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General anesthesia in patient with Brain Injury

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Abstracts

The basic concept of Neuroanesthesia & Neuro Critical Care is referred to as the ABCDE of neuroanesthesia. Early Brain Injury (EBI) was formerly known as primary brain injury. In EBI there is loss of autoregulation, loss of blood-brain barrier integrity. The presence of Cushing's triad indicates the presence of intracranial hypertension. The target of blood pressure in traumatic brain injury (TBI) is to avoid blood pressure systolic <110 mmHg, maintain cerebral perfusion pressure (CPP) 60-70 mmHg, target PaCO2 regulation is normocarbia, PaCO2 35–40 mmHg, prophylactic use of phenytoin or valproate is not recommended to prevent late post-traumatic seizures (late PTS).

There is still a need to analyze decompressive craniectomy (DECRA) therapy compared with continuous medical therapy for refractory intracranial pressure (ICP) elevations after TBI. General anesthesia for patients with severe TBI is preferable to total intravenous anesthesia (TIVA), the administration of fluids should consider the osmolarity of these fluids. In the new concept, in patients with elevated ICP, the volatile anesthetic concentration should be limited to 0.5 MAC. The target blood sugar is normoglycemia. Prophylactic or therapeutic hypothermia appears to have no place in the management of severe brain injury.

Keywords: : Blood-Brain Barrier, Anesthesia, Intravenous Post-Traumatic, Brain Injuries, Cerebrovascular Circulation, Intracranial Hypertension

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Injuries, Cerebrovascular Circulation, Intracranial Hypertension

1. Introduction

Management of patients with severe traumatic brain injury based on the guidelines of the 4th edition of the Brain Trauma Foundation Guidelines 2016. However, the 2016 guideline does not discuss how to use neuroanesthesia techniques and drugs if the patient requires surgery. This paper examines the thoughts of what happened or what articles existed from the period after the publication of the 2016 BTF Guideline to the present (period 2016 – 2021), whether anything has changed after 5 years, especially in relation to neuroanesthesia[1,2].

The basic concepts of Neuroanesthesia & Neuro Critical Care are referred to as the ABCDE of Neuroanesthesia, namely: A is Airway, clear airway means the airway must be free at all times, B is breathing by controlling ventilation to achieve normocapnia targets in traumatic brain injury (TBI). C is circulation to avoid increasing or decreasing blood pressure, avoiding increased cerebral venous pressure, normovolemia, iso osmolar. D stands for drugs, meaning give drugs that have a brain-protective effect and avoid drugs that can increase intracranial pressure (ICP). is the environment, controlling body temperature with a target of 350C in the operating room and 36O c in the intensive care unit (ICU). The content of this topic is to examine whether anything has changed in the management of severe TBI patients from start to finish the issuance of the 2016 BTF guidelines until now (in 2021). Airway, Breathing talk about PaCO2 regulation, Circulation about cerebral autoregulation/pressure blood and ICP, D-drug on anesthetic drugs and Environment on regulating the patient's body temperature discusses therapeutic and prophylactic hypothermia[1,2].

II. Early Brain Injury (EBI) Pathophysiology Early brain injury was formerly known as primary brain injury. In EBI there is a loss of autoregulation, loss of integrity of the blood-brain barrier. The historic concept of EBI is the presence of systemic hypertension secondary to the presence of the Cushing reflex. The presence of Cushing's triad indicates the presence of intracranial hypertension. In EBI there is a massive release of catecholamines which can be seen in ECG changes and abnormalities of heart wall movement[3,4].

There is a release of cytokines which causes capillary leakage and pulmonary oedema. The new concept of hypertension after EBI occurs is the occurrence of massive sympathetic release that causes hypertension and tachycardia. The release of cytokines can cause capillary leakage resulting in pulmonary edema[6,7].

When blood pressure decreases, ischemia occurs and when blood pressure increases, edema occurs. The question is what is the target blood pressure for TBI, what is the upper limit and what is the lower limit for safe blood pressure? So, what's new when compared to the 2016 BTF Guidelines? What is different is in terms of the definition of hypotension. The definition of hypotension for TBI in the 2007 BTF guidelines is when the systolic pressure is less than 90 mmHg, while in the 2016 BTF guidelines the definition of hypotension is if the systolic pressure is less than 110 mmHg[2,7-9].

What is the target mean arterial pressure (MAP)?

III. Blood Pressure Regulation: Systolic blood pressure and cerebral perfusion pressure, Target blood pressure in neurosurgery patients, their upper and lower limits[10,11]. A new concept of dynamics of the cerebral autoregulatory curve In the past, autoregulation was the MAP 50-150 mmHg, whereas in the MAP range of 50-150 mmHg, cerebral blood flow (CBF) was maintained between 50-54 mL/100 g tissue/ minute. At a MAP below 50 mm Hg even with maximum dilatation, CBF will decrease and cerebral ischemia, cerebral infarction and neuronal cell death may occur. At MAP above 150 mmHg even with maximum constriction, CBF cannot be maintained at 50–54 mL/100 g tissue/min but increases and the blood-brain barrier is broken and brain edema develops[12,13].

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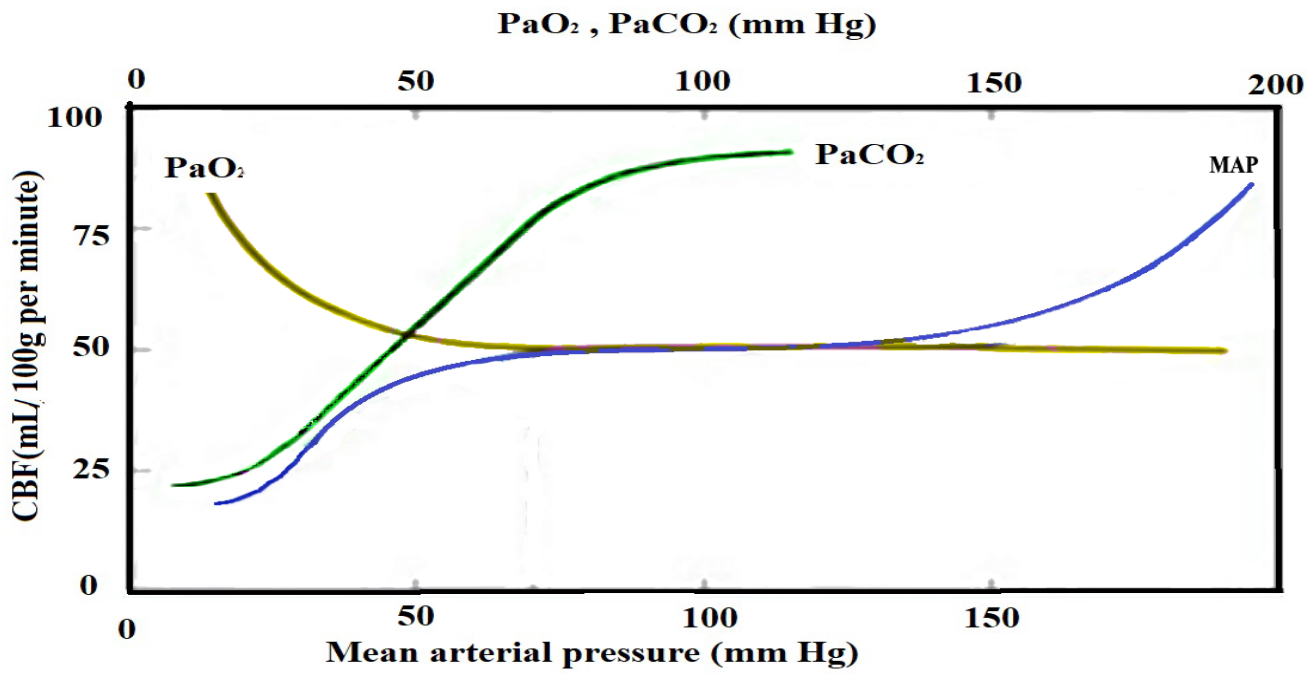

Figure 1. cerebral autoregulation carves with Partial vapor pressure for co2 and O2

The lower limit of autoregulation according to Miller Anesthesia 2020. 9-Edition, To answer this question, the formula below can be used as a guide To answer this, the formula below

This can be used as a guide:

CPP = MAP – ICP

MAP = CPP + ICP

MAP = 60 + (20 or 25) = 80-85

MAP = 70 + (20 or 25) = 90-95

CPP = Cerebral Perfusion Pressure

MAP = Mean Arterial Pressure

ICP = Intracranial Pressure

Target Brain Perfusion Pressure (Cerebral Perfusion

Pressure/CPP)

According to BTF 2007: target CPP 50-60 mmHg.

4 According to BTF 2016: target CPP 60-70 mmHg.

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In Miller Anesthesia 2020, the lower limit of autoregulation is a MAP of 70 mmHg[14].Monitoring of CPP is necessary because management of severe head injury using guidelines based on recommendations for monitoring CPP can reduce 2-week mortality. The recommended target CPP value for a good outcome is between 60–70 mmHg. Whether 60 or 70 mmHg is the minimum optimal CPP threshold is unclear and depends on the autoregulatory status of the patient avoid aggressive measures to maintain CPP >70 mmHg with fluids and pressors due to the risk of adult respiratory failure, avoid CPP <50 mmHg because of the risk of cerebral ischemia[10,11].

However, the MAP range so narrow in Traumatic brain injury (TBI) this is due to the loss of cerebral autoregulation, loss of the blood-brain barrier so that cerebral blood flow depends on MAP and hypotension will cause hypoperfusion, whereas hypertension causes hyper perfusion and brain edema[10,15].

Based on the 2016 BTF Guideline, the recommended blood pressure threshold is to maintain a systolic blood pressure of 100 mmHg for patients aged 50 to 69 years or 110 mmHg for patients 15 to 49 years or >70 years, is considered to reduce mortality and improve outcome. So what's new? What's new is that CPP is maintained at 60-70 mmHg, and avoid systolic blood pressure <110 mmHg[13-15].

IV. PaCO2 regulation in TBI: Why PaCO2 . Assessment needs as often as possible Cerebral blood flow (CBF) regulated mainly by autoregulation, PaCO2 and PaO2.

Normal CBF is 50-54 mL/100 g tissue/minute and every 1 mmHg change in PaCO2 CBF changes 4%. The 2016 BTF Guidelines stated that up to 4 hours after severe traumatic brain injury, CBF decrease by 50% of normal values and reaches 80% of normal values at the end of 24–72 hours, therefore, hyperventilation is contraindicated for the first 24 hours after TBI. In Miller Anesthesia 2020 it is stated that the target is normocarbia, PaCO2 35-40 mmHg, hypercarbia will increase CBF and eliminate autoregulatory curves while hypocarbia maintains autoregulatory curve by lowering CBF[16].

V. Pressure Monitoring Recommendations

Intracranial Pressure (ICP) Management of severe TBI using information from ICP monitoring is recommended to reduce length of hospital stay and reduce mortality 2 weeks after injury. ICP should be monitored in all salvageable patients with TBI (Glasgow Coma Scale/GCS 3-8 after resuscitation) and abnormal computed tomography (CT)-scans. An abnormal head CT scan is the presence of a hematoma, contusion, swelling, herniation, or compression of the basal cisternae. Indications for ICP monitoring are patients with severe TBI with a normal CT scan if 2 of the following features are present at the time of admission[17].

Admission to hospital: age >40 years, unilateral or bilateral motor posturing, or systolic blood pressure <90 mmHg.4,5 In the BTF 2007 guideline, recommended ICP thresholds to be initiated therapy increases ICP if ICP > 20 mmHg, however, according to the 2016 BTF guidelines, therapy if ICP > 22 mmHg because above this level is associated with increased mortality. Combination of values ICP, clinical findings and brain CT scan are used for patient management decisions[10-12].

VI. Anesthesia Management: Total Intravenous Anesthesia (TIVA) vs volatile anesthetics : Induction of anesthesia Perform preoxygenation, monitor blood pressure every 1-2 minutes, for induction give intravenous anesthetics propofol or pentothal or etomidate, analgesics fentanyl or remifentanil, muscle relaxant rocuronium and perform gentle hyperventilation, quick gentle intubation. Target during induction, do not let blood pressure drop below 110 mmHg, target CPP 60-70 mmHg, MAP 85-85 mmHg[18].

3.7 Maintenance of anesthesia

Use total intravenous anesthesia (TIVA) In Miller Anesthesia 2020, the lower limit of autoregulation is a MAP of 70 mmHg1.Monitoring of CPP is necessary because management of severe head injury

using guidelines based on recommendations for monitoring CPP can reduce 2-week mortality. The recommended target CPP value for a good outcome is between 60–70 mmHg. Whether 60 or 70 mmHg is the minimum optimal CPP threshold is unclear and depends on the autoregulatory status of the patient. Avoid aggressive measures to maintain CPP >70 mmHg with fluids and pressors due to the risk of adult respiratory failure, avoid CPP <50 mmHg because of the risk of cerebral ischemia[5,18-20].

Why is the MAP range so narrow in TBI? This is due to the loss of cerebral autoregulation, loss of the blood-brain barrier so that cerebral blood flow depends on MAP and hypotension will cause hypoperfusion, whereas hypertension causes hyper perfusion and brain edema. Based on the 2016 BTF Guideline, the recommended blood pressure threshold is to maintain a systolic blood pressure of 100 mmHg for patients aged 50 to 69 years or 110 mmHg for patients 15 to 49 years or >70 years, is considered to reduce mortality and improve outcome. So, what's new? What's new is that CPP is maintained at 60-70 mmHg, and avoid systolic blood pressure <110 mmHg[5].

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3.7 Maintenance of anesthesia Use total intravenous anesthesia (TIVA) with propofol/remifentanil/fentanyl infusion. Pure TIVA or in combination with low concentrations of volatile anesthetics are popular anesthetic techniques in neuroanaesthesia, avoid volatile anesthetics until the dura is opened.

Maintain CPP 60–70 mmHg, administer ventilation based on end tidal CO2 Give mannitol 1 gm/kg over 15–20 minutes.

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Why is total intravenous anesthesia preferred in neuroanaesthesia? Propofol maintains the cerebral autoregulatory curve and lowers ICP, volatile anesthetics suppress cerebral autoregulation, volatile anesthetic concentrations greater than 1 MAC should be avoided.

Which volatile anesthetic should be used? Must understand about the cerebral vasodilating effects which is desflurane > isoflurane > sevoflurane[25].

How to lower ICP quickly if after opening the dura, the brain is swollen? Stop administering volatile anesthetics, administer bolus of propofol with phenyl epinephrine, start TIVA as primary anaesthetic, hyperventilate until problem resolves and then maintain How to lower ICP quickly if after opening the dura, the brain is swollen? Discontinue volatile anesthetics, give bolus of propofol with phenyl epinephrine, start TIVA as primary anaesthetic, hyperventilate until problem resolves and then maintain normocarbia[24].

3,7 In the new concept, in patients with elevated ICP, volatile anesthetic concentrations should be limited to 0.5 MAC. In increasing intracranial volume as in TBI, volatile anesthetics should be avoided at least until the dura is opened[24].

VII. Antiseizure Prophylaxis Anti-seizure prophylaxis Complaints of acute seizures may occur as a result of severe traumatic brain injury (TBI). Post-traumatic seizures (PTS) are classified as early if they occur within 7 days of the injury or late if they occur 7 days after the injury. Post-traumatic epilepsy (PTE) was defined as a recurrent seizure more than 7 days after the injury.

In patients with severe TBI, the clinical PTS rate is approximately 12%, whereas electroencephalography detectable subclinical seizures is about 20-25%. Risk factors for early PTS Glasgow Coma Scale (GCS) score 10, immediate seizures, post-traumatic amnesia lasting more than 30 minutes, linear or depressed skull fracture, penetrating head injury, subdural, epidural, or intracerebral hematoma, cortical contusion, age ≤65 years , chronic alcoholism[23].

One study in 2010 showed that the rate of PTE was higher than the general risk of developing epilepsy. Risk for PTE are individuals with severe TBI, early PTS, acute intracerebral hematoma or cortical contusion, posttraumatic amnesia lasting more than 24 hours, age >65 years, or a history of premorbid depression[26].

Seizure prophylaxis for PTS refers to the practice of administering anticonvulsants in TBI patients are on target to prevent seizures.

The rationale for routine administration of seizure prophylaxis is because the incidence of seizures in severe TBI patients is relatively high and there may be advantages of prevention after TBI (eg limiting acute physiologic derangement, preventing epilepsy)

chronic condition, preventing herniation and death). However, it is also desirable to prevent neurobehavioral and other side effects of these drugs, especially if they are not effective in preventing seizures. Therefore, it is important to thoroughly evaluate the benefits, as well as the harm, of anticonvulsant drugs used to prevent PTS[26].

The use of levetiracetam is increasing for seizure prophylaxis from a variety of pathologies, including TBI. Availability of comparative studies, insufficient to support recommendations or prohibit the use of levetiracetam, further research is needed to understand the possible benefits or harms of levetiracetam in the treatment of TBI patients[29].

The prophylactic use of phenytoin or valproate is not recommended to prevent late PTS. Phenytoin has recommended to reduce the incidence of early PTS (within 7 days of injury), where the overall benefit is felt to exceed complications associated with this therapy. However, early PTS was not associated with worse outcome. At this time there is insufficient evidence to recommend levetiracetam over phenytoin in terms of the benefits of preventing early post-traumatic seizures and toxicity. Dosage of phenytoin in adults; 10-15 mg/kg body weight, given by slow injection or infusion at a rate of not more than 50 mg/minute. The maintenance dose is

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100 mg given every 6-8 hours (or orally). The lethal dose in adults is 2–5 g/day. Dosage of phenytoin for children; 15-20 mg/kg BW at a rate not exceeding 1-3 mg/kg per minute[30]. A meta-analysis and systematic review published in 2020 on anti-seizure prophylaxis in TBI consisted of 3 RCTs (n = 750) and 6 an observational study (n=3362) showed that the benefit was limited to the prevention of early seizures[28-30].

VIII. Decompressive Craniectomy (DC) Bifrontal decompressive craniectomy is not recommended to improve outcome as assessed by the Extended Glasgow Outcome Scale (GOSE) score at 6 months after injury in severe traumatic brain injury patients.

with diffuse injury (without mass lesion), and with an increase in ICP >20 mmHg over 15 minutes in a refractory 1-hour period to first-tier therapy. However, this DC procedure has been shown to reduce ICP and reduce ICU stay. A large front temporoparietal DC (not less than 12 x 15 cm or 15 cm in diameter) is recommended rather than Small front temporoparietal DCs to reduce mortality and improve neurologic outcome in patients with severe TBI.

The Randomized Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intracranial Pressure (RESCUEicp) study, was conducted in 408 patients with refractory ICP elevation (>25 mmHg). The study compared DECRA therapy with continuous medical therapy for refractory increase in ICP after TBI. Assessment at 6 months DC showed lower mortality but higher vegetative state, lower severe disability and upper severe disability than medical therapy. Meanwhile, moderate disability and good recovery were the same between the two groups[31].

IX. Fluid Management in TBI Normal saline osmolality is close to plasma whereas Ringer's lactate is slightly hypotonic. Chloride-rich fluids can contribute to hyperchloremic acidosis and impair renal blood flow, so Ringer's lactate is commonly used in neurosurgical operations. However, the osmolarity of RL is 273 mOsm/L and NaCl is 304 mOsm/L while plasma is 290 mOsm/L, Ringer lactate balanced solution has an osmolarity of 309 mOsm/L. Thus, it is necessary to use fluids that exceed plasma osmolarity Fluid replacement during neurosurgery surgery is 50% of the total urine when given mannitol, the goal is euvolemia. If large amounts of fluid (more than 2 liters) are needed, NaCl or RL can be given. No need to do fluid rewarming, adequate to operating room temperature.

The Saline versus Albumin Fluid Evaluation (SAFE) trial in patients with TBI severe (GCS3-4), indicating that administration of 4% albumin showed a higher 30-day mortality than those given NaCl, however, there are limitations to this SAFE trial, namely that TBI patients were not randomized, 4% albumin was hypo osmolar (osmolarity was 274 mOsm/L). On the other hand, research in Sweden, which was a retrospective study (n=93), showed lower 28-day and 18-month mortality in severe TBI when zero or negative fluid balance albumin was used. What about hyperosmolar therapy? Which mannitol with hypertonic saline is better? Hypertonic saline (HTS) reduced ICP better than mannitol with no difference in short-term outcome. In the 2007 BTF guidelines it is stated that HTS preferred over mannitol in TBI patients. In the 2016 BTF guidelines, either mannitol or bycatch are acceptable. A 2019 meta-analysis (12 RCTs, n=464) showed a better reduction in ICP with HTS at 30–120 min intervals, but no difference in outcome at discharge from the hospital. Unfortunately, in this meta-analysis there were no long-term outcome studies[5,12,31].

The Cochrane review in 2021 (n=287, 6 RCTs) comparing mannitol vs HTS, 91% of patients had a GCS of less than 8 (TBI weight), and the conclusion was that there was no difference in outcome[32].

X. Glucose Management Hyperglycemia often occurs in the early phase after TBI and is associated with poor outcome. While acute hyperglycemia is toxic, the magnitude of the increase in blood glucose required to cause damage remains uncertain. Pathogenesis of hyperglycemic stress It is widely thought to represent a complex interaction between

endogenous catecholamines, cytokines and activation of the hypothalamic-pituitary-adrenal axis resulting in excessive cortisol secretion and triggering gluconeogenesis. It is getting exacerbated by therapeutic interventions such as administration of catecholamines and exogenous steroids, and underlying insulin resistance or impaired insulin secretion. The pathophysiological mechanism by which Hyperglycemia may worsen TBI outcomes, including promotion of oxidative stress pathways and induction of neuroinflammation The current management of hyperglycemia in TBI involves the use of short-acting insulin given as a continuous intravenous infusion, titrated to maintain blood glucose systemically within the target ranges obtained from randomized controlled trials in medical or surgical intensive care units general (ICU) [33]. The study of *Cefalu WT.,* resulted in a paradigm shift in the approach to blood glucose management incritically ill patients, based on their finding that targeting intensive glycemic control (4.4–6.1 mmol/L) reduced mortality in the surgical ICU population, despite an increased incidence of hypoglycemia. While there is considerable use of this method, subsequent multi-center RCT studies confirmed a higher incidence of severe hypoglycemia with intensive glycemic control and refutes early observations by identifying increased mortality with intensive blood glucose control therapy[34].

Intensive glycemic control does not reduce mortality in patients with TBI but greatly increases the risk of hypoglycemia. Signal to increase yield Neurological studies with intensive glycemic control in post-TBI patients require investigation. This is probably best done with use a safer approach to glycemic control that reduces the risk of hypoglycemia, using blood sugar targets stratified that takes into account the physiological heterogeneity in patients with TBI[33,34].

XI. Hypothermia

Deliberate hypothermia (prophylactic hypothermia) in the initial management of TBI did not improve neurologic outcome at 6 months. Induced hypothermia may also increase the risk of pneumonia. Hypothermia (33–35o C) is sometimes induced to try and limit brain damage in people with severe head injuries. However, the evidence for its safety and effectiveness has been mixed. A 2015 study (Eurotherm 3235) found that therapeutic hypothermia, for adults with elevated intracranial pressure, did not produce a better outcome than standard therapy that did not involve therapeutic hypothermia[35]. The Prophylactic Hypothermia Trial to Lessen Traumatic Brain Injury- Randomized Clinical Trial (POLAR-RCT) these are larger, and hypothermia is induced as soon as possible after brain injury, regardless of signs of brain swelling. Overall, this trial does not support the practice of prophylactic or therapeutic hypothermia after head injury[36].

Why is this study necessary? In 2016/2017 nearly 156,000 people were treated in a British hospital after a head injury. Approximately 1/5 will have features that suggest a skull fracture or have evidence of brain damage. The long-term effects of TBI can vary widely in severity, but include cognitive (thinking), functional (activity), behavioral and emotional difficulties[37].

Swelling or bleeding in or around the brain due to TBI can increase the pressure within the skull, causing damage to the brain or restrict blood supply. Hypothermia can be effective in reducing intracranial pressure, but it is not clear whether it improves survival or brain function[17,18),37]. Prophylactic hypothermia made no difference to favorable neurologic outcome at 6 months. At 6 months, there was no difference in mortality between the hypothermic and usual care groups (21.1% vs. 18.4%). There is also no difference in the time of death. At 10 days, there were slightly more side effects in the hypothermic group, but not significant. Pneumonia 55.0% in hypothermia and 51.3% in treatment[5,17,38)]

The National Institute for Health and Care Excellence 2017 (NICE) guidelines on the management of head injuries, do not mention the use of therapeutic hypothermia.

Pooled results from 6 medium-quality randomized controlled trials suggest that hypothermia can

improve outcomes neurologic disease, but no consistent or statistically significant reduction in mortality[39].

Finally prophylactic or therapeutic hypothermia appears to have no place in the management of severe brain injury, Prophylactic Hypothermia: Early (within 2.5 hours), short-term (48 hours after injury), prophylactic hypothermia is not recommended to improve outcome in patients with diffuse injury[5,39.40].

Conclusions

The conclusions that can be drawn from this paper are: not much has changed from the 2016 Brain Trauma Foundation Guideline until now. Maintain CPP 60–70 mmHg, and Avoid systolic blood pressure <110 mmHg. Target PaCO2 regulation is normocarbia, PaCO2 35–40 mmHg, hypercarbia will be increase CBF and abolish the autoregulatory curve while hypocarbia maintains the autoregulatory curve by decreasing CBF. The prophylactic use of phenytoin or valproate is not recommended to prevent late PTS. It is still necessary to analyze DECRA therapy with continuous medical therapy for refractory increase in ICP after TBI. The target blood sugar is normoglycemia.

Prophylactic or therapeutic hypothermia appears to have no place in the management of severe brain injury. General anesthesia for patients with severe TBI, preferably with total intravenous anesthesia (TIVA). Fluid administration must consider the osmolarity of the fluid and there is no difference in the outcome of administering mannitol with HTS.

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