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# Oxygenation and Hemodynamic Changes in Traumatic Brain Injury: A Literature Review

**Riyadh Firdaus[1], Aida Rosita Tantri[1*]**

[1] Department of Anesthesiology and Intensive Care, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

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***Corresponding author:**
Aida Rosita Tantri

**E-mail address:**
*aidatantri@gmail.com*

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**ABSTRACT**

Traumatic brain injury (TBI) is a major public health problem and the main cause of death and disability worldwide. TBI can causing primary and secondary injury. Primary brain injury occurs within a moment after a collision and worsen by acute systemic damage such as hypoxia, bleeding, and neurotoxic pathway activation. Under normal conditions, brain has several mechanisms for regulating pressure and volume to prevent ischemia. The purpose of these mechanisms is to maintain a continuous cerebral blood flow (CBF) and adequate oxygen supply.

## 1. Introduction

Traumatic brain injury (TBI) is a major public health problem and the main cause of death and disability worldwide.[1] TBI is a major cause of mortality in the United States, contributing to 30% of all injury-related deaths. TBI occurs most often in children aged 0–4 years, adolescents aged 15–19 years and elderly aged 65 years and more.[2]

TBI can causing primary and secondary injury. Primary brain injury occurs within a moment after a collision and worsen by acute systemic damage such as hypoxia, bleeding, and neurotoxic pathway activation.[3] Then, hypoxia activates secondary brain injury cascade pathway (blood brain barrier damage, excitotoxicity, oxidative stress, mitochondria dysfunction, and neuroinflammation) that causing cell death. All these processes contribute to neurological deficits separately, but at the same time, these cell death processes interact, worsening the progressive outcome of TBI.[4] Therefore, it is essential to learn more about oxygenation and hemodynamic changes in traumatic brain injury.

### Traumatic brain injury

Traumatic brain injury (TBI) is divided in two,

primary TBI and secondary TBI. Primary TBI is defined as a trauma that is caused by direct mechanical force in the brain tissue. This condition happens when the trauma occurs. Secondary TBI happens when primary TBI causing continuous trauma that appear slowly.[4-8] The impact of secondary TBI caused by dysregulation of brain vessels and blood–brain barrier (BBB) disruption may be magnified by these processes, leading to the development of brain edema, increased intracranial pressure (ICP), and finally, decreased cerebral perfusion pressure.[9]

**Hemodynamic regulation in brain**

Under normal conditions, brain has several mechanisms for regulating pressure and volume. The purpose of these mechanisms is to maintain a continuous cerebral blood flow (CBF) and adequate oxygen supply. This regulation is very essential especially when there is changes in both systemic arterial pressure (SAP) and cerebral metabolic requirements. The key mechanism is the change in cerebrovascular resistance through vasoconstriction and vasodilatation of brain vessels. Under normal circumstances, an increase in SAP will lead to increased cerebrovascular resistance, thus keeping the CBF constant with 60-160mmHg mean arterial pressure (MAP).[9]

**Hemodynamic regulation in TBI**

When the autoregulation mechanism fails and the BBB is also disrupted, the CBF becomes dependent on SAP. This condition leads to harmful and critical conditions, such as hypoperfusion (brain ischemia) or hyperperfusion (e.g., hyperemia). These may lead to an irreversible increase in ICP, make the autoregulation curve shifts to the right when the MAP are above 160mmHg.

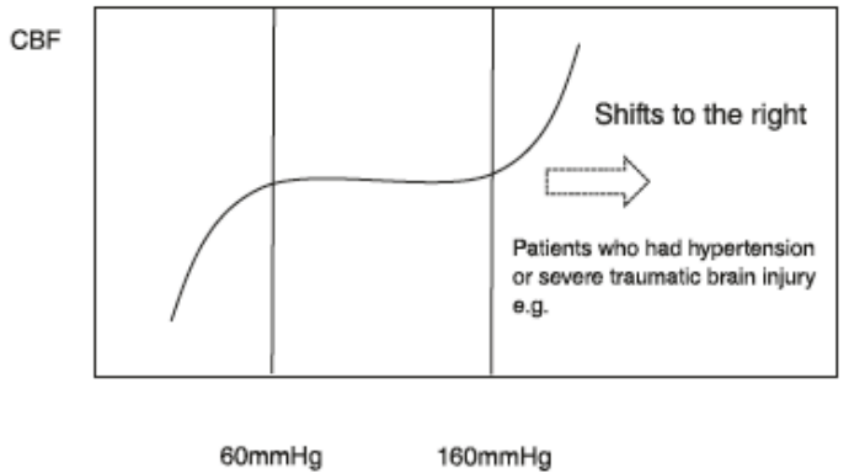


Figure 1. Shift to the right in TBI. (source: Kinoshita K. Traumatic brain injury: pathophysiology for neurocritical care. Journal of Intensive Care. 2016;4(1))

**Hyperemia after TBI**

Hyperemia is associated with elevated blood volume in brain and decreasing distal cerebrovascular resistance. Many drivers, such as lactic acid, neuropeptides, and adenosine, regulated by vasodilatory metabolites, have been considered to be part of the mechanism for causing a decrease in distal cerebrovascular resistance. Alternatively, dysfunctional pressure or volume autoregulation may stimulate hyperemia that is associated with intracranial hypertension. Hyperemia combines with BBB disruption may cause brain edema. Increased CBF and blood volume in brain due to vasodilation with BBB disruption may lead to aggravated vascular engorgement and brain edema, developing irreversible intracranial hypertension. In the patient with intact

vasoconstriction cascade, hyperventilation therapy may reduce PaCO2 levels, which might appropriate for treating brain edema.[9]

**Oxygenation after TBI**

CBF is significantly influenced by PaCO2 levels. When PaCO2 drops below 20mmHg, vasoconstriction of brain blood vessel occurs, leading to a decrease in ICP as the final outcome. Many conditions are responsible for maintain oxygenation balance in brain, divided into extracranial and intracranial causes. Hypoxia, hypotension, hypo/hyper PaCO2, and anemia are extracranial causes while intracranial causes is increased in ICP. When there is a rise in PaCO2 may induce a brain blood vessel dilatation causing ICP increase and contribute to an increase in cerebral blood volume leading to brain edema, likely resulting in a poor outcome for patients. On the other hand, when PaCO2 drops, vasoconstriction of brain blood vessel occurs, leading to a decrease in ICP and cerebral blood volume.[9]

Patients that experience TBI often develop hypercapnia, a condition where CO2 level in circulation is rising. When hypercapnia develops after a TBI that can be caused by airway obstruction or respiratory insult, hyperventilation therapy is mandatory for decreasing the ICP.[9,10]

**Hemodynamic after TBI**

In an emergency state, elevating SAP with large-volume fluid resuscitation or blood transfusion is one critical approach for patients with severe TBI, although these approach may aggravate brain edema and increase ICP. Brain edema after TBI can be of cytotoxic or vasogenic origin or may be caused by capillary leakage. Vasogenic origin is mainly a damage in blood vessel. This process lead by tight junction disruption that causing intravascular and interstitial space pressure gradient thus lead to fluid extravasation. This condition will occupy extracellular space. Fluid with protein that enter interstitial space increasing oncotic pressure and causing small vessel occlusion, therefore hypoperfusion or even local ischemia happen.[10]

## 2. Conclusion

Under normal conditions, brain has several mechanisms for regulating pressure and volume. The purpose of these mechanisms is to maintain a continuous cerebral blood flow (CBF) and adequate oxygen supply. This regulation is very essential especially when there is changes in both systemic arterial pressure (SAP) and cerebral metabolic requirements. When the autoregulation mechanism fails and the BBB is also disrupted, the CBF becomes dependent on SAP. This condition leads to harmful and critical conditions, such as hypoperfusion (brain ischemia) or hyperperfusion (e.g., hyperemia). These may lead to an irreversible increase in ICP, make the autoregulation curve shifts to the right. With all that reason, hyperventilation therapy is mandatory for decreasing the ICP.

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