

MANAGEMENT OF INTRACRANIAL HIGH PRESSURE ON STROKE: A NARRATIVE REVIEW

FRANSISCA KRISTINA ELISABET*

Research Assistant at Infectious Disease Control and Management (TB-HIV Working Group), Padjadjaran University, Bandung, West Java, Indonesia. Email: fransiscakristina24@gmail.com

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ABSTRACT

Intracranial pressure (ICP) is a dynamic and fluctuating pressure within the cranial vault influenced by cerebrospinal fluid (CSF), brain tissue, and blood. Any increase in the volume of its contents will increase the pressure within the cranial vault. As a response to ICP, compensation begins by the movement of CSF from the ventricle to cerebral subarachnoid space and increases CSF absorption. Increased ICP is a state of neurological emergency caused by various neurological injuries. It is associated with poor outcomes, including traumatic brain injury, large acute ischemic stroke, intracerebral hemorrhage, or even death. This article aims to review the symptoms and signs of increased ICP, particularly on infarction stroke and hemorrhagic stroke condition, how the therapeutic goals of the treatment strategies and pharmacological or non-pharmacological management to mitigate the elevation of ICP on stroke.

Keywords: Cranial vault, Hemorrhage stroke, Intracranial high pressure (ICP), Management, Stroke infarction, Treatment.

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INTRODUCTION

Intracranial high pressure or intracranial hypertension (IH) is a clinical condition associated with an elevation of the intracranial pressure (ICP) within the cranium of the skull. The cranial vault's pressure is in millimeters of mercury (mmHg) and usually is <20 mmHg [1-3]. Due to the skull's fixed nature, an increase in the volume of any one of the intracranial components will also cause an increase in pressure. If one of the elements within the cranium increases, a different component's volume must decrease to maintain this equilibrium and sustain a normal ICP. Many disease processes can result in IH, including traumatic brain injury, large acute ischemic stroke, and intracerebral hemorrhage (ICH).

THE FUNDAMENTAL OF ICP

The Monro-Kellie doctrine states that the intracranial space is a fixed volume inside the skull [3]. This intracranial space has three components: Brain tissue, blood, and cerebrospinal fluid (CSF). In an average adult, the brain tissue volume is 1400 mL; the blood volume is 150 mL; and the CSF volume is 150 mL [3]. Normal ICP ranges 3–15 mmHg; in the intensive care unit, ICP values <20 mmHg are still acceptable. The pathological status in the intracranial vault that results in increased volume may be associated with the extrinsic mass lesion, increase in blood volume, increase in CSF volume, and increase in brain tissue.

There is a reduction in the volume of other compartments as a compensatory mechanism to maintain the normal value of ICP. As compliance decreases, there is a more significant change in pressure for a given change in volume (i.e., there are dramatic changes in pressure for small incremental changes in volume) (Fig. 1). This ICP is reflected in the ICP waveform, as shown in Fig. 2.

Changes in ICP are associated with volume changes in one or more constituents inside the cranial vault [4]. The cranial vault and spinal canal, along with the relatively inelastic dura, form a rigid vessel. Hence, any increase in brain, blood, or CSF will tend to increase ICP. Moreover, any enhancement of one component must be at the other two's expense (Monro-Kellie's doctrine, Fig. 3). The small increase in brain volume does not lead to an immediate increase of ICP, because CSF is transferred to the spinal canal and stretching the cerebral falx.

However, once the ICP has reached about 25 mmHg, a small increase in brain volume can increase ICP [5-8].

The cerebrovascular circulation is a complex network consisting of arteries and veins. The pressure difference that forces blood to enter this system is known as the cerebral perfusion pressure (CPP). We define CPP as mean arterial pressure (MAP) minus ICP (i.e., $CPP = MAP - ICP$). Therefore, as ICP rises, CPP will fall. Normal CPP is 60–150 mmHg; CPP <60 mmHg may result in ischemic brain injury; while CPP >150 mmHg can lead to hyperemia and cerebral hyperperfusion injury [9]. Cerebral blood flow (CBF) is the blood flow supply to the brain. In adults, normal CBF is around 15% of cardiac output. We can calculate CBF values with CPP values divided by cerebrovascular resistance (CVR) values. We can simplify CPP approximately as the same inlet pressure with MAP minus the exit pressure equal to the ICP ($CPP = MAP - ICP$) [4,7,8,10].

PATHOPHYSIOLOGY OF INCREASED ICP ON STROKE

The main pathophysiology's of ICP consists of cerebral edema, obstruction of CSF flow, increased cerebral blood volume, and expansion of brain mass. Cerebral edema is a complication of all types of stroke. The timing varies from the first 24 h to approximately 72 h after onset [11]. Cerebral edema can be divided into cytotoxic, vasogenic, and interstitial edema (IE) [12].

Cytotoxic edema (CE)

CE is an inflammation of the brain cell elements (neurons, glia, and endothelial cells) due to metabolism failure of cellular energy. It is typically accompanied by brain swelling and can result from almost any insult to the brain, including trauma, infarction, neoplasm, abscess, or conditions such as hypoxia, toxic, and metabolic perturbation [12].

Vasogenic edema (VE)

VE occurs due to increased permeability of the blood-brain barrier (BBB) against serum elements. Its development reflects damage to the endothelial cells that form the BBB.

Interstitial edema (IE)

IE occurs due to osmotic differences between plasma and brain tissue. In general, it happens in obstruction hydrocephalus that increases transependymal CSF flow. There is an increase in sodium and water

in the periventricular substantia alba (white matter) with the displacement of CSF passing through the ventricular epithelium.

INTRACRANIAL HIGH PRESSURE ON STROKE CASES

Stroke infarction

Ischemic areas are formed due to decreased regional CBF of the brain isolated from the bloodstream. In that region, we find: (1) Low perfusion pressure, (2) decreased pressure of oxygen, and (3) CO₂ and accumulated lactic acid. Autoregulation and vasomotor in that area work together to overcome ischemic conditions by providing maximum vasodilation. Collateral vasodilation occurred at the border of the ischemic areas so

that we can save it. Central ischemic areas that cannot be resolved by autoregulation mechanisms and vasomotor management will develop irreversible degeneration processes. All blood vessels in the central part of the ischemic area lose tone and therefore in a vasoparalysis state. The picture is appropriate with the ischemic circumstances. We can repair because blood vessels' smooth muscle cells can survive long enough in an anoxic environment. However, the brain cells of the ischemic region do not stay long. Swelling of nerve fibers and myelin sheath (cerebral edema) is an early degenerative reaction followed by diapedesis of erythrocytes and leukocytes. Eventually, the nerve cells are destroyed, resulting in an infarct image [13,14].

In areas of edema, cerebral autoregulation is impaired. Vasodilators dilate the lumen of healthy cerebral blood vessels, but the swollen area's veins cannot do the same. Blood will flow through the dilated healthy cerebral arteries. Thereby, only healthy brain tissue will receive it. This phenomenon is known as cerebral steal syndrome. On the contrary, vasoconstriction due to hypocapnia or specific anesthetic agents, such as thiopentone, will reduce blood flow to healthy brain tissue, resulting in a redistribution of blood ischemic areas. This phenomenon is called inverse steal syndrome [15].

Cerebral edema increases the resistance, thereby decreasing blood flow to the brain [14]. Efforts to improve regional cerebral ischemia will be fruitless if the cerebral edema region is not eradicated quickly. A reduction in total CBF will reduce regional CBF. The first action in the acute stage of ischemic stroke aims to maintain optimal perfusion pressure. For high systemic blood pressure to increase total CBF, intracranial resistance due to cerebral edema must be overcome [14]. CE occurs in the 1st h after stroke ischemia onset, and we can detect a decrease in the apparent diffusion coefficient (ADC) of water. We also observe a reduction of ADC in acute hemorrhage stroke, but it is different from ischemic stroke. ADC continues to decline for up to 100 days after onset [15].

Normal cell volume depends on the electrolyte balance, also extracellular and intracellular fluid exchanges. This balance depends on the low permeability of the membrane to sodium, the energy-dependent membrane pump, and the energy supply in adenosine triphosphate (ATP). When the ischemic process becomes an infarction, the homeostatic function that controls cell volume and the BBB permeability is impaired because the ATP-dependent Na⁺/K⁺ ATPase pump cannot function without ATP. This infarction results in the entry of Na⁺ into the cells, followed by Cl⁻ and water, resulting in edema. This cascade impairs control mechanisms of the cell volume and develops CE [14]. As with the CE that precedes it, VE also grows due to energy failure, glutamate release, and increased intracellular calcium concentration. Free radicals also play a role in the formation of ischemic VE [14]. This edema process will increase ICP, which occurs in large stroke infarction.

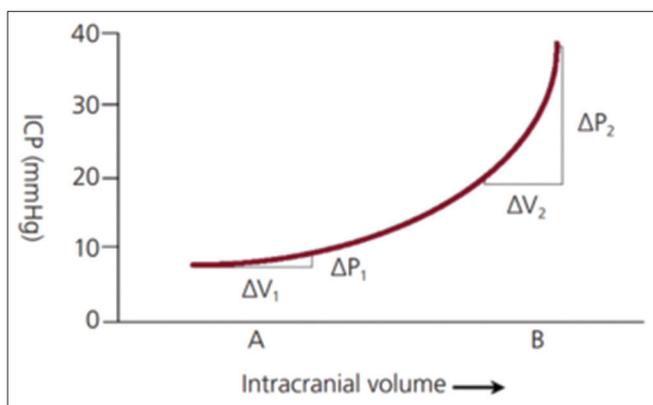


Fig. 1: Intracranial pressure-volume relationship

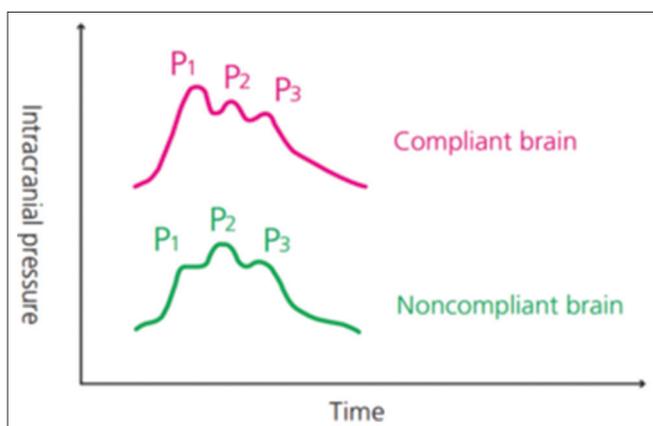


Fig. 2: Intracranial pressure waveform. Normal compliance and decreased compliance

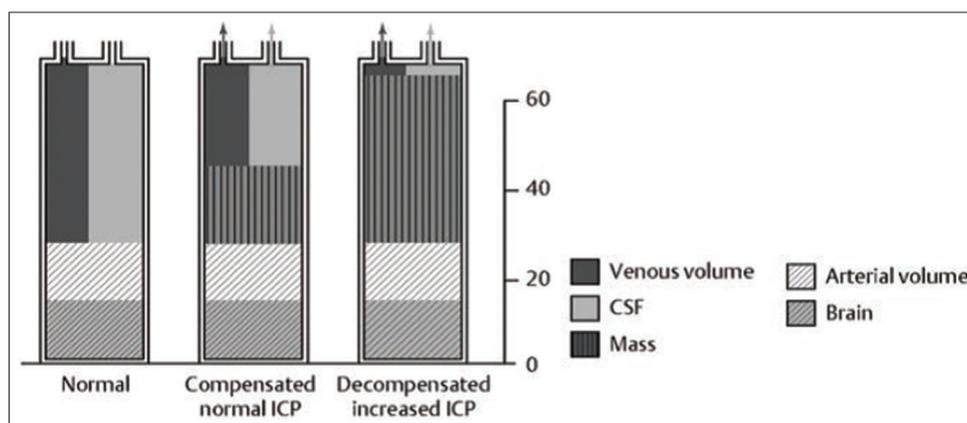


Fig. 3: Compensated intracranial pressure (ICP). The intracranial space's normal condition includes the parenchyma brain, arterial volume, venous blood, and cerebrospinal fluid (CSF). If there is a mass, there are blood suction and CSF to reach the compensated normal ICP.

Hemorrhage stroke

Perihematomal edema appears in the first few days after the onset of ICH. Experimental studies suggest that the multifactorial perihematomal edema mechanism can form CE and/or VE [16]. In an ICH, CE is predominantly present. This edema is related to the size of the hematoma, not to the severity of the hypoperfusion. Perihematomal CE may be associated with the accumulation of cytotoxic factors such as thrombin or iron.

The formation of edema in ICH occurs through the three stages; in the first few hours after ICH, there is a clot formation. Intact red blood cells in the area of the hematoma do not cause edema. After the coagulation cascade becomes active over the next 24–48 h, thrombin becomes active and impairs the BBBs integrity, allowing intravascular fluid to enter the extracellular space. The third phase occurs when the hematoma red blood cells begin to lysis. Hemoglobin and its degradation products are stored in the brain parenchyma, causing a potent inflammatory reaction and edema formation. Blood in the subarachnoid causes vasospasm that increases CVR, decreases CBF, even though CPP is normal [17].

The ICP will continue to increase, thereby endangering the CPP. The CPP will be zero if the ICP is equal to the mean artery. The brain becomes ischemic with irreversible neurological damage. Brain death occurs when the ICP equals arterial pressure [5,10].

MANIFESTATIONS OF ICP AND ITS MANAGEMENT

Early signs of ICP are headache, vomiting, seizure, and decreased consciousness. Several complications include breathing and airway problems, reduced life span, difficulty communicating, permanent loss of brain functions and movement, or sensation [11]. Moreover, patients may develop altered midline shifts that show dilated or unreactive pupils, asymmetric pupils, extensor posturing, progressive neurologic deterioration, and decrease in the Glasgow Coma Scale score [13]. We can observe other manifestations such as papilledema, bradycardia, progressive increase in blood pressure, and endocrine disorders. In children, the head circumference may be enlarged by widening the cranial sutures. The most common neurological disorders are paralysis of the VI and III nerves and positive Babinski's sign on both sides [4-6,10,18,19].

Prompt recognition and aggressive management of complication may prevent permanent neurological dysfunction or death by the interdisciplinary [20]. Management of increased ICP has to be established into pharmacotherapy and non-pharmacotherapy approaches. Pharmacotherapy intervention comprises hyperosmolar therapy, euglycemia, hyperventilation, optimal oxygenation, controlling cerebral metabolism (sedation), barbiturate coma, anticonvulsant prophylaxis, maintenance of normothermia, and coagulation therapy. Furthermore, non-pharmacotherapy consists of positioning, initial trauma assessment (airway, breathing, and circulation), suctioning, control fluid and electrolytes, and nutrition [21].

Clinician and medical personnel should have sufficient knowledge and skills to support ventilation and deal with oxygenation problems (airway obstruction, increased PaCO₂, and hypoxemia), positioning (head on the bed 15–30 degree), reduce increased metabolic rate, reduce stressors (pain, disturbing conversation, noise, and bright lights), and others such as avoiding Valsalva maneuver, coughing, and vomiting. Furthermore, they have to establish a baseline neurologic assessment, patients' electrolytes, oxygen saturation, and carbon dioxide levels [22] and monitor cerebral tissue perfusion, fluid volume, breathing pattern, body temperature, risk infection, injury, and altered nutrition [23,24]. Initial and promptly assessment and early aggressive resuscitation of critically ill patients may prolong life.

CONCLUSION

The progressive increase in intracranial compartment volume can cause an increase in intracranial pressure. Increased ICP is an emergency case where we can avoid irreversible brain injury with appropriate

intervention on time. On stroke cases, the progressive increase of ICP very important and essential to determine the brain function. Increased ICP can also decrease the cerebral blood flow or brain herniation resulting in compression and cerebral ischemia. Common symptoms of ICP are headache, projectile vomiting, seizures, and changes in mental status, while the most reliable physical signs are papilledema. Handling ICP aims to reduce pressure intracranial, increase cerebral blood flow, and restore brain herniation. Management of increased intracranial pressure on stroke consists of pharmacotherapy and non-pharmacotherapy administration. The clinician and medical personnel should have sufficient knowledge and skills regarding ICP management on stroke to decrease its morbidity and mortality.

REFERENCES

- Marmarou A, Anderson RL, Ward JD, Choi SC, Young HF, Eisenberg HM, *et al.* Impact of ICP instability and hypotension on outcome in patients with severe head trauma. *J Neurosurg* 1991;75:S1-66.
- Cremer OL, van Dijk GW, van Wensen E, Brekelmans GJ, Moons KG, Leenen LP, *et al.* Effect of intracranial pressure monitoring and targeted intensive care on functional outcome after severe head injury. *Crit Care Med* 2005;33:2207-13.
- Mayer SA, Chong JY. Critical care management of increased intracranial pressure. *J Intensive Care Med* 2002;17:55-67.
- Snell RS. *Clinical Neuroanatomy for Medical Student*. 3rd ed. Boston: Little Brown and Company; 1982. p. 1-9.
- Lee KR, Hooff JT. Intracranial pressure. In: *Neurological Surgery*. 4th ed. Philadelphia, PA: WB Saunders Company; 1996. p. 491-514.
- Lindsay KW, Bone I, Callander R. *Neurology and Neurosurgery Illustrated*. 3rd ed. New York: Churchill Livingstone; 1997. p. 72-80.
- Victor M, Ropper AH. *Adam and Victor's Principles of Neurology*. 7th ed. New York: McGraw-Hill Medical Publishing Division; 2001. p. 655-74.
- Walter FJ. Intracranial pressure and cerebral blood flow. *Physiology* 1998;8:1-4.
- Latorre JG, Greer DM. Management of acute intracranial hypertension: A review. *Neurologist* 2009;15:193-207.
- Ward JD, Mopulton RJ, Muizelaar JP, Marmarou A. Cerebral homeostasis and protection. In: *Neurological Critical Care*. 7th ed., Vol. 1. Baltimore: Williams and Wilkins; 1991. p. 187-206.
- Rockoff MA, Ropper AH. Physiology and clinical aspect of raised intracranial pressure. In: *Neurological and Neurosurgical Intensive Care*. 3rd ed. New York: Raven Press; 1993. p. 11-66.
- Juul N, Duch B, Rasmussen M. Clinical management of patients with head injury. *Curr Anaesth Crit Care* 2009;20:132-7.
- Rosand J, Schwamm LH. Management of brain edema complicating stroke. *J Intensive Care Med* 2001;16:128-41.
- Raslan A, Bhardwaj A. Medical management of cerebral edema. *Neurosurg Focus* 2007;22:E12.
- Mishra LD. Cerebral blood flow and anaesthesia: A review. *Indian J Anaesth* 2002;46:87-95.
- Olivot JM, Mlynash M, Kleinman JT, Straka M, Venkatasubramanian C, Bammer R, *et al.* MRI profile of the perihematomal region in acute intracerebral hemorrhage. *Stroke* 2010;41:2681-3.
- Thiex R, Tsirka SE. Brain edema after intracerebral hemorrhage: Mechanisms, treatment options, management strategies, and operative indications. *Neurosurg Focus* 2007;22:E6.
- Miller JD, Piper IR, Statham PF. ICP monitoring: Indications and techniques. In: Narayan RK, Wreberger JE, Poulshock JT, editors. *Neurotrauma*. New York: McGraw Hill; 2002. p. 429-44.
- Ayata C, Ropper AH. Ischaemic brain oedema. *J Clin Neurosci* 2002;9:113-24.
- Posner JB, Plum F. *The Diagnosis of Stupor and Coma*. 3rd ed. England: Oxford University Press; 1983. p. 4-830.
- Ignatavicius DD, Workman ML. *Medical-Surgical Nursing: Critical Thinking for Collaborative Care*. St. Louis: Saunders; 2006. p. 1-3.
- Goh J, Gupta AK. The management of head injury and intracranial pressure. *Curr Anaesth Crit Care* 2002;13:129-37.
- Lejeune M, Howard T. Caring for patients with increased intracranial pressure: Find out what causes pressure to rise and how to intervene before complications occur. *Nursing* 2002;32:32cc31-5.
- Hudak CM, Gallo BM, Morton PG. *Critical Care Nursing: A Holistic Approach*. 7th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 1998. p. 1-7.