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Regulation and Intervention of Intracranial Pressure

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ABSTRACT

Intracranial pressure is the total amount of pressure exerted by the brain, blood and cerebrocinal fluid in the rigid cranial space. Compliance is an indicator of the brain's tolerance for increased ICP, when compliance is exceeded, there will be a dramatic increase in the pressure/volume curve so that ICP will increase rapidly. In the injured brain, cerebral blood flow (CBF) is regulated to supply sufficient oxygen and substrates to the brain. Certain physiological factors such as hypercarbia, acidosis and hypoxemia cause vasodilation which causes an increase in CBF, seizure activity and fever will increase the level of brain metabolism and CBF. Cerebral edema is the most common cause of non-traumatic brain injury such as central nervous system infections, metabolic and systemic encephalopathy. Vasogenic brain edema occurs due to injury to the blood-brain barrier and increased capillary permeability in the area around the injury, or to inflammation, especially in CNS infections. Medical management of elevated intracranial pressure includes sedation, cerebrospinal fluid drainage, and osmotherapy with either mannitol or hypertonic salts.

1. Introduction

Intracranial pressure (ICP) is the total amount of pressure exerted by the blood, brain, and cerebrospinal fluid in the rigid cranial space. Intracranial pressure is influenced by 3 factors, namely the brain (80%), cerebrospinal fluid (10%), and blood (10%). The range of normal intracranial pressure values varies with age, normal values are 10 to 15 mmHg in adults and the elderly, 3 to 7 mmHg for children and 1.5 to 6 mmHg in infants.[1,2]

Variations in contractile cardiac muscle have two distinct effects on intracranial dynamics, namely periodic changes in pressure and periodic changes in flow. While pressure and fluid flow are related physical phenomena, they must be considered separate things for a major reason; pressure pulses propagate through the brain at the speed of sound and the precise point for measurement is not a problem. Whereas fluid flow requires the movement of fluid from one compartment to another and the pulsation of the current varies depending on the location.[3]

Dynamics of Intracranial Fluid

Pressure is the total amount of pressure exerted by the brain, blood and cerebrocinal fluid in the rigid cranial space. Compliance is an indicator of the brain's tolerance for increased ICP, when compliance is exceeded, there will be a dramatic increase in the pressure/volume curve so that ICP will increase rapidly.

In the injured brain, cerebral blood flow (CBF) is

regulated to supply sufficient oxygen and substrates to the brain. Certain physiological factors such as hypercarbia, acidosis and hypoxemia cause vasodilation which causes an increase in CBF, seizure activity and fever will increase the level of brain metabolism and CBF.[4,5]

Cerebral cranial pressure (CCP) is the pressure at which the brain gets perfused. CCP allows indirect measurement of CBF adequacy. It is calculated by measuring the difference between mean arterial pressure (MAP) and ICP (MAP - ICP), where MAP = 1/3 systolic pressure plus 2/3 diastolic pressure. Normal values for CPP that are accepted as the minimum pressure needed to prevent ischemia are: adults > 70 mmHg; child > 50-60 mmHg; infants/toddlers > 40-50 mmHg. CCP <40 mmHg was a significant predictor of mortality in children with TBI.

Increased intracranial pressure

An increase in ICP is usually caused by an increase in brain volume (cerebral edema), blood (intracranial haemorrhage), space-occupying lesions, or CSF (hydrocephalus). The relationship between ICP and intracranial volume is depicted in the form of a curve (Figure 1) which is divided into three parts, namely the first part of the curve is flat because the compensatory reserve is adequate and ICP remains low even though the intracerebral volume (AB) is increased. When this compensatory mechanism is weak, the curve will rise rapidly. Intracranial compliance is greatly decreased and a slight increase in volume will lead to an increase in ICP (BC). At high ICP, the curve flattens again due to loss of capacity of cerebral arterioles to dilate in response to decreased CCP. High brain tissue pressure causes the failure of blood vessel function as a cerebrovascular response (C-D). [6]

Cerebral Edema is most often the cause of non-traumatic brain injury as the central nervous system infections, metabolic and systemic encephalopathy. Vasogenic brain edema occurs due to injury to the blood-brain barrier and increased capillary permeability in the area surrounding the injury, or to inflammation, especially in CNS infections. Interstitial cerebral edema occurs due to increased hydrostatic pressure of the CSF, often seen in patients with obstructive hydrocephalus or excessive CSF production.

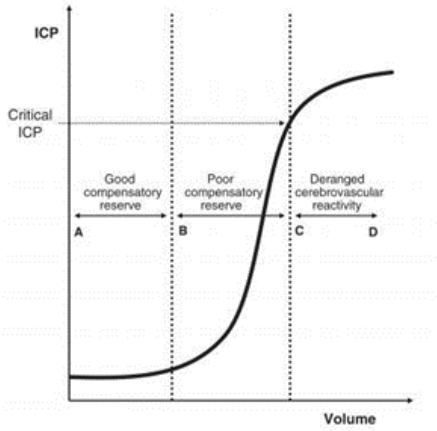

Figure 1. The curve of the relationship between ICP and intracranial volume Intracranial

Hypertension that occurs after traumatic brain injury or traumatic brain injury is multifactorial. Trauma due to subdural or epidural hematoma, hemorrhagic contusion, depressed skull fracture, cerebral edema, hyperemia due to loss of autoregulation, hypoventilation leading to hypercarbia and cerebral vasodilation, hydrocephalus due to obstruction of CSF flow or absorption.[7,8]

In conditions with increased intracranial pressure, the common symptoms found are headache, vomiting, papilledema, and neurological deficits. Headache is a common symptom of increased ICP. Headaches occur due to traction or distortion of arteries and veins and dura mater will give severe symptoms in the morning and are aggravated by activity, coughing, lifting, sneezing. Papilledema is caused by the transmission of

pressure through the sheath of the optic nerve that communicates with the subarachnoid space in the brain. This is a good clinical indicator of intracranial hypertension. Neurological deficits such as symptoms of altered level of consciousness; restlessness, irritability, lethargy, and decreased motor function. If the increase in ICP continues and is progressively associated with displacement of brain tissue, a herniation syndrome develops and the general signs of Cushing's triad (hypertension, bradycardia, irregular respirations) appear. Breathing patterns can help localize the level of injury.

Intracranial pressure monitoring procedure

Brain Trauma Foundation 2007 Recommends that ICP should be monitored in all patients with the following indications. Patients who should be monitored for ICP consist of severe head injuries with GCS 3 to 8 post resuscitation, abnormal CT scan results (hematoma, contusion, edema, herniation or compression of the basal cistern) and patients with severe head injury, with normal CT scans if followed or more following criteria; over 40 years of age, systolic blood pressure < 90 mmHg, with assisted ventilation, with motor posture. [9]

To determine intracranial pressure, there are two monitoring methods, namely invasive (direct) or non-invasive (indirect) methods. Non-invasive monitoring methods can be done by monitoring clinical status, neuroimaging and neurosonology. Several clinical conditions that must be assessed in increasing ICP are level of consciousness (GCS), pupillary examination, ocular motor examination (special attention to nerves III and VI), motor examination (presence of hemiparesis), presence of nausea or vomiting, headache and vital signs.

Ophthalmoscopy is one of the most significant assessments of ICP improvement. Papilledema is found when the increase in ICP has been present for more than a day. But should still be assessed at the initial evaluation, the presence or absence of papilledema can provide information about the course of the disease. In patients with suspected increased ICP, a head CT scan should be performed. Some findings on neuroimaging that suspected pathological conditions that cause increased ICP are intracranial hemorrhage, obstructive hydrocephalus, cerebral edema, midline shift, compression of the basal cistern, obliteration of the third ventricle.

Characteristics of CT scanning can provide an objective assessment of the structure of brain damage and can be associated with outcomes that occur after brain injury. The most frequently used classification for traumatic brain injury is the 1991 Marshall Classification. Where classifications I and II can predict better outcomes and classifications III and IV have worse outcomes.

Table 1. Marshall computerized tomography (CT) classification

Category	Definition
Diffuse injury I (no visible pathology)	No visible intracranial pathology seen on CT scan.
Diffuse injury II	Cisterns are present with mid-line shift 0-5 mm and / or lesion densities present; no high or mixed density lesion > 25 cc may include bone fragments and foreign bodies.
Diffuse injury III (swelling)	Cisterns compressed or absent with mid-line shift 0-5 mm; no high or mixed density lesion > 25 mm.
Diffuse injury IV (shift)	Mid-line shift >5 mm; no high or mixed density lesion > 25 cc.
Evacuated mass lesion	Any lesion surgically evacuated.
- Mass lesion	High or mixed density lesion > 25 cc; not surgically evacuated.

Management of intracranial hypertension

Medical management of increased intracranial pressure includes sedation, cerebrospinal fluid drainage, and osmotherapy either with mannitol or hypertonic saline. For intracranial hypertension refractory to initial medical management, barbiturate-induced coma, hypothermia, or decompressive craniectomy should be considered[2]. Hyperosmolar therapy by bolus or infusion. Hyperosmolar agents that are often used for severe head injuries are mannitol and hypertonic NaCl solution. Based on the recommendations of The Brain Trauma Foundation, the main therapy for intracranial hypertension is mannitol, but hypertonic NaCl is a potential alternative to be used.[10]

Mannitol

Mannitol can reduce intracranial pressure by two mechanisms. Mannitol rapidly lowers intracranial pressure by reducing blood viscosity and reducing blood vessel diameter. This occurs as a compensation for the autoregulatory function of cerebral blood flow (CBF). CBF levels are maintained through reflex vasoconstriction, resulting in a decrease in cerebral blood volume and a decrease in intracranial pressure. Administration of mannitol also reduces intracranial pressure via an osmotic mechanism, which occurs more slowly (15–30 minutes), associated with the gradual movement of water content from the parenchyma into the blood circulation.[2] The effect of mannitol lasts up to 6 hours and requires an intact blood-brain barrier.

Mannitol can accumulate in traumatized brain areas, where osmotic disturbances occur and fluid moves from the intravascular compartment to the brain parenchyma, possibly resulting in increased intracranial pressure. Various studies conducted in humans and animals have shown that mannitol administration has a beneficial effect on conditions of increased intracranial pressure, CPP, CBF, and brain metabolism[2]. Mannitol has the main effect on the brain, namely causing a rapid increase in plasma volume (hemodilution) so that blood viscosity decreases, CBF increases, microcirculation perfusion increases, and there is an increase in oxygen delivery to the brain.[11-14]

The effect of mannitol on reducing blood viscosity is achieved by lowering the hematocrit, the hematocrit drops drastically 10 minutes after administering mannitol, and remains low for the next 30 minutes. The increase in plasma volume will cause an increase in systemic blood volume, resulting in an increase in cardiac output and blood pressure, this is then followed by a strong diuretic effect that can cause hypovolemia. The osmotic effect of mannitol begins to appear at 10 to 30 minutes, at which time a gradient is formed between plasma and cells, the effect persists for a period that varies from 90 minutes to 6 hours or more depending on clinical conditions.

Mannitol can be used as a continuous infusion or as repeated boluses. Bolus administration is more effective than continuous infusion. The dose of mannitol required for an increase in CBF and a decrease in intracranial pressure is generally about 0.5–1g/kg body weight.[13] However, often less than the recommended dose is sufficient to cause improvement. In adult patients, 100ml of a solution containing 20g of 20g mannitol is generally sufficient to produce a therapeutic effect. The recommended dose for bolus administration of mannitol is 0.25–1g/kg body weight. Currently, mannitol is available in 5%, 10%, 15%, and 20% solutions. The most frequently used dosage forms are 15% and 20%. Mannitol is not metabolized primarily by glomerular filtration, little or no tubular reabsorption and secretion or even practically non-reabsorbed. [15,16]

Mannitol increases osmotic pressure in glomerular filtration and prevents tubular reabsorption of water and sodium. Thus, mannitol is the most commonly used of these drugs. By definition, osmotic diuretics are poorly absorbed orally, which means that they must be administered parenterally. Mannitol is excreted by glomerular filtration within 30-60 minutes after administration. The effect immediately felt by the client is an increase in the amount of urine. When administered orally, mannitol causes osmotic diarrhea. Because of this effect, mannitol can also be used to

enhance the binding effect of K^+ and resins or to remove toxic substances from the gastrointestinal tract associated with activated charcoal.[17-19]

Osmotic diuretics (mannitol) have major sites: the proximal tubule, loop of Henle and collecting duct. Osmotic diuresis is used to treat excess fluid in the tissues (intra-cells) of the brain. Osmotic diuretics that remain in the intravascular compartment are effective in reducing brain swelling. Mannitol results in a reduction of brain tissue through the withdrawal of fluid from brain cells which causes a temporary improvement in cerebral blood flow and oxygenation. Mannitol is a hyperosmolar solution that is used for therapy to increase serum osmolality. For this physiological reason, the mechanism of action of osmotic diuretics (mannitol) is to increase plasma osmolality and draw normal fluid from low osmolar brain cells to high osmolar intravascular fluid, to reduce brain oedema.[18-20]

The renal system works to limit water reabsorption, especially in the segments where the nephron is highly permeable to water, namely the proximal tubule and the descending loop of Henle. The presence of substances that cannot be reabsorbed by normal water by entering an osmotic pressure that opposes the balance. As a result, urine volume increases with the excretion of mannitol. An increase in the urine flow rate decreases the contact time between the fluid and the tubular epithelium thereby decreasing Na^+reabsorption. however, the natriureis that occurs is less significant than the diuresis of water, which may cause hypernatremia. Because osmotic diuretics increase water excretion rather than sodium excretion, they are not used to treat sodium retention. Mannitol has the effect of increasing the excretion of sodium, water, potassium and chloride, as well as other electrolytes. [21-25]

The mechanism of action of the present action of mannitol is as follows: Decreases blood viscosity by reducing hematocrit, which is important for reducing cerebrovascular resistance and increasing cerebral blood flow, which is followed by rapid vasoconstriction of arteriolar vessels and decreased cerebral blood volume. This effect occurs quickly (minutes). Mannitol has not been shown to work to reduce water content in injured brain tissue, it reduces water content in the uninjured part of the brain, which can leave more room for the injured part of the brain to swell (enlarge). Rapid intravenous bolus administration is more effective than slow infusion in reducing increased intracranial pressure. Too often high doses of mannitol can cause kidney failure. This is due to the effect of osmolality which immediately stimulates tubular activity in secreting urine and can reduce renal circulation. Administration of mannitol with furosemide will have a synergistic effect in reducing intracranial pressure. The best response will occur if mannitol is given 15 minutes before furosemide is given. This should be followed by maintenance of fluid volume and electrolyte status management during diuretic therapy.[26-28]

Toxicity

Expansion of extracellular fluid

Mannitol is rapidly distributed to the extracellular space and removes water from the intracellular space. Initially, this will cause expansion of the extracellular fluid and hyponatremia. This effect can cause complications of congestive heart failure and will cause pulmonary edema. Headache, nausea, and vomiting are found in patients receiving this diuretic. [20,21]

Dehydration and hypernatremia

Excessive use of mannitol without adequate water changes can lead to severe dehydration, water loss and hypernatremia. This complication can be avoided by paying attention to serum ion and fluid balance.

Elevated ICP returns after mannitol administration

Although these osmotics have long been considered to cause a reverse risk, the intracranial pressure returns to high. Or to be higher than the initial pressure of handling, this kind of phenomenon is now being questioned again. Some researchers believe that this risk should not occur if the drug is administered properly. For this reason the administration of mannitol must be careful, precise and monitoring or monitoring of the client's response is

correct and adequate.

2. Conclusion

Medical management of elevated intracranial pressure includes sedation, cerebrospinal fluid drainage, and osmotherapy with either mannitol or hypertonic salts.

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