Anesthetic Considerations for Traumatic Brain Injury

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SECTION 2. 마취관리 PART II (Anesthetic Management) 마취



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Management of traumatic brain injury patients

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Brain Trauma FOUNDATION

Qureshi et al. Clin Med Rev Case Rep 2017, 4:159 DOI: 10.23937/2378-3656/1410159

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ORIGINAL REVIEW

Anesthetic Management of Traumatic Brain Injury

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Pathophysiology of TBI

- Primary injury
 - The initial injury due to physical or mechanical forces on the brain parenchyma and skull
 - Leads to an inflammatory cascade cerebral edema, axonal injury, decreased cerebral perfusion pressure
- Secondary injury
 - Electrolyte abnormalities, hypoxemia, glycemic imbalance, hypotension, increased intracranial pressure, hyper or hypocarbia

Preoperative management

• Should avoid hypercarbia related to the administration of hypnotic agents or sedatives such as benzodiazepines, narcotics, etc., prior to induction of anesthesia.

Evaluation of TBI patients

- Careful airway assessments
- Thorough neurological examination to determine baseline sensation, motor function, and the presence of new focal neurological deficits to establish degree of TBI or cervical spine injury severity.
- Other traumatic related injuries such as bleeding, pneumothorax, cardiac tamponade, etc.

CSI after TBI

• Early assessment of cervical spine integrity is essential to rule out a hidden cervical spine fracture, especially in the TBI patient.

Injury Severity (GCS score)	Total Number of Trauma Admission	Number of Pts ^a with C-Spine Injury (%)		
13–15	14,088	198 (1.4)		
9-12	383	26 (6.8)		
≤8	667	68 (10.2)		

^a Pts, patients.

Demetriades, et al. J Trauma Inj Infect Crit Care 48: 724-727, 2000.

Initial management of a TBI patient with possible CSI

- Prevention of further neurological injury: Critical
- Stabilizing the cervical spine
 - Use of cervical collar
 - Interfere with direct laryngoscope during intubation
 - Removal of only the anterior portion of cervical collar prior to intubation
- Maintaining spinal alignment
 - Manual In-Line Immobilization (MILI)
 - Application of cricoid pressure
 - Removal of anterior half of the cervical collar
 - Manual posterior cervical spine support



- Manual In Line Immobilization (MILI) of the cervical spine Approach from the head of
- MILI of the cervical spine. Approach from the lateral side of the patient to facilitate airway management from the head of the bed.
- In addition to MILI, if the clinician is going to apply cricoid pressure, manual posterior cervical spine support is recommended to decrease cervical spine movement.

Keep airway

- **Orotracheal intubation** after rapid sequence induction and direct laryngoscopy, video laryngoscopes, or fiberoptic bronchoscopes with MILI
- Nasotracheal intubation with flexible bronchoscopy
 - Contraindication in suspected basal skull fracture
 - Trauma to the nares and epistaxis
- Awake fiberoptic approach
 - Experienced practitioner
 - Maintains cervical spine in a neutral position, preserves airway reflexes, allows simultaneous neurological assessments.
- LMA
 - Downward pressure produce a potential displacement of the upper cervical spine.
- Surgical airway, cricothyroidotomy

Muscle relaxants

- Succinylcholine is choice for rapid sequence induction
 - Negative effect: a transient increase in ICP
 - Positive effect: rapid onset and short duration of action, prevention of coughing during direct laryngoscopy
 - Prevent its side effect by a defasciculating dose of a non-depolarizing muscle relaxant.
- Rocuronium 0.9-1.2 mg/kg
 - Intubation conditions at 60-90 seconds
 - No transient increase in ICP
 - Muscle paralysis last for 30-40 minutes.

Ventilation in TBI

Goals of Intubation

- Prevention of aspiration of gastric contents
- Prevention of hypoxia and hypercarbia
 - Tissue hypoxia leads to release of catecholamines which further dilate cerebral veins and increase ICP
 - Hypercarbia: cerebral veins dilate, causing an increase in ICP
- Avoid a PaO₂ less than 60 mmHg
- Maintain an oxygen saturation greater than 90%
- Excessive hyperventilation can lead to cerebral vasoconstriction and oxygen deprivation.

Induction agents

• Ketamine

- Positive effects : the blockade of reuptake of catecholamines, which can prevent hypotensive episodes by maintaining MAP and CPP within normal range.
- Logically ketamine should be avoided in hypertensive TBI patients due to the risk of further elevating BP and consequently increasing ICP.
- In TBI patients with suspected elevated ICP and low-to-normal blood pressure, ketamine use might be indicated to preserve normotension during induction of anesthesia.

• Etomidate

- another appropriate induction agent choice in hemodynamically unstable patients.
- Negative effects: a dose dependent inhibition of 11-beta-hydroxylase and 17-alphahydroxylase leading to adrenal suppression.
 - This complication can occur after a single dose and may cause maximal adrenal suppression 4 to 6 hours after its administration.

• Propofol

- Positive effects
 - Quick onset and offset of action
 - Decrease neuronal oxidative stress.
- Negative effects
 - Sympathetic blockade resulting in hypotension.
 - Propofol infusion syndrome. This condition occurs generally if propofol is delivered for more than 48 hours, at doses above 4 mg/kg/hr.
 - Although propofol is recommended for the control of ICP, it is not recommended for improvement in mortality or 6-month outcomes. High dose propofol can produce significant morbidity (Level IIB)
- Indicated in the treatment of refractory status epilepticus with a recommended starting loading dose of 1 mg/kg.

• Dexmedetomidine

- Alpha-2 receptor agonist, it exerts its effects in the locus coeruleus.
- Despite its sedative and anxiolytic action it preserves adequate respiratory function when compared with benzodiazepines or narcotics. This property makes it an ideal agent in the non intubated TBI patient.
- In the Intensive Care Unit (ICU) setting dexemedetomidine has proved to decrease the incidence of delirium. Further research is needed to determine the impact of dexemedetomidine in the outcomes of TBI patients, however the data presented makes it suitable alternative to propofol for sedation purposes.

• Opioids

- Positive effect
 - Suppress airway reflexes, decrease required dose of induction agents and inhalation anesthetic maintenance as well as to blunt the sympathetic response to direct laryngoscopy.
- Negative effect
 - hypotension secondary to a reduction in sympathetic tone and potential histamine release
- Fentanyl, sufentanil, and remifentanil are commonly used in TBI patients.
- Careful opioid titration should be observed to avoid hypotension.

Maintenance of anesthesia

- Intravenous anesthetics (**sodium thiopental**, etomidate, midazolam and propofol)
 - decrease CBF, CBV, CMRO₂ and ICP under controlled ventilation conditions.
 - They achieve these effects by producing cerebral vasoconstriction and acting at the neurons' GABA receptors to open chloride channels.
 - Do not use barbiturates to induce burse suppression measured by EEG as prophylaxis against the development of intracranial hypertension (Level IIB)
 - Use high-dose barbiturates to control elevated ICP refractory to maximum standard medical and surgical treatment (Level IIB)



Intraoperative management

- Choice of anesthetic drugs: no ideal anesthetics
- The BTF clinical guidelines (should <u>avoid</u>)
 - Hypoxemia
 - PaO₂ below 60 mmHg,
 - Oxygen saturation below 90%,
 - Hypercarbia
 - Hypotension (systolic blood pressure below 90 mmHg)



그림 22-2. 두개내압(ICP), 동맥혈산소분압(PaO₂), 동맥혈이산화탄소분 압(PaCO₂), 평균동맥압(MAP)의 변화에 따른 뇌혈류의 변화

Blood pressure

- The current recommendations are to keep the systolic blood pressure above <u>90 mmHg</u> and the CPP between 50 and 70 mm Hg to avoid further brain ischemia.
 - Maintaining SBP at ≥100mmHg for patients 50-69 or at ≥110mmHg for patients 15-49 or over 70 years old may be considered to decrease mortality and improve outcomes (Level III).
 - The recommended target CPP value for survival and favorable outcomes is between 60 and 70mmHg; however, the optimal threshold is unclear and may depend upon the patient's autoregulatory status (Level IIB)



Management of hypotension

- Incidence of hypotension in TBI patients of around 34.6%, but what was of great concern is that in this subset of patients there was a 150% mortality increased.
- Sookplung, et al. examined patients with severe TBI who received phenylephrine, norepinephrine, or dopamine.
 - Based on this study, phenylephrine had the greatest increase in MAP and CPP.
 - It was unclear whether the improved MAP and CPP improved CBF and oxygenation.
 - In conclusion, the best choice of vasopressor for patients with TBI remains unclear.

Management of hypertension

- The ideal medication for treatment of hypertension should be one that is easily titratable and <u>should not cause cerebral vasodilatation</u> such as nitroglycerine, nitroprusside, and hydralazine to avoid further increase in ICP.
- Therefore the antihypertensive drugs recommended include propranolol, esmolol, labetalol, and nicardipine.

Robertson CS, et al. J Neurosurgery 59: 455-460, 1983.

Management of ICP

- The Brain Trauma Foundation states that ICP > 20 mmHg is associated with increased mortality and worse outcomes.
- **The fastest way** to decrease ICP > 20 mmHg is to allow Cerebrospinal Fluid (CSF) drainage from a CSF drain if present.
- Another relatively quick and effective alternative is to elevate the patient's head and maintain the neck in a neutral position, to improve venous blood return.
- Less rapid methods include slow administration of 0.25-1 gm/kg of mannitol in stable patients over 15 minutes. (Level II in 3rd Edition)
 - This can result in an ICP reduction, a transient increase in oxygen transport, and an increase in cerebral blood flow.
 - Additional dosing at a rate of 0.25-0.5 gm/kg can be repeated every six to eight hours.
 - Importantly, when using mannitol, it is important to monitor and replace urinary loses to prevent intravascular volume depletion and hypotension.
- **Hypertonic saline**: an average reduction in ICP ranging from 20-60% with time to peak effect range between 10 minutes and 5 hours post infusion.
- Hyperventilation can temporarily treat intracranial hypertension.
 - Maintaining a normal PaCO₂ of 35-40 mmHg is recommended in TBI patients to improve cerebral perfusion unless signs and symptoms of increased ICP are present

Fluid resuscitation for TBI

- Hypotonic solutions are contraindicated bec that might lead to cerebral edema and worse
- 0.9% normal saline solutions are indicated b isotonic than Ringer's lactate.
- Glucose containing solutions should be avoi is present.
- Fluid resuscitation with albumin was association with patients receivir (mmHg) and the patients receivir (mmHg) and the

J Neurotrauma 2013; 30(7): 512–518.



ICP monitoring ceased during second week (day 8-14)



ICP monitoring ceased during first week (day 1-7)

Coagulopathy and hemoglobin level

- INR in TBI patients should be maintained less than or equal to 1.4 and the platelet count maintained above 75 k/uL.
- Hemoglobin levels should be maintained at or above 7 g/dl to avoid a decrease in brain oxygen delivery.

Glycemic control

• The presence of hyperglycemia might produce an increase in neuronal metabolism and increase neuronal death after TBI. These events occur due to increased tissue acidosis through anaerobic metabolism, the creation of free radicals, and increased blood brain barrier permeability. Therefore the ideal blood glucose level should range from 80-180

mg/dl.



J Trauma 2005; 58: 47-50.

Thermoregulation

- No benefit of induced hypothermia on mortality or neurological outcomes after TBI. (Clifton, et al. Lancet Neurol 2011; 10: 131-139.)
 - it is important to remember that fever worsens the severity of brain injury by increasing cerebral metabolic rate.
 - In addition, early hyperthermia after TBI has been found to be a possible predictor of paroxysmal sympathetic hyperactivity (Hinson HE, et al. J Head Trauma Rehabil. 2017 Sep-Oct; 32(5): E50–E54)
- The final BTF recommendation : to avoid hyperthermia and to maintain normothermia with antipyretics and surface cooling devices.
 - Early (within 2.5 hours), short-term (48 hours post-injury) prophylactic hypothermia is not recommended (Level IIB)



Outcome and mortality rates

	Poor outcome			Died		
	n (%)	RR (95% CI)	p value	n (%)	RR (95% CI)	p value
Primary analysis						
All patients (n=97)	56 (58%)	- 21	34	20 (21%)	10210	
Hypothermia (n=52)	31 (60%)	1.08 (0.76-1.53)	0.67	12 (23%)	1.30 (0.58-2.89)	0.52
Normothermia (n=45)	25 (56%)	56 C	55	8 (18%)	12.5	11.0
Diffuse brain injury (n=69)	42 (61%)	35	59	13 (19%)	5453	-
Subgroup analysis	12 (010)			(1.20)	227.91	2011
Hypothermia (n=37)	26 (70%)	1.44 (0.95–2.17)	0.09	10 (27%)	2.88 (0.87-9.57)	0.08
Normothermia (n=32)	16 (50%)	36.	- 10	3 (9%)	(#)	14 C
Surgically removed haematomas (n=28)	14 (50%)	37		7 (25%)	(445)	-
Hypothermia (n=15)	5 (33%)	0.44 (0.22-0.88)	0.02	2 (13%)	0.35 (0.08–1.50)	0 16
		22.1		5 (39%)	14245	144

Data are number (%). RR=relative risk.

Clifton, et al. Lancet Neurol 2011; 10: 131-139.

그림 22-1. 체온과 뇌대사율(CMRO₂)의 관계

Maintenance of parameters in general

- MAP > 80 mmHg or SBP > 100 mmHg
- Hb > 7 g/dl
- PaO₂ 60-100 mmHg
- PaCO₂ 35-40 mmHg
- PLT > 50,000/mm³ , A greater PLT count would be advisable in emergency neurosurgery
- PT/aPTT < 1.5 normal control
- If massive transfusion, start with 1 RBCs/1 Plasma/1 PLTs
- TEG/ROTEM, if available
- Osmotherapy in case of impending cerebral herniation or cerebral edema
- CPP \geq 60 mmHg

Picetti et al. World Journal of Emergency Surgery (2019) 14:53

Take Home Message

- Recognizing CSI
- Control
 - Hypoxia
 - Hypocarbia
 - Hypotension
 - ICP

