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An article on increased intracranial pressure

MRS. TINTU C FRANCIS

Senior Lecturer, T John College of Nursing, Bangalore

ABSTRACT

Neuroscience nursing is a highly specialized field because neurologically impaired patients tend to be severely disabled and deserve to benefit from nursing profession. Increased Intracranial pressure (ICP) can result in irreversible damage to cranial contents by impairing blood flow and eventually cause death if left untreated. Astute nursing assessment and early aggressive resuscitation of patients with increased ICP prolong life and increases the potential for optimal recovery. Hence as the nurse place a vital role in management of patients with increased ICP, it is crucial that they should possess adequate knowledge to prevent the catastrophic consequences of increased ICP

Keywords : Increased intracranial pressure,cerebrospinalfluid,mean arterial pressure,ventricular circulation, herniation

INTRODUCTION

The skull is a hard, bony vault filled with brain tissue, blood, and cerebrospinal fluid (CSF). A balance between these three components maintains the pressure within the cranium. The modified Munro-Kellie hypothesis, a theory for understanding intracranial pressure (ICP), states that because the bony skull cannot expand, when one of the three components expands, the other two must compensate by decreasing in volume for the total brain volume and pressure to remain constant.

ICP is measured with a monitor in the ventricle, the brain parenchyma, or the subarachnoid space. Increased ICP seriously impairs cerebral perfusion and recognition of increased ICP is one of the most important assessments made by nurses caring for clients with neurologic disorders**.**

DEFINITION

Intracranial pressure is the pressure exerted in the cranium by its contents; the brain, blood and CSF. The normal ICP is 5to 15mmHg. Pressures greater than 20mmHg are considered to represent increased ICP

General information regarding cerebrospinal fluid(CSF)

Cerebrospinal fluid is normal a clear, colorless, odorless solution that fills the ventricles of the brain and the subarachnoid space of the brain and spinal cord. It is produced by active transport and diffusion, and thought to be formed from three different sources. The major source of CSF is the

secretions from the choroid plexus, a cauliflower like structure located in portions of the lateral, third, and fourth ventricles. The purpose of the CSF is to act as a shock absorber and to cushion the brain and spinal cord against injury caused by movement.s It is estimated that the amount of CSF daily produced by the choroid plexus is a about 500ml. A lesser proportion of CSF is secreted from the second source, the ependymal cells, which line the ventricles and blood vessels of the meninges. The last source of l production is the blood vessels of the brain and spinal cord. The amount produced from this source is very small

Most of the cerebrospinal fluid produced daily is reabsorbed into the arachnoid villi, which are projections from the subarachnoid space into the venous sinuses of the brain. CSF drains into the superiorsagittal sinus.

Flow of Cerebrospinal Fluid:

Cerebrospinal fluid circulation has been termed as the "third circulation." It is a closed system. Fluid formed in the two lateral ventricles passes into the third ventricle by way of the two foramina of Munro. The single cerebral aqueduct, aqueduct of sylvius connects third and fourth ventricle. CSF flows through the two lateral foramina of Luschka and midline through the one foramen of Magendie to the cisternal magnum. At this point the foramen of Magendie allows CSF to circulate around the cord; while the foramen of Lucschka directs it around the brain. Expanded areas of the subarachnoid space are called cisterns. Cerebral perfusion pressure is the amount of blood flow from the systemic circulation required to provide adequate oxygen and glucose for the brain. The formula for calculating CPP is as follows:

CPP = MAP-ICP, where MAP is mean arterial pressure(average pressure during cardiac cycle)

When MAP and ICP are equal, there is no CPP and brain perfusion ceases. Therefore it is crucial to maintain control of ICP and MAP

Conditions associated with aincreased Intracranial Pressure.

Three conditions associated with increased intracranial pressure are intracranial hypertension, cerebral edema and hydrocephalus

1. Intracranial Hypertension:

Normally intracranial pressure is less then 10 mmHg when the measurement is taken at the level of the formanen of munero in a patient who has assumed the supine position. A sustained intracranial pressure of 15mmHg or more is considered as abnormal and it is defined as intracranial hypertension.

Conditions that cause intracranial hypertension can be classified as follows.

1. Conditions that increase brain volume (space occupying masses eg:Hematomas , cerebral edema eg. head injury and rays syndrome)
2. Conditions that increase blood volume(obstruction of venous outflow,hyperemia , hypercapnia)
3. Conditions that increase cerebrospinal fluid volume(increased production of CSF, decreased absorption of CSF and obstruction to flow of CSF)

II Cerebral Edema:

Cerebral edema can be defined as an abnormal accumulation of water or fluid in the intracellular

space, extracellular space or both spaces that is associated with an increase in brain tissue volume. The edema can be local or generalized problem within the intracranial space. Three types of cerebral edema have been recognized; Vasogenic edema, Cytoxic edema and Interstitial edema..

1. Vasogenic Edema:

Vasogenic edema which predominantly effects the white matter is the most common type of cerebral edema seen. The pathophysiological defect is thought to be increased capillary permeability of the arterial walls to large molecules caused by a breakdown of the blood brain barrier. As a result, there is a leakage of a plasma like filtrate, including large molecules of protein into extracellular space.

2. Cytotoxic edema:

Cytotoxic edema is defined as an increase of fluid within the intracellular c space, chiefly the grey matter. The development of cytotoxic edema is associated with a hypoxic or anoxic episode such as acute hypoventilation or a cardiac arrest. The patientsbloodgases will slow a significant lowering of oxygen and elevation of carbon dioxide.

3. Interstitial Edema:

Interstitial edema involves the movement of cerebrospinal fluid across the ventricular walls so that there is an increase of sodium and water in the peri ventricular white matter. Interstitial edema is associated with obstructive hydrocephalus

III Hydrocephalus:

Hydrocephalus refers to progressive dilation of the cerebral ventricular system because the production of cebrospinal fluid exceeds the absorption rate.

Factors that Increase ICP:

Much of the knowledge about the relationship between certain activities and increased ICP have been gained from the data collected from continuous ICP monitoring. They are

1**. Hypercapnia and Hypoxemia:** causes cerebral vasodilatation, increased blood volume in the brain and subsequent increased intracranial pressure.

2. **Vasodilating Drugs:** Vasodilating drugs increase the blood flow to the brain, which can enhance an already increased level ICP. Example of such drugs are nicotinic acid and histamine.

3. **Valsalva Maneuver:** It is defined as exertion against epiglottis. The increased intra thoracic pressure that occurs during valsalva maneuver impedes the venous flow from brain, thereby increasing the ICP.

4. **Body positions and turning:**

Positions that obstruct venous return from the brain cause increase in ICP. The following positions like trendelenburg , angulation of neck and lateral position should beavoided because they are known to cause increased ICP.

5. **Isometric Muscle Contractions:**

An isometric muscle contraction results an muscle tension without lengthening the muscle. Such contractions raise blood pressure and further elevate ICP.

6. **Coughing and Sneezing:**

Both will elevate intra abdominal and intra thoracic pressure. The increased pressure from these areas transmitted through the spinal subarachnoid space to intracranial subarachnoid space, as well as through veins that communicate with dural sinuses and intracranial space. The venous return from the cranial vault impeded resulting in increased ICP.

7. **Emotional upset;**

Studies have shown emotional upsets can cause elevation in ICP.

8. **Noxious Stimulus** :

Common noxious stimuli the patient might experience include plugging of drainage tube , painful nursing or medical procedures and local noises or sudden jarring of bed.

Pathophysiology:

Basic to an understanding of the pathophysiology related to increased intracranial pressure is the modified monero –kellie hypothesis. This hypothesis can be simply stated as follows; The skull is a rigid compartment, filled with essentially non compressible content brain matter (80%), blood (10%) and cerebrospinal fluid (10%). The volume of these the component remains really constant. If any one component increases in volume, other component must decrease for the overall volume remain constant.

It can be explained in the following stages

1st stage ;As the intracranial mass enlarges initial compensation occurs through displacement of CSF into spinal cord The ability of the brain to adapt to increasing pressure without increasing ICP is called compliance. This is first and major compensatory mechanism

When the compliance of the brain exceeds, the ICP rises, clinical manifestations begin and other compensatory efforts to reduce pressure begin.

2nd stage; The second form of compensation is reduction of blood volume in the brain. When blood flow is reduced by 40%, cerebral tissue becomes acidotic.

When 60% of blood flow is lost, the electroencephalogram (EEG) begins to change This stage of compensation alters cerebral metabolism, eventually leading to brain tissue hypoxia and areas of brain tissue ischemia.

3rd stage; In this stage Intracranial pressure approaches arterial blood pressure. It is the beginning stage of decomposition and is also called as the pre terminal stage

4th stage; This is the last stage . The most lethal is the displacement of tissue across the tentorium ,under the falxcerberi or through the foramen of magnum into the spinal canal. This process is called herniation .

Death results from brain stem compression.

Herniation Syndromes:

Herniation syndromes have been classified into five types.

Supratentorial Herniation Syndromes

A.Transcalvarial Herniation:

Transcalvarial herniation occurs with open head injuries when brain tissue is extruded through an unstable skull fracture. Clinical manifestations vary greatly, depending on the location and extent of the open skull fracture.

B.CentralTranstentoralHerniaton:

Central transtentorial herniation is the result of the downward displacement of the diencephalons through the tentorial notch. It is caused by injuries or masses located in the cerebral cortex or on the outward perimeter of the cerebrum. An early indication of central transtentorial herniation is a rapid change in the level of consciousness. As the pressure increases, changes in respiratory patterns are seen: first, Cheyne-Stokes respirations and then central neurogenic hyperventilation; later, apneustic breathing and also ataxic breathing and, finally, Pupils become small but at first remain reactive, with progression to a dilated and fixed state. Pathologic reflexes begin with Babinski's sign and then progress from abnormal flexion to abnormal extensor posturing. Doll's eye reflex and a positive response to caloric testing are noted when brain stem function is still intact but are absent if the brain stem dies.

C.LateralTranstentorial Herniation:

Lateral transtentorial herniation occurs from displacement by masses in or along the temporal lobe. It is also called uncal herniation because as the temporal lobe is compressed, the uncus (the anteromedial portion of the hippocampus) or the hippocampal gyrus shifts from the middle fossa through the tentorial notch into the posterior fossa. As the herniation progresses, the pupils first become sluggish in response to light and then become unresponsive; lack of response is seen first in the ipsilaeral pupil and then in the contra lateral pupil, secondary to third cranial nerve compression at the midbrain level.

D.Cingulate Herniation:

Cingulated herniation occurs when the frontal lobes of the cerebrum are compressed, resulting in compression of the cingulated gyrus (an arch shaped convolution situated just above the corpus callosum) under the falxcerebri. Manifestations are related to cerebral artery compression resulting in ischemia and congestion, edema, and increasing ICP.

Infratentorial (Tonsillar) Herniation Syndrome:

Tonsillar herniation, also known as cerebellar herniation, occurs when the cerebellar tonsil shifts through the foramen magnum, compressing the medulla and upper portion of the spinal cord. Increasing pressure in the posterior fossa, often secondary to cerebellar bleeding, is the usual underlying problem. Manifestations often progress rapidly and include erratic changes in blood pressure, pulse rate, and breathing; decreased level of consciousness; an arched, stiff neck; and quadriparesis.

Signs and symptoms of increased ICP:

The brainstem signs of intracranial pressure are some time referred as classical signs of increased ICP. The specific classical signs and symptom of increased ICP are a rising systolic blood pressure, a widening of pulse pressure and bradycardia

The signs and symptoms can be classified into early findings and later findings.

EARLIER FINDINGS: Includes deterioration in LOC, papillary dysfunction, motor weakness such as monoparesis or hemiparesis and possible head ache

LATER FINDINGS: Includes continued deterioration in level of consciousness (coma) , possible vomiting , hemiparesis ,alternation in vital signs (increase in body tempertature) , respiratory irregularities and impaired brainstem reflexes

The presenting signs and symptoms of increased intracranial pressure observed in a patient will depend on the specific location of mass and the degree of intracranial compression

Measuring and Monitoring Increased ICP

Lumbar puncture: Technically ICP can be measured by means of lumbar puncture. In presence of suspected gross increase in intracranial pressure, lumberpuncture is contradicted because of the risk of brain stem herniation. There are other limits to use the Lumbar puncture as a means of determining ICP. For example a Lumbar puncture can give valid indication of ICP only if CSF is flowing freely within the subarchnoid space. Adhesions, constrictions or space occupying lesion can modify the flow of CFS thus resulting in a misleading measurement of ICP.

Three basic Techniques for ICP Monitoring are

1. Intraventricular catheter: It requires implantation through a burr hole. A polyethylene catheter into the anterior horn of the lateral ventricle of the nondominant hemisphere with the addition of a tranducer and a recording instrument, recordings of ICP are available for interpretation.
2. Subarachnoid Screw or Bolt: This is the most common intracranial pressure monitoring used today. A small tiwst drill hole is to made into subarchnoid space. The possibility of infection and herniation of brain are major concerns in this technique.
3. Epidural Monitor: This can be accompanied using various system designs .First design consists of a fibro optic sensor implanted into epidural space a transduser and recorder. Another monitoring design is the flow sensor and monitor. This system includes a sensor, pneumatic system, microprocessor and a display unit. The sensor is placed in the epidural space and acts as a pneumatic flow switch. The principle advantage of using Epidural Monitoring regardless of specific design is the brain or subarchnoid space is not penetrated and consequently there is less risk of infection.

Medical management and Treatment

The common protocols used by the physician to treat patient with increased intracranial pressure include.

1. Drug therapy (osmotic diuretics, steroids, and anticonvulsants)
2. Fluid restriction
3. Hyperventilation
4. Cerebrospinal fluid drainage
5. Barbiturate coma
6. Surgery

1. **Drug Therapy**

The general classifications of drugs that are commonly administered in the management of increased intra pressure are (1) osmotic diuretics (2) corticosteroids, and (3) anticonvulsants.

(A) **Osmotic Diuretics:** Osmotic diuretics are also referred as hyperosmolaric agents. The high osmotic concentration of the drug causes water to be drawn from the edemateous tissue. The most commonly used Osmotic Diuretics is mannitol.

(a) **Mannitol ; (Osmitrol).**

Mannitol's osmotic effect causes intravascular fluid to be drawn from the cells to the extracellular fluid and from redblood cells to the plasma.

Major use of Osmitrol is to greatly reduce increased intracranial pressure prior to and during neurosurgery and in the treatment of cerebral edema. Osmitrol is indicated when there is evidence of developing brain stem compression or herniation. A rebound increase in intracranial pressure may occur 12 hours after osmotic diuretics have been administered, but the occurrence of this phenomenon is less frequent with Osmitrol than with urea. Average dose of Osmitrol is 1.5 gm to 2 gm /Kg of weight. It is administered rapidly in a 15%, 20%, or solution on (in 500ml) over a period of 30 to 60 minutes

(b) **Glycerol:** Glycerol is a trivalent alcohol that acts as an osmotic diuretic . Its effect on the brain is similar to that of mannitol. One advantage of glycerol is that it can be taken intravenously and achieve maximum decrease in intracranial pressure within 30 to 60 minutes.

(c) **Glucose and Urea:** Although both glucose and urea are classified as osmotic diuretics, their use in the clinical area is minimal because of side-effects and rebound swelling..

(d) **Loop Diuretics:** The drug is effective in reducing cerebral edema and lowering intracranial pressure and is especially suited to managing children who respond to cerebral trauma with hyperemia.

(B) **Corticosteroids**: The use of corticosteroids in the neurological – neurosurgical patient is very controversial. It is generally accepted that the use of steroids in the management of vasogenic edema associated with brain tumors is very effective. The drug of choice is usually dexamethasone (Decadron). For those who support the use of high-does dexamethasone, the following protocol is typical: after a loading dose of 100 mg given intravenously, give 100 mg every 6 hours intravenously or orally for 8 days, then taper the dose.The drug is tapered over the next 7 to 10 days. If a low dose protocol is selected ;after giving a 12 mg loading dose intravenously ,give 4mg every 6hours for 8 days intravenously or orally, then taper the drug as with a high dose protocol

(C) **Anticonvulsants.** The drug of choice for prevention of seizure activity in the patient with increased intracranial pressure is phenytoin (Dialantin). The average dosage of Dilantin is 100 mg orally, three or four times daily.

(2) **Fluid Restriction:** The underlying principle of fluid restriction is to cause slight dehydration. The Physician may choose to limit the patient's fluid intake to a specific amount for the given period (usually written in millimeters [ml] of fluid for a 24-hour period). Fluid intake can range from 900 ml/24 hours to 2500 ml/24 hours.

(3) **Hyperventilation**

Controlled hyperventilation is an important part of the approach for reducing increased intracranial pressure. Controlled hyperventilation can be achieved for the patient on a ventilator by adjusting the controls on the ventilator. For the patient who is not on a ventilator, hypertension can be carried out with the use of an Ambu bag.

(4) **Cerebrospinal Fluid Drainage**

Physician will insert drainage catheters into a cerebral ventricle so that excess cerebrospinal fluid can be removed; this controls erratic increases in intracranial pressure. Ventricular drainage is a temporary treatment used in conjunction with the other modalities of care.

(5) **Temperature Control**

Hypothermia ($<35^0$C) has been used during intracranial surgery and in the treatment of severe craniocerebral trauma. The underlying principle for the use of hypothermia is as the body temperature drops, there is a decrease of all metabolic processes including that of brain.

(6) **Barbiturate Coma Therapy**

Barbiturate coma therapy is a treatment protocol developed for the management of uncontrolled intracranial hypertension that has not responded to conventional treatment such as surgical decompression, osmotic diuretics, fluid restriction, steroids, hyperventilation, and cerebrospinal fluid drainage. Barbiturate therapy, introduced by Shapiro and associates, consists of administering large doses of short acting barbiturates to induce and maintain coma. The drug most often used is pentobarbital(Nembutal) .Barbiturate coma is induced by administering a loading dose of pentobarbital, 3 mg to 5 mg/kg of body weight, by slow intravenous push. It is expected that the ICP will decrease by 10mm Hg within 10-15 minutes. A second loading dose should be administered in 2 hours if the expected drop in ICP has not occurred. The major problems associated with barbiturate coma therapy are hypotension, cardiac depression and erractic dose response.

(6) **Surgery**

If there is a localized hematoma or mass such as a tumor or abscess, surgical removal is indicated.

NURSING MANAGEMENT OF PATIENTS WITH INCREASED ICP

(1) NEUROLOGICAL ASSESSMENT

Frequent assessment of neurological signs (including vital signs) must be made to ascertain any changes in the neurological status of the patient. This means that at frequent intervals (every 15 minutes to every 4 hours), depending on the circumstances, a neurological assessment of the patient is carried out, including

Level of consciousness

Pupillary size and direct light reaction

Eye movement

Motor function (and sensory function)

Vital signs

(2) POSITION THE PATIENT

Place the patient supine with head elevated30 degrees unless contraindicated keep the head in the neutral position to facilitate venous drainage from the brain. Avoid extreme rotation and flexion of the neck as these positions compress the jugular veins and increase the ICP. As the coma lightens the patient become disoriented making it difficult to maintain proper positioning.

(3) MAINTAING PATENT AIR WAY

Patients need to main to maintain a patent airway even in the presence of an increased ICP. Suctioning assists in preventing build up of secretions co_2 and resultant elevation of ICP. Adequately oxygenate intubated patients before intiatingsuctioning , between suctioning efforts and after suctioning. Try to limit suctioning two three passes, and limit each pass to 10 seconds. Nasal drainage may indicate a dural tear, therefore suctioning of nares is contraindicated in patients with increased ICP.

CONCLUSION

Intracranial pressure is the pressure exerted by the cerebrospinal fluid within the ventricles of the brain. The intracranial pressure is a centrally fluctuating phenomenon that responds to such factors as arterial pulsation and respiratory cycle activities such as coughing and sneezing and straining at stool result an intracranial pressure, while activities such as standing up or assuming an erect position will lead to a decrease in intracranial pressure. Raised intra cranial pressure is the final common pathway for many intracranial problems. Nurses caring for patients with increased ICP should have a thorough knowledge regarding increased ICP to prevent the irreversible damage to the brain

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