycemia, sepsis, coagulopathy, and rebound increase in ICP during and after rewarming.[52,53] Early (within 2.5 h) and short-term (48-h postinjury) prophylactic hypothermia is not recommended to improve outcomes in patients with diffuse head injury.[36] Importantly, hyperthermia should be avoided because it causes increase in cerebral metabolic demand, lipid peroxidation, inflammation, excitotoxicity, and may lower seizure thresholds.[54] Thus, the current practice is to maintain normothermia.

Perioperative steroids

A number of studies in adults including the large multicenter MRC CRASH (Medical research council; corticosteroid randomization after significant head injury) trial have shown no benefit with the use of steroids in TBI.[55] Complications such as adrenal suppression, hyperglycemia, increased risk of infection, and gastrointestinal bleeding attributed to steroid administration have been shown to worsen outcome. The 4th BTF guidelines recommend against the use of steroids for improving outcome or reducing ICP.

Seizure prophylaxis

Post-traumatic seizures (PTS) are a major cause of secondary brain injury following TBI and are associated with higher injury severity and worse outcomes.[56] PTS affect patients through multiple mechanisms: by increasing or prolonging cerebral hypoxia, increasing release of excitotoxic neurotransmitters, increasing $CMRO_2$ and ICP, and by causing fluctuations in systemic BP.[57,58] Recent data suggest that seizures occur in up to 20% of patients with TBI. These seizures are usually nonconvulsive in nature and cannot be detected clinically, making continuous electroencephalography (cEEG) recordings a vital tool in diagnosis and management.[59] Prophylactic administration of phenytoin has been shown to reduce the incidence of early but not late PTS after 7 days of injury.[60,61]

Antibiotic prophylaxis

Penetrating TBI might cause local wound infection, meningitis, ventriculitis, or cerebral abscess formation. The risk of systemic infection is much higher in severe extracerebral injuries and is associated with higher morbidity and mortality.[62] Broad spectrum antibiotic prophylaxis is universally recommended to be started as early as possible in all TBI cases with extracranial injuries and continued for at least 6 weeks.[63]

Thromboprophylaxis

Patients with TBI are at significant risk of experiencing thromboembolic events.[64] The risk is further multiplied in concomitant CSI, lower limb long bone, and pelvic bone fractures and patients on mechanical ventilation. Options for prevention include mechanical (graduated compression stockings or intermittent pneumatic compression), pharmacological (low-dose or low-molecular-weight heparin) prophylaxis, or a combination of both. Pharmacological thromboprophylaxis is usually initiated 48–72 h after surgical intervention and in the absence of other contraindications.[65]

Multimodal Monitoring

Routine monitoring should include non-invasive BP, ECG, percentage of oxygen saturation (SpO_2), end-tidal carbon dioxide ($EtCO_2$), temperature, and urine output measurements. Direct arterial monitoring is used to achieve beat-to-beat control of BP so that extensive swings can be treated quickly. When monitoring an arterial line, one must zero the transducer at the level of the right atrium or phlebostatic axis. However, when calculating CPP during the management of TBI and in sitting position surgeries, the arterial transducer should be positioned at the level of the external auditory meatus (corresponding to the level of Circle of Willis).[66] Central venous pressure (CVP)

monitoring allows more rational approach to fluid replacement, particularly when osmotic diuretics are used. ABG should be performed to confirm levels of acid-base, carbon dioxide, hemoglobin or hematocrit, and electrolytes. Special emphasis should be placed on $PaCO_2$ monitoring as a means of assessing the level of hyperventilation. The pupils should be monitored for anisocoria, if the face is accessible. ICP monitoring should be done in comatose patients due to severe head injury (GCS <9) with abnormal CT scan or in patients with normal CT scan with two of the features, such as age >40 years, motor posturing, or SBP <90 mmHg.[36] The placement of intraventricular catheter allows accurate measurement of ICP as well as therapeutic drainage of CSF if required. Routine ICP monitoring in mild to moderate head injury is not recommended; however, case-by-case assessment may be warranted. Other neurological monitoring techniques such as TCD, jugular venous oximetry ($SjvO_2$), brain tissue oxygen tension ($PbtO_2$) monitoring, NIRS, and cerebral microdialysis are being used in TBI patients, but their use in polytrauma patients is yet to be established. $SjvO_2$ monitoring allows detection of episodes of desaturation associated with raised ICP. The normal range of $SjvO_2$ is 60%–70% and episodes of $SjvO_2$ <50% in severe TBI are commonly due to ICH and systemic causes, such as hypoxia, hypotension, and pyrexia.[67] $SjvO_2$ monitoring also aids in therapeutic hyperventilation. Brain $PbtO_2$ monitoring is useful in providing a focal measurement of cerebral oxygenation and critical perfusion thresholds and can warn against impending ischemia associated with hyperventilation. The normal brain $PbtO_2$ usually ranges from 20 to 35 mmHg and the ischemic threshold ranges from 10 to 15 mmHg.[68] Literature also supports the use of continuous EEG monitoring in TBI patients due to high incidence (33%) of seizure activity resulting in prolonged elevation in ICP and signs of *in vivo* metabolic stress.[69] Other neuromonitoring tools, such as somatosensory-evoked potentials and motor-evoked potentials have a complementary role, surveying the integrity of the neural tracts as an indicator of prognosis or illness progression in both acute brain and spinal injuries.

Considerations for Specific Surgeries

Abdominal surgeries

In polytrauma patients, there can be generalized increase in multiple compartmental pressures. Elevated intra-abdominal pressure (IAP) not only increases ICP secondary to the increase in intrathoracic pressure and CVP but also decreases cardiac, renal, hepatic, and gastrointestinal functions.[70] In a prospective study by Citerio *et al.*, patients with moderate to severe head injury showed an increase in ICP through placement of an external water bag (15 L) on the patient's abdomen.[71] Typically, intra-abdominal hypertension (IAH) is defined as IAP >12 mmHg. Abdominal compartment syndrome (ACS) is defined as IAP >20 mmHg [with or without an abdominal perfusion pressure <60 mmHg] associated new onset organ dysfunction/failure.[72] The World Society for the Abdominal Compartment Syndrome (WSACS) has developed the stepwise management of IAH and ACS. In refractory ACS, decompressive laparotomy (DL) is recommended in both TBI and non-TBI patients.[72] In a retrospective analysis of 102 patients with severe TBI, both DC and DL were done in 24 patients with a mean IAP of 28 mmHg. Mean ICP significantly decreased from 28 to 19 mmHg.[70] Similarly, Miglietta *et al.* reported two cases of refractory ICH with raised IAP who were treated successfully by abdominal compartment decompression.[73] However, to perform DL in IAH without ACS to reduce ICP in TBI patients is still a matter of debate. Laparoscopy, though minimally invasive, can increase ICP secondary to abdominal insufflation and IAH and thus should be used cautiously, in patients who present with baseline elevated ICP.[74] The use of gasless laparoscopy has not been shown to increase ICP in animal model and can be the other option to be considered in the near future.[75] Head down tilt which is a frequent requirement in many exploratory laparotomies should also be minimized to prevent aggravation of raised ICP.

Orthopedic surgeries

There is no concrete evidence to support either early or delayed fixation of long bony fractures in patients with head injuries.[76] Clinical factors that determines the appropriateness of early long-bone stabilization include severity of the brain injury (GCS, CT scan, ICP), severity of pulmonary dysfunction (PaO_2/FIO_2, lung compliance, positive end-expiratory pressure), and evidence of hypotension. Compound fractures can be debrided and cleaned during resuscitation and stabilization. Fixation of the long-bone fracture can be delayed until physiological parameters are stabilized. Preferably, long-bone fixation should be undertaken after 48–72 h. Skeletal traction and Thomas splint improves anatomical alignment and functional results. These can be used in the interim period until fracture fixation is undertaken. Induced hypotension is sometimes requested by orthopedic surgeons during spine stabilization procedure in patients with intact spinal cord function or fixed cord injury. Many of these patients suffer concomitant head injury, and the cerebral circulation may behave abnormally. Certainly, induced hypotension is contraindicated in patients with known head injury. But even in patients who have only suffered a brief concussion, the use of this technique remains hazardous because the autoregulatory capacity may still be impaired.

Maxillofacial surgery

The incidence of head injuries associated with maxillofacial trauma has been reported to be as high as 67% and the most frequent maxillofacial injury is the fractured mandible.[77] Upper facial dislocation and zygomatic–orbital–maxillary complex fracture significantly more often co-exists with skull, dura mater or cranial nerve injuries, and zygomatico-orbital fracture with the injuries of the brain.[78] The number of facial fractures has also been shown to be inversely related to GCS.[77] Early fixation of facial fractures is associated with better esthetic results. However, principles similar to those with long bone fractures apply in this situation and maxillofacial surgery can be delayed till optimization of ICP. A conservative approach is reasonable in simple fractures, and displaced fractures can be managed with miniplate and screw fixation.[79] In severe injury, the airway remains the priority; however, low GCS and longer duration of surgery have been associated with poor overall outcome.[80]

Role of Regional Anesthesia

With certain limitations of GA, a regional technique may seem preferable in a patient with a recent head injury. However, these techniques have their own disadvantages.

Epidural anesthesia

Standard epidural injections of 5 or 10 mL of anesthetic solutions (or 0.9% saline) produced a substantial rise in ICP in two patients who had suffered a head injury more than a week previously, explicable as the effect of compression of the dural sac shifting CSF back into the intracranial compartment.[81] It was concluded that epidural anesthesia should be used with extreme caution in patients with decreased intracranial compliance. The dramatic effect of epidural injection on ICP in the presence of an intracranial mass lesion has also been demonstrated in an animal model.[82] Tentorial herniation can also occur if dura is punctured. If epidural anesthesia is used, injections should be of small volume and made very slowly. A patient receiving anti-thrombotic or thrombolytic therapy should have cautious administration of neuraxial anesthesia. A drug-free period is often required prior to and after administration of neuraxial blockade and/or catheter placement. This time period has been recently updated (Fourth Edition) by the American Society of Regional Anesthesia and Pain Medicine (ASRA), in conjunction with the European Society of Anaesthesiology (ESA).[83]

Spinal anesthesia

Despite the standard teaching that spinal anesthesia is contraindicated following head injury because of the risk of precipitating tentorial herniation, there are several circumstances where lumbar puncture has been widely used in patients with reduced intracranial compliance without apparent adverse effect: to diagnose subarachnoid hemorrhage and intracranial infections,[84] and in the management of both benign ICH and refractory increase in ICP in severely head injured children.[85] A patient who has suffered a mild head injury without clinical manifestations including normal CT scan is unlikely to be at risk from a spinal anesthetic, but this conclusion cannot be made in the absence of a CT scan. However, there are no studies in literature on this aspect of anesthesia in head injured patients.

IV Regional Anesthesia and tourniquets

There are several case reports on the effect of ICP in head-injured patients of deflating arterial tourniquets applied to the lower limbs to improve operating conditions.[86-88] In each case, tourniquet release was followed by a rise in ICP which, together with the normal and expected fall in BP which occurs at this time, resulted in a substantial fall in CPP. As ICP was being monitored in each case, immediate measures could be taken to correct the situation and none of the patients apparently suffered any sequel. Tourniquet release has been shown to significantly increase end tidal carbon dioxide tension, an indirect monitor of plasma carbon dioxide tension.[89] The effect of releasing an arm tourniquet is, of course, likely to be smaller. Another potential danger from the use of tourniquets is the possibility of sudden severe bleeding when the tourniquet is released and this, in conjunction with the rise in ICP, can produce significant effect on the neurological outcome.

Peripheral nerve blocks

In contrast to the potential problems with epidural, spinal, or IV regional anesthesia techniques, local nerve blocks seem to be a good choice for providing anesthesia in patients with head injury. An additional problem which is common to all regional techniques, however, is the question of sedation. Patients with a recent injury may be uncooperative, but their response to sedation may be unpredictable, and if respiratory depression or airway obstruction occurs, the result could be hazardous.

Peripheral nerve blocks (PNBs) such as digital or ankle blocks or blocks of a major plexus such as brachial, sciatic, or femoral may be acceptable as long as conscious sedation is maintained. Care must be taken to avoid a local anesthetic induced seizure. Ultrasound-guided nerve blocks improve precision and accuracy. PNBs are being increasingly used for management of postoperative pain. They are administered either as a single injection or continuous infusion via a perineural catheter.[90]

Emergence and Recovery

Patients with severe head injury usually need continuing postoperative care in the neurointensive care unit, and their

trachea should not be extubated at the end of the procedure. In patients with mild to moderate head injury, it is not unreasonable to allow the patient wake up at the end of the procedure. Care must be taken to prevent excessive coughing and bucking that may cause transient rise in ICP with increased risk of venous bleeding. Continuation of care in ICU is directed toward enhancing the healing process and preventing or treating any complication. Appropriate analgesia and sedation should be ensured to prevent deleterious complications of pain. Optimization of BP, glucose levels, temperature, electrolytes and acid-base status, correction of coagulation abnormalities, and optimal nutritional support should be the utmost concerns. A multidisciplinary rehabilitation approach should be employed to promote early recovery and facilitate a smooth transition to positive long-term outcome.

Conclusion

TBI with concomitant extracranial injuries presents a myriad of challenges. Management of such patients requires thorough understanding of neuroanesthetic considerations. Selection of anesthetic drugs that minimally interferes with cerebral dynamics, maintenance of hemodynamics and CPP, optimal utilization of multimodal monitoring, and aggressive rehabilitation approach are the key factors for improving overall patient outcome.

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