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Review Article

ICU management of Traumatic Brain Injury (TBI)

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Abstract:

Traumatic brain injury (TBI) can be defined as the disruption in brain function, or other evidence of brain pathology, caused by an external physical force. Management of TBI depends on, if the injury is focal or diffuse. Focal can be epidural or intra cerebral and diffuse lesions can present as multiple contusions/DAI.

A basic understanding of anatomy of central nervous system and some working knowledge of physiology of brain help in management of TBI. According to severity there are three types of TBI patients. They are: mild (GCS 13-15), moderate (GCS 9-12) and severe (GCS 3-8). TBI may be primary or secondary. Primary injuries include contusion, hematoma, subarachnoid hemorrhage, diffuse axonal injury etc. Secondary injuries can manifest as cerebral oedema, raised intra cranial pressure etc.

Airway management and cervical spine immobilization are important early steps in management. Hypoxia and hypercarbia must be avoided and attention is always paid to ventilation status of patients. Mean arterial pressure (MAP) should be monitored and maintained at 80-90 mm of Hg. Intracranial pressure (ICP) should be checked whenever feasible and monitored so raised ICP can be controlled. Adequate pain control and sedation, using mannitol, hypertonic saline should be used as needed to control raised ICP. Invasive ICP monitoring and cerebrospinal fluid diversion should be used in patient who decompensate and decision to do decompressive craniectomy is often made by neurosurgeons. Body temperature in TBI patients should be maintained under 37^{o} C. Neurologic conditions like brainstem dysfunction, intracranial hypertension, altered level of consciousness etc. and respiratory conditions like ARDS, hypoxaemia , neurogenic pulmonary oedema etc. warrants endotracheal intubation and mechanical ventilation. TBI patients requiring haemo dialysis usually need special modification in protocol to avoid pulmonary oedema and fluctuation in blood pressure.

Monitoring TBI patients is done by routine assessment of GCS, pupillary size and reaction, motor responses. ICP monitoring is to be done if feasible. By aggressive monitoring secondary brain injuries can be avoided or managed.

Outcome of management of TBI patients in ICU depends on severity of primary and secondary injuries of brain, status of GCS on presentation, advanced age (>65yrs), presence or absence of co morbidities and severity of associated injuries.

Keywords: *Traumatic Brain Injury (TBI), ICU, Management.*

INTRODUCTION

Traumatic brain injury (TBI) can be defined as the disruption in brain function, or other evidence of brain pathology, caused by an external physical force[1]

Critical care management of TBI is influenced by factor like mechanisms of injury whether it is blunt or penetrating. Severity of injury whether it is minor, moderate or severe, dictates its management according to severity. Similarly focal lesion like epidural, subdural or intra cerebral collections and diffuse lesions like concussion, multiple contusions and hypoxic ischemic injury of brain require specific guidelines for the management. It is also important to consider morphology of skull fractures during evaluation and management of TBI.

Neurocritical care specialists as well as general intensive care unit (ICU) specialists involved with care of TBI patients need to have basic understanding of anatomy of brain, meninges with different compartments of brain and ventricular system and some working knowledge of physiology of brain pertaining to intracranial pressure (ICP) and cerebral blood flow (CBF).

ICU management requires close cooperation between neurosurgical team and neurocritical care team. The following issues are routinely considered initially during ICU/ emergency management of TBI.[2]

a) Age of the patient and mechanism and time of injury.

b) Respiratory and cardiovascular status particularly blood pressure and oxygen saturation.

c) Neurological examination consisting of the Glasgow coma scale (GCS) score with particular emphasis on the motor response and assessment of pupil size and reaction to light.

d) Presence and type of associated injuries.

e) Results of diagnostic tests like CT scan of brain.

f) Treatment of hypotension and hypoxia.

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Admission of TBI patients in ICU is usually guided by its severity. Mild types (GCS score 13-15) usually need admission into hospital but do not need ICU admission unless a coexisting condition makes it necessary. Moderate TBI (GCS score 9-12) are often admitted into ICU for observation and concomitant management. Severe TBI (GCS score 3-8) or coma patients are either admitted into ICU for early resuscitation and care or this group of patients or are usually referred back to ICU following definitive care in operating room.

Traumatic brain injury requires an understanding of two phenomenon e.g. Primary and Secondary brain injuries[3]. The primary injuries include contusions (bruises to brain parenchyma), hematomas (subdural, epidural, intra parenchymal, intra ventricular and sub arachnoid), diffuse axonal injury (stress or damage to axons), direct cellular damage (neurons, axons and other supportive cells), loss of blood brain barrier, disruption of the neurochemical homeostasis and the loss of electrochemical function. The secondary brain injuries are unleashed by the impact that results in a series of deleterious cellular and subcellular events (also known as the secondary neurotoxic cascade). Manifestations of secondary brain injuries are cerebral oedema, increased ICP, mitochondrial dysfunction leading to brain cell death, oxidative stress, loss of cerebrovascular auto regulation and carbon dioxide reactivity, cerebral metabolic dysfunction etc.

Principles of management of severe TBI (including moderate TBI) in ICU aims at avoidance of secondary injury[4]. Primary causes of secondary brain injury comprise hypotension, hypoxia, hypercarbia, and worsening ICP. A regimented and organized approach are needed when TBI patient is seen in emergency and later followed in the ICU.

AIRWAY AND CERVICAL SPINE ISSUES

Severe TBI by definition has depressed level of consciousness. Such patient has to be intubated for airway protection and to ensure proper oxygenation and ventilation[5]. When endotracheal intubation is not feasible and when basic airway maneuvers like jaw thrust fails, supraglottic airway devices should be considered. Such maneuvers often raise ICP thereby reducing cerebral blood flow potentially compromising cerebral perfusion pressure (CPP). To prevent significant rises in ICP patient should be sedated and paralyzed using standard doses of rapid sequence intubation drugs. A hemodynamically neutral induction agents such as ketamine or etomidate is preferred as sedating agents to prevent transient drops in CPP[6].

Cervical spine immobilization should be maintained as brain injured patients have been shown to have concomitant cervical spine injury [7].

BREATHING AND VENTILATION ISSUES

Hypoxia is a very important cause of secondary brain injury. And can lead to cerebral oedema. Oxygenation should be maintained greater than 90% with supplemental oxygen if needed[5] and PaO2 should be maintained above 60 mm of Hg. Hyperoxia should be avoided[8]. A reasonable strategy is to provide the smallest amount of supplemental oxygen necessary to avoid hypoxia.

Ventilation and CO2 management are equally important in patients with TBI, as hypercarbia can result in cerebral vasodilatation with subsequent development of cerebral edema, and therefore a reduction of CPP. PaCO2 measured by blood gas analyzer or by end tidal CO2 detector should be kept between 35 -40 mm of Hg. If there is concern for acute worsening of ICP or development of herniation syndrome, the patient can be briefly hyperventilated to decrease the PaCO2 to achieve a PaCO2 level of 30-35 mm of Hg. This is a temporizing measure and should be limited to 15-30 min[9]. Long term cerebral vasoconstriction from hyperventilation will eventually lead to cerebral ischemia, increased ICP and worsened neurologic outcome.

HEMODYNAMIC MANAGEMENT

Maintaining hemodynamic stability in severely brain injured patient is very important as hypotension is a major cause of secondary brain injury. CPP is the key marker for adequate cerebral blood flow and is calculated by subtracting ICP from mean arterial pressure (MAP). Maintenance of normal MAP with goal of 80-90 mm of Hg or maintaining systolic blood pressure (SBP)> 110 mm of Hg is critical in maintain adequate CPP[10]. Transient hypotension can contribute to worse outcomes. Even a single episode of hypotension (SBP < 90 mm of Hg) can increase mortality by 50%[11]. Continuous blood pressure monitoring with invasive blood pressure monitoring such as an arterial line should be used if available in ICU.

Isotonic fluids and vasopressors should be used judiciously as first line to maintain hemodynamic stability. Blood products need to be used for trauma resuscitation as required. Hypotonic and dextrose containing fluid should be avoided as they can worsen cerebral oedema[12]. Up to one third patients of severe TBI often develop a coagulopathy making identification of occult injury more important as such it can markedly worsen neurologic and trauma outcomes[13].

MANAGING NEURO DISABILITY

All TBI patients with post traumatic seizure or CT finding of oedema , intracranial hemorrhage or mid line shift should get a bolus of available anticonvulsant drug like phenytoin (15 mg /kg) followed by maintenance dosing[14].

As hypoglycemia is associated with poor outcome it is advisable to check and correct glucose level as part of initial survey for a TBI patient with altered mental status.

Degree and duration of elevated ICP in severe TBI correlates closely with worse neurologic outcome[15]. All severe TBI patients should be positioned with head of bed raised to 30°C to facilitate venous return from brain. Loosening the cervical collar and endotracheal tube ties, as much as safety will allow can decrease external pressure on cervical venous outflow tracts[5].

Early and aggressive pain control and sedation should be a priority to prevent spikes of ICP. Anxiolytics or pain

medications that have less effect on hemodynamics (including fentanyl and long acting benzodiazepines like diazepam) may reduce iatrogenic harm from cerebral hypo perfusion. Continuous hemodynamic monitoring is needed for all sedated patients. It should be kept in mind that over sedation can make serial neurological examination more difficult to follow. One study noted that bolus dosing of thiopental and propofol can effectively control agitation particularly during uncomfortable procedures[5]

Despite adequate sedation and analgesia TBI patients may have progressive neurological decompensation and such patients will need hyperosmotic therapy. Mannitol (0.5-1.5g/Kg /dose) and 3% hypertonic saline (150-250 ml /dose) both effectively pull excess fluid from brain parenchyma and reduce ICP[10]. Administration of mannitol results in osmotic diuresis with resultant decrease in blood pressure and therefore reduction of CPP. For patient in whom hypotension is a concern and hypertonic saline is not available, sodium bicarbonate is a viable alternative and has been shown to effectively reduce ICP for up to six hours[16].These therapies are given as temporizing measure until definitive care for TBI is maintained in ICU with serial neurological monitoring.

Invasive ICP monitoring and cerebrospinal fluid diversion can be utilized for patients who decompensate or whose conditions are refractory to conservative therapy[10]. The decision to do decompressive craniectomy is best determined by experienced neurosurgical consultants.

MAINTAINIG BODY TEMPERATURE

Hyperpyrexia promotes cerebral metabolism and core temperature should be maintained below 37°C with paracetamol or cooling devices. At the same time hypothermia in TBI is not therapeutic and potentially harmful in coagulopathic poly trauma patients[10]. Unmonitored cooling with wet sheets and fans on exposed patients should be avoided.

TBI REQUIRING MECHANICAL VENTILATION

Ventilatory management in TBI patients is challenging. Principles of lung protection and brain directed therapies are in direct conflict[17]. Management of brain injured patients is complicated by the high incidence extra cerebral complications in particular pulmonary, making ventilator management extremely challenging[18]. A significant number of brain injured patients (about 20%) require endotracheal intubation and ventilation[19].

Indications for endotracheal intubation and mechanical ventilation in TBI can be described under headings of neurologic and respiratory indications[17].

Neurologic indications are the following.

- Altered level of consciousness /airway protection.
- Brainstem dysfunction
- Intracranial hypertension.
- Anticipated neurologic deterioration.

Respiratory indications are as follows.

- Hypoxaemic respiratory failure due to aspiration, pneumonia, atelectasis, pulmonary embolism and ventilator associated pneumonia.
- Acute lung injury/acute respiratory distress syndrome (ALI/ARDS). It is an independent predictor of poor outcome in the setting of brain injury. Early ALI/ARDS occurs at day two to three and late occurs at day seven to eight post initiation of mechanical ventilation.
- Neurogenic pulmonary edema. Mechanism for this is not clearly understood.

Current practice guidelines for ventilator management advocate protective lung strategies to prevent volutrauma, barotrauma, atelectrauma and bio trauma[20].The principles are to use low tidal volume (Vt) 5-6 ml /kg ideal body weight, maintenance of low mean airway pressure ≤ 30 cm of H2O, judicious use of positive end expiratory pressure (PEEP), higher respiratory rates and permissive hypercapnea. There is proven mortality benefit of low Vt but permissive hypercapnea may precipitate intracranial hypertension[21].

Mechanical ventilation predisposes to potentially significant hemodynamic fluctuations. These may be harmful in TBI due to impaired auto regulation rendering the brain extremely vulnerable to CPP fluctuations.

Positive end expiratory pressure (PEEP) improves oxygenation by recruitment of atalectatic alveolar units, improving functional residual capacity (FRC) and preventing atlectrauma. However it may have detrimental neurologic effects like alteration of ICP and CPP in severe TBI [22]. Because PEEP is applied in non-compliant lungs (as in ARDS) as opposed to compliant lung (in healthy subject), effect on CBF and CPP is usually modest. As such PEEP is therefore safe to apply as part of ventilator strategy to improve oxygenation.

The plan to liberate the patient from mechanical ventilation should be made at initiation of mechanical ventilation. Patients with neurological injury associated with TBI are often difficult to assess, leading to frequent extubation delays. Assessment for extubation readiness can be simplified into three criteria namely respiratory, hemodynamic and neurologic. Neurologic criteria requires that ICP and CPP should be at least ≤ 20 mm of Hg and ≤ 60 mm of Hg respectively.

Association of brain injury with pulmonary dysfunction makes management of such patient a greater therapeutic challenge.

TBI PATIENTS REQUIRING HEMODIALYSIS

Dialysis in patients with TBI is made more complicated by their propensity to develop pulmonary edema and by their sensitivity to fluctuations of blood pressure, all of which can exacerbate secondary brain injury[23]. The normal pre-packaged dialysate fluids have the wrong electrolyte for TBI patients and the normal dialysis prescription designed to maximize the efficiency of the circuit can lead to unacceptable episode of

ICP elevation. Both chronic renal failure and acute renal failure are associated with worse outcome in TBI. Davenport wrote two articles[24,25] and Yeh et al[26] published one article focusing on prevention of ICP fluctuation during renal replacement therapy (RRT) by modifying intermittent hemodialysis (IHD) protocol. They are summarized at Table 1

Table 1

Domain	Recommendations	Rationale
Access	Avoid internal jugular lines	Promote venous drainage from the brain
Modality	Prefer CRRT Low efficiency IHD/SLED	Produces a more gradual solute clearance; less likely to produce cerebral oedema
Frequency	Daily, if not continuous	Daily treatments decrease the fluctuations of urea
Blood flow	Start low, increase slowly	Minimize haemodynamic effects
Dialysate flow	Start low, increase slowly	Minimize solute clearance
Dose	Under-dialyse (by half)	Minimize solute clearance per unit time
Solute clearance	Pre-dilution haemofiltration	Minimize urea clearance: decrease the resulting urea gradient between brain parenchyma and blood, minimizing cerebral oedema
Filtration	Low volume fluid removal	Minimize dialysis-associated hypotension to prevent cerebral hypoperfusion
Anticoagulation	Regional, or none	Prevent cerebral haemorrhage extension due to anticoagulation. Minimal anticoagulation is recommended for 2 weeks following TBI.
Dialysate	Add sodium	Minimize the hyponatremia which develops due to exposure to hyponatremic dialysate (to keep sodium around 145-150 mmol/L)
	Add urea	Minimize urea clearance
	Minimize bicarbonate	Prevent intracellular acidosis (may be hypothetical)
Fluid warmer	Temperature matching	Maintain therapeutic hypothermia if this is being used for ICP control

TBI AND HEART FROM ICU PERSPCTIVE

Cardiovascular complications are common after brain injury and are associated with increased mortality and morbidity[27]. Neurogenic cardiac injury is related to brain injury-induced catecholamine and inflammatory responses. The spectrum of abnormalities includes hypertension, hypotension, ECG changes, cardiac arrhythmias, release of biomarkers of cardiac injury, and left ventricular (LV) dysfunction. The abnormalities are usually reversible and management should therefore focus on general supportive care and on treatment of the underlying brain injury.

High sympathetic tone persists for some time after brain injury, with circulating catecholamine levels remaining high for up to 10 days. The intense systemic vasoconstriction associated with the catecholamine 'storm' increases cardiac afterload, myocardial workload, and oxygen demand. Because of simultaneous coronary vasoconstriction, the increase in myocardial oxygen demand is not associated with an increase in oxygen delivery and sub-endocardial ischaemia and impaired ventricular function may follow. This can lead to cardiogenic pulmonary oedema and systemic hypotension[28].

Hypotension may also occur because of head injury-related disruption of brain stem centers for hemodynamic control, commonly related to diffuse axonal injury. Although neurogenic hypotension is uncommon after isolated head injury in adults, it is associated with higher mortality than hemorrhagic hypotension.

The neurogenic stunned myocardium (NSM) syndrome is a reversible neurologically mediated cardiac injury characterized by ECG changes, arrhythmias, LV dysfunction, and release of biomarkers of cardiac injury. The severity of NSM and hence the degree of myocardial damage is related to the severity of the underlying brain injury[29].Although most commonly associated with subarachnoid hemorrhage (SAH), NSM is seen after other types of brain injury and also in non-neurological conditions such as phaeochromocytoma.

Neurogenic cardiovascular dysfunction may cause minimal clinical effects but, in severe cases, can lead to cardiogenic shock and pulmonary oedema.

The initial catecholamine surge results in hypertension and tachycardia, and early studies demonstrated that β-adrenergic blockade reduces myocardial injury and improves neurological outcome after SAH[27].

As the catecholamine surge subsides, the initial hyperdynamic response is often followed by significant hypotension because of unopposed peripheral vasodilatation and ventricular dysfunction. Arterial pressure usually responds to fluid resuscitation and standard vasopressor/inotropic support. Norepinephrine is widely used and provides predictable

control of arterial pressure and CPP after TBI. Vasopressin may be effective in refractory hypotension but is associated with cerebral vasoconstriction and a risk of brain ischaemia so should be used with caution. Dobutamine is effective in normalizing cardiac index in NSM-related low cardiac output states after SAH.

Brain injury-related ECG abnormalities have been recognized for more than five decades and are particularly common after SAH where they are reported in 49–100% of cases. The most common findings are ST segment changes, flat or inverted T waves, prominent U waves, and prolongation of the QTc interval (QTc is the QT interval corrected for heart rate)[30].

ECG changes occur most commonly in the first few days after injury and are often transient because repolarization normalizes as the neurological insult resolves.

Neurogenic ECG changes are generally asymptomatic, but abnormalities such as ST segment depression and abnormal T waves can be associated with the development of a delayed ischaemic neurological deficit, poor outcome, and death after SAH. Excessive prolongation of the QTc interval may be a cause of sudden cardiac death after brain injury. Drugs that prolong the QTc interval should therefore be avoided after brain injury, even into the rehabilitation phase.

Cardiac rhythm disturbances, including sinus tachycardia, atrial fibrillation, premature atrial and ventricular contractions, and AV dissociation, are also common after brain injury and usually occur within the first 7 days[30].

A 12-lead ECG should be recorded on admission and repeated at 24 h intervals until any abnormalities have resolved. There is no specific treatment for brain injury-induced cardiac arrhythmias, and although standard therapies such as correction of electrolyte disturbances should be provided, management of the underlying intracranial pathology is the most effective way to prevent and treat the arrhythmia. It is wise to call for a cardiology consultation in this situation.

The propofol infusion syndrome (PRIS) is characterized by unexplained metabolic acidosis, elevated creatinine kinase, rhabdomyolysis, and widespread ECG changes and can lead to cardiac myocytolysis, rhabdomyolysis, and acute renal failure[31].Propofol is widely used to sedate brain-injured patients and to control ICP, and the ECG consequences of PRIS can be difficult to differentiate from those associated with the NSM syndrome. Propofol infusion should be discontinued immediately if PRIS is suspected and attempts made to exclude NSM as a differential or coincidental diagnosis in brain-injured patients.

Elevation of cardiac troponin I (cTnI) has been reported in 20–68% of patients after SAH (mean incidence 36%) and usually peaks within 24–36 h[32].The peak concentration is usually below the threshold for the diagnosis of myocardial infarction but may be associated with a mild, transient impairment of ventricular function in 50% of patients. The degree of cTnI increase is related to the severity of the initial brain injury.

Elevated serum B-type natriuretic peptide (BNP) is also independently associated with LV dysfunction, pulmonary oedema, and adverse neurological outcomes after SAH[33].

Impaired LV contractility, hypokinesia, and low ejection fractions are associated with the NSM syndrome. There is a characteristic pattern of regional wall motion abnormalities (RWMAs) involving the basal and middle portions of the antero septal and anterior ventricular walls, with relative apical sparing[34].

Coronary angiography is the definitive diagnostic test to exclude coronary artery disease but is seldom indicated in this high-risk group of patients[28].In any case, the presence of significant coronary artery disease does not exclude co-incidental NSM. Brain injury-related cardiovascular dysfunction is essentially a diagnosis of exclusion. Following features strongly suggest a neurogenic cause.

- No history of cardiac problems,
- Temporal relationship between brain injury and cardiovascular abnormalities,
- ECG changes in isolation,
- Modest elevations in cTnI,
- New onset LV dysfunction,
- Cardiac wall motion abnormalities that do not correspond with coronary vascular territories,
- Inconsistency between echocardiographic and ECG findings,
- Inconsistency between cTnI and LV ejection fraction (cTI<2.8 μg litre^{-1} in association with LV ejection fraction <40%),
- Spontaneous, early resolution.

MONITORING TBI PATIENTS IN ICU

Regular monitoring of patients with severe TBI in the ICU is critical for early detection and diagnosis and management of secondary injuries. They are [35] as follows.

a) Clinical assessment : GCS , Pupillary size and reaction and motor responses

b) ICP monitoring: Measurement of ICP with intra ventricular catheter. Advantages include zero calibration, CSF drainage for raised ICP.

c) Cerebral blood flow with jugular bulb oxymetry (SjO2): Involves fiber optic placement of a retrograde catheter in the jugular valve. SjO2 < 55% is suggestive of cerebral hypo perfusion and SjO2 > 60% is suggestive of hyper perfusion.

d) Cerebral lactate fluxes: From blood samples of jugular bulb catheter. Measures arterio jugular lactate differences to measure cerebral perfusion.

e) Near Infrared Spectroscopy: A noninvasive method of measuring regional cerebral oxygen saturation and cerebral blood volume.

f) Trans cranial Doppler: A non-invasive method of measuring CBF velocity and useful in diagnosing critical elevations of ICP and decreases in CPP in TBI patients.

CONCLUSION

While managing patients in ICU, clinicians need to keep in mind the factors that determine outcome of TBI e.g. a) Severity of primary and secondary injuries of brain, b) Low GCS on presentation, c) Advanced age (>65yrs), d) Comorbidities and e) Severity of associated injuries.

Critical care management of TBI patients ideally includes round the clock follow-up of vital signs, intake and output, pulmonary and cardio-vascular parameters, follow-up of pupillary status and GCS documented by bed side critical care nurses in a 24 hour flow chart. Neurosurgical team and critical care team work hand in hand to monitor and manage such patient whether the patient receives operative care or not.

REFERENCES

1) Timofeev I, Santarius T, Kolias AG, Hutchinson PJA. Decompressive craniectomy:operative technique and perioperative care. Advances and technical standards in neurosurgery (2012) Springer, New York, pp 115–136.

2) ATLS student course manual 8th Edition. American college of surgeons committee on Trauma. Page 132.

3) Wright DW, Merck LH. Head Trauma. In Tintinalli JE. Editor in Chief. Tintinalli's Emergency Medicine, A comprehensive study Guide. 8Th edition. New York. McGraw Hill education. P 1696.

4) Dixon J, Comestock G, Whitfield J, Richards D, Burkholder TW, Leifer N et al. Emergency department management of traumatic brain injuries: A resource tiered review. African journal of emergency medicine. 10(2020); 159-166.

5) Mittal R, Vermani E, Tweedie I, Nee PA. Critical care in emergency department traumatic brain injury .Emerg Med J 2009;26(7):513-7.

6) Upchurch CP, Grisalva CG,Russ S,Collins SP, Semler MW, Rice TW et al. Comparison etomidate and ketamine for induction during raptd sequence intubation of adult trauma patients. Ann Emerg Med 2017 ; 69: 24-33.

7) Holly LT, Kelly DF, Counelis GJ, BlinmanT, McArthur DL, Cryer HG. Cervical spine trauma associated with moderate and severe head injury: incidence, risk factors, and injury characteristics. J Neurosurger 2002;96(3 suppl): 285-91.

8) Rincon F, Kang J, Vibbert M, Urtecho J, Athar MK, Jallo J. Significance of arterial hyperoxia and relationship with case fatality in traumatic brain injury: a multicenter cohort study. J Neurol Neurosurg Psychiatry2014; 85(7):799-805.

9) Oertel M, Kelly DF, Lee JH, McArthur DL, Glenn TC, Vespa P et al. Efficacy of hyperventilation, blood pressure elevation and metabolic suppression therapy in controlling intra cranial pressure after head injury. J Neurosurg 2002;97(5):1045-53.

10) Carney N, Totten AM, O'Reilly C, Ullman JS, Hawryluk GW, Bell MJ et al. Guidelines for management of severe traumatic brain injury. Fourth edition. Neurosurgery2017; 80(i):6-15.

11) Chesnut RM, Marshall LF, Klauber MR, Blunt BA, Baldwin N, Eisenberg HM et al. The role of secondary brain injury in determining outcome from severe head injury. J Trauma 1993; 34(2):216-22.

12) Alvis-Miranda HR, Castellar-Leones SM, Moscote-Salazar LR. Intravenous fluid therapy in traumatic brain injury and decompressive craniectomy. Bull Emerg Trauma 2014;2(1):3-14.

13) Wafaisade A, Lefering R, Tjardes T, Wutzler S, Simanski C, Paffrath T et al. Acute coagulopathy in isolated blunt traumatic brain injury. Neurocrit care 2010; 12(2):211-9.

14) Wyatt JP, Illingworth RN. Graham CA, Clansy MJ, Robertson CE. Chapter 8. Head Injuries. Oxford Handbook of Emergency Medicine.3rd ed. Oxford (UK): Oxford University Press; 2006. P 362-67.

15) Narayan RK, Kishore PR, Becker DP, Ward JD, Enas GG, Greenberg RP et al. Intracranial pressure to monitor or not to monitor ? A review of our experience with severe head injury. J Neurosurg. 1982; 56(5):650-9.

16) Bourdeaux CP, Brown JM. Randomized controlled trial comparing the effect of 8.4% sodium bicarbonate and 5% sodium chloride on raised intracranial pressure after traumatic brain injury. Neurocrit care 2011; 15(1): 42-5.

17) Ngubane T. Mechanical ventilation and the injured brain. South African J Anesthesia and Analgesia 2011. 17:1,76-80. DOI:10.1080/22201173.2011.10872737

18) Pelosi P, Severgnini P, Chiaranda M. An integrated approach to prevent and treat respiratory failure in brain injured patients. Current opinion in Crtical care, 2005; 11:37-42.

19) Stevens RD, lazaridis C, Chalea JA. The role of mechanical ventilation in in acute brain injury. Neurologic Clinics. 2008; 26:543-563.

20) Ventilation with low tidal volumes as compared with traditional tidal volumes for acute lung injury and acute respiratory distress syndrome. The Acute Respiratory Syndrome Network. NEJM 2000; 342(18):1301-8.

21) Bratton SL. Chapter 13: Hyperventilation. Pediatric Crit Care Med 2012. Vol. 13 No 1 (suppl. 1).

22) Huynh T, Messer M, Sing RF, Miles W, Jacobs DG, Thomson MH. Positive end expiratory pressure alters intracranial and cerebral perfusion pressure in severe traumatic brain injury.. Journal of Trauma. 2002; 53(3): 488-93.

23) Moore EM, Bellomo R, Nichol A, Harley N, Macisaac C, Cooper DJ. The incidence of acute kidney injury in patients with traumatic brain injury. Renal failure. 2010; 32(9):1060-5.

24) Davenport A. Renal replacement therapy for the patient with acute traumatic brain injury and severe kidney injury. Acute kidney injury. Krager publishers.2007; 156: 333-339.

25) Davenport A. Practical guidance for dialyzing a hemodialysis patient following acute brain injury. Hemodialysis International 2008;12(3):307-12

26) Yeh S-H, Wang C-Y, Lin C-M. Preventing intracranial pressure fluctuation in severe traumatic brain injury during hemodialysis. J Med Sciences 2016; 36(4):152.

27) VanderBilt IAC, Hasan D, Vandertop WP,Wilde AM, Algra FC, Visser G et al .Impact of cardiac complications on outcome after aneurysmal subarachnoid hemorrhage: a meta-analysis. Neurology. 2009; 72: 635-42.

28) Nguyen H, Zaroff JG. Neurogenic stunned myocardium. Curr Neurol Neurosci Rep. 2009; 9:486-91.

29) Tung P, Kopelnik A ,Banki N, Ong K, Ko N, Lawton MT et al .Predictors of neuro cardiac injury after subarachnoid hemorrhage. Stroke.2004; 35: 548-51.

30) Grunsfeld A, Fletcher JJ, Nathan BR. Cardiopulmonary complications of brain injury. Curr Neurol Neurosci.Rep. 2005; 5: 488-93.

31) Ottersproor LC, Kalkman CJ, Cremer OL. Update on propophol infusion syndrome in ICU. Curr Opin Anesthesiol. 2008; 21:544-51.

32) Bruder N, Robinson A. Cardiovascular and pulmonary complication of aneurysmal sub arachnoid hemorrhage. Neurocrit care. 2011; 15:257-69.

33) Tung PP, Olmsted E, Kopelnik A, Banki NM, Drew BJ, Ko N et al. Plasma B-type natriuretic peptide levels are associated with early cardiac dysfunction after subarachnoid hemorrhage. Stroke. 2005;36: 1567-9

34) Banki N, Kopelnik A, Tung P, Lawton MT, Gress D, Drew B et al. Prospective analysis of prevalence, distribution, and rate of recovery of left ventricular systolic dysfunction in patients with subarachnoid hemorrhage. J Neuro surg. 2006; 105:15-20.

35) Tobi KU. Monitoring traumatic brain injury patients in ICU: A useful pneumonic and review of literature. Anesthesiol Clin Sci Res. 2019; 3(1):8-11.