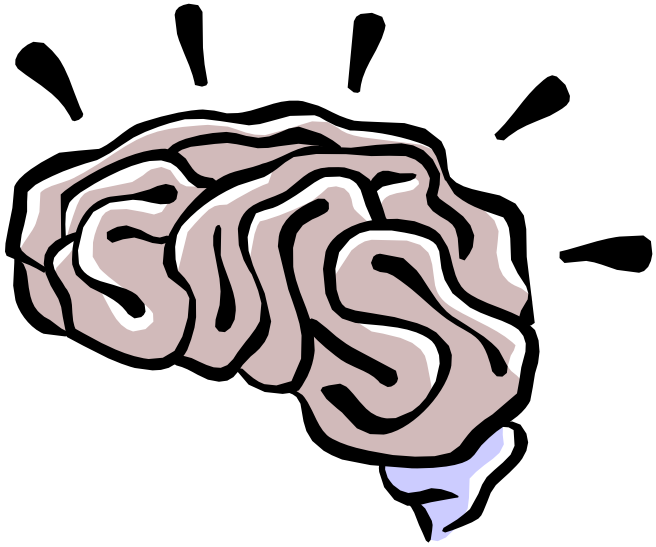
John Hunter Hospital



Neurological Self - Directed Learning Package

Revised by James Wilson CNE

AIM OF THE PACKAGE

To provide the registered nurse with a beginning level of knowledge on which to base safe nursing practice when caring for patients who have suffered a neurological insult.

OBJECTIVES OF THE PACKAGE

On completion of this package the registered nurse will be able to:

1 identify the relevant gross anatomy of the brain;

2 discuss the pathophysiology of brain injury;

3 outline the importance of the prevention or minimisation of secondary brain injury through nursing care;

4. employ the Monroe Kellie hypothesis as a means of explaining the pathophysiology of raised intracranial pressure;

5 provide a rational for the nursing and medical management of a patient with the potential for further brain injury as a result of raised intracranial pressure.

6. outline the nursing principles included in the “Head injury Clinical Practice Guidelines” John Hunter Intensive Care Unit.

7. perform clinical assessment of a patient with a neurological impairment

8. understand principles of management of external ventricular drains

WHAT TO DO WITH THE PACKAGE

The package has been divided into sections. Each section relates to other sections. There is a combination of directed learning either in the form of readings (learning tasks) from references and activities which relate to the reading or clinical practice.

The references will provide a starting point for your learning- the list is by no means exhaustive and if you wish to do your own research or use your own references, please do so.

Many of the staff in the ICU will be able to provide additional assistance to complete the activities if you experience any difficulties.

Answering the questions.

1. You should attempt all activities in the package.
2. While you will decide the amount of detail to be included in your answers, your work should give the marker the impression that you have a good understanding of the underlying principles and concepts covered in each question. It is expected that the amount of detail will vary from person to person.

The package has been divided into several sections

Section 1 Neuro Anatomy
Section 2 Cranial Nerves
Section 3 Neurological Assessment & Pharmacology
Section 4 Principles of raised intracranial pressure
Section 5 Subarachnoid Haemorrhage Cerebral vasospasm
Section 6 Intracranial Pressure Monitoring Devices
Section 7 External Ventricular Drains

NEURO ANATOMY

Learning Task

Review the anatomy of the cranial vault,

Activity 1

a) *Name the bones of the skull and cranial vault.*

b) *Describe and discuss why the following structures are important for the protection of brain function?*

1) Skull 2) meninges 3) Cerebrospinal fluid.

References

McCance & Heuther (2002) p. 363-400
Hickey (2003) p. 45-92

Clinical Implications

A fracture of the base of skull is considered a serious injury because of the closeness of the fracture to the brain stem. A base of skull fracture can also result in the tear of the dura mater leading to communication between the brain and air. CSF may also leak through the nose or ears in a base of skull fracture. Most importantly if the cribiform plate is damaged. Anything passed through the nose, such as naso-gastric tubes or suction catheters, may go directly into the brain. For this reason ***nothing should be passed through the nose of a patient with a suspected base of skull fracture.***

Learning Task

Review the blood supply to the Brain

Activity 2

Name the vessels and draw the Circle of Willis.

References

McCance & Heuther (2002) p. 363-400
Hickey (2003) p. 45-92

In favourable instances where the Circle of Willis is anatomically correct, a constant, adequate blood supply is permitted to reach all parts of the brain even after one or more of the four supplying vessels has been ligated. The four supplying blood vessels are comprised of two internal carotid arteries (anterior supply) and two vertebral arteries (posterior supply).

The table below (taken from McCance & Heuther (2002) p. 383 outlines the clinical alterations caused by occlusion of the major cerebral arteries

Arterial Origin	**Structure Served**	**Effects of occlusion**
Anterior Cerebral Artery	Basal ganglia, corpus callosum, medial surface of cerebral hemispheres, superior surface of frontal & parietal lobes	Hemiplegia on contralateral side of body greater in lower than upper limbs
Middle cerebral Artery	Frontal lobes, parietal lobes, temporal lobes (primarily cortical surfaces)	Aphasia in dominant hemisphere and contralateral hemiplegia
Posterior Cerebral artery	Parts of diencephalon and temporal lobe, occipital lobe	Visual loss sensory loss contralateral hemiplegia if cerebral peduncle affected

PART 2 CRANIAL NERVES

Learning Task

Review the Cranial Nerves

Activity

Read the information provided in the "components" and "function" columns of the following table and identify the cranial nerve that best fits the description. In the 4th column list the nursing implications of dysfunction of each nerve.

References

McCance & Heuther (2002) p. 363-400
Hickey (2003) p. 45-92

NERVE	COMPONENTS	FUNCTION	IMPLICATIONS
	Contain both motor and sensory fibers.	Motor portion: innervates the superior oblique muscle of the eyeball. Sensory portion: responsible for muscle sense (proprioception) for the above muscles.	
	Contains sensory fibers only.	Conveys impulses associated with hearing, and equilibrium.	
	Contains both motor and sensory fibers.	Motor portion: innervates the muscle of the tongue, and conduct impulses related to speech and swallowing. Sensory portion: conducts impulses for muscle sense (proprioception) for the above muscles.	
	Contains sensory fibers only	Conveys impulses related to smell.	
	Contains both motor and sensory fibers.	Motor portion: the superior branch innervates the superior rectus muscle of the eyeball and the levator palpebrae muscle of the eyelid: the inferior branch innervates medial rectus, inferior rectus, and inferior oblique muscles of the eyeball and controls the ciliary muscle of the eye and the sphincter muscle of the iris. Sensory portion: conducts impulses for muscle sense (proprioception)for the above muscles.	
	Contains both motor and sensory fibers.	Motor portion: innervates the muscles of the pharynx, larynx, oesophagus, stomach, small intestine, large intestine and gall bladder. Sensory portion: conveys impulses for sensation from the larynx and viscera.	

NERVE	COMPONENTS	FUNCTION	IMPLICATIONS
	Contains both motor and sensory fibers.	Motor portion: innervate the muscle of mastication. Sensory portion: conveys impulses related to touch, pain and temperature from the skin over the eyelids, the eyeball, lacrimal glands, nasal cavity, the side of the nose, forehead, anterior scalp, the mucosa of the nose, palate, pharynx, teeth, upper lip, check, two thirds of the tongue, and skin over the mandible, side of head and floor of the mouth.	
	Contains sensory fibers only.	Conveys impulses initiated by the rods and cones of the retina.	
	Contains both motor and sensory fibers.	Motor portion: innervates the voluntary muscle of the pharynx, larynx and soft palate as well as conveying impulses to the sternocleidomastoid and trapezium muscles. Sensory portion: conducts impulses for muscle sense (proprioception) for the above muscles.	
	Contains both motor and sensory fibers.	Motor portion: innervates the lateral rectus muscle of the eyeball. Sensory portion: conducts impulses for muscle sense (proprioception) for above muscle.	
	Contains both motor and sensory fibers.	Motor portion: innervate the swallowing muscle of the pharynx and the parotid gland. Sensory portion: conduct impulses from the pharynx and taste buds of the posterior third of the tongue, and the carotid sinus: also conducts impulses for muscle sense (proprioception) for the lateral rectus muscle.	
	Contains both motor and sensory fibers.	Motor portion: innervate the facial and scalp muscles, the sublingual and submandibular glands. Sensory portion: conduct impulses from the taste buds of the anterior two thirds of the tongue: also conducts impulses for muscle sense (proprioception) for the face and scalp.	

PART 3 NEURO ASSESSMENT & PHARMACOLOGY

Learning Task

Obtain a neuroscience observation chart. Review the correct method of eliciting the Glascow Coma Score responses.

Reference

Hickey (2003) pp. 159-184

Activity

Outline the differences in neurological assessment of adults and children

In most situations Intensive Care patients receive some form of medications which alter their level of consciousness.

Learning Task

Review the effects of the following common ICU medications;

midazolam	morphine	mannitol,
dilantin	sodium pentothal	magnesium sulphate
fentanyl	vecuronuim	suxamethonium
hypertonic saline (23%)	nimodipine	

Activity

Complete the following medication reviews of the same drugs in the following format;

- Mode of action
- Indications
- Dosage
- Mode of administration
- Adverse effects
- Nursing considerations when administering the drug

SEDATION SCORING & ADMINISTRATION

Learning Task

Obtain a copy of the Clinical Practice Guideline -Sedation & Analgesia and the Sedation Score in the help library

Activity

Review the chart of any patient in the unit- did they have a sedation score recorded- if not did the patient fit the criteria for not being sedation scored? If the patient was sedation scored is there any evidence that it changed patient care?

SECTION 4 PRINCIPLES OF RAISED INTRACRANIAL PRESSURE

Learning Task

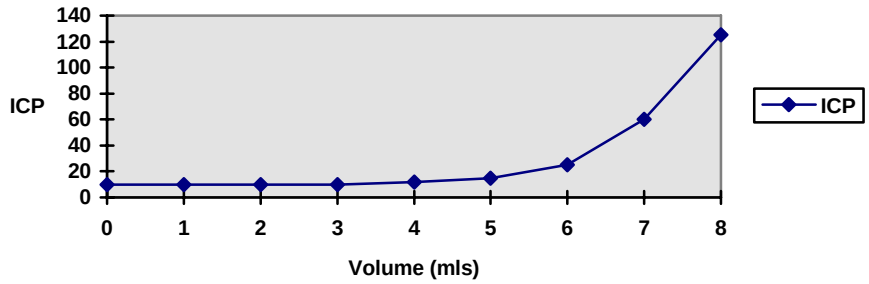
Read
Clinical Practice Guidelines for Intensive Care Management of Head Injuries John Hunter Hospital Intensive Care Unit HELP Library
Oh (2003) pp. 689-710.
Hickey (2003) pp. 285-318
Urden et al (2002) pp. 701-710.

The primary goals in the management of the patient with increased intracranial pressure in the intensive care unit are to prevent secondary neurological injury and limit possible complications that could occur in other organs systems (T.E. Oh 2003). Secondary injury refers to events or complications that contribute to further brain injury after the initial primary event. Events that may cause secondary brain injury include systemic hypotension, hypoxaemia, hypercapnia, sustained increased intracranial pressure and sustained cerebral oedema. In order to understand the means of preventing secondary injury it is necessary to have a comprehensive understanding of what causes raised intracranial pressure. Raised intracranial pressure and the basis for preventing secondary injury are based on the Monroe Kellie doctrine.

Monroe Kellie Doctrine:

The skull is a rigid non-expandable cavity that contains 10% blood, 80% brain tissue and 10% cerebrospinal fluid (CSF). The pressure exerted by these components within the skull is the ***intracranial pressure (ICP)***. With a change in the volume of any of these components there must be a compensatory decrease in the volume of another compartment to maintain a normal ICP. The mechanism by which this reduction in volume occurs is called ***compensation***. Increased volume can be compensated for through a decrease in the volume of intracerebral blood flow, a decrease in the volume of the CSF, stretching of the dural membrane, or remoulding of the brain tissue. Although blood volume may be decreased through the narrowing of the cerebral vasculature, most compensation is due to the migration of the CSF into the relatively distensible spinal subarachnoid space via the subarachnoid cisterns. As individual compartments reach mechanical limits however, volume shifts fail to compensate for pathological processes. As these compensatory mechanisms are exhausted, small incremental volume changes cause large increases in pressure. The relationship between volume and pressure is best illustrated through the volume pressure curve (see diagram below).

Figure Volume Pressure curve



As intracranial volume increases, intracranial pressure (ICP) initially remains quite stable due to an effective compensation mechanism. However as compensation mechanisms become exhausted further increases in intracranial volume will result in an exponential increase in ICP. The initial phase of compensation (the horizontal part of the curve) is accounted for by the displacement of a volume of CSF, equal to the volume introduced. If for example, 1 ml of blood was introduced into the cranium we would expect 1 ml of CSF to be displaced in order to compensate for the increased volume. The vertical portion of the curve - the phase of ***decompensation*** - represents the relatively incompressible brain tissue.

Further Reading:

Mayer and Chong (2002) pp.55-67

OH (2003) pp. 505-514

Hickey (2003) pp. 285-318

Elastance and Compliance:

The terms elastance and compliance indicate the degree of compensation. The ability of the brain to compensate for an increase in volume is known as elastance. ***Elastance*** refers to the relative stiffness of the intracranial components in response to increased volume, and is dependant on the physical condition of the brain tissue. Elastance is increased by arterial hypertension and decreased by osmotic diuretics. With adequate elastance, small increases in volume result in minimal changes in ICP. However, when elastance is high, a dramatic increase in ICP occurs with even small increases in volume (Hudak & Gallo p.676).

Compliance is the reciprocal of elastance and refers to the slackness in response of intracranial components to increased volume. When compliance is adequate, little or no increase in ICP occurs in response to small increases in volume. However, when compliance is low, ICP increases dramatically with small increases in volume. Therefore, the results of high elastance and low compliance are similar. As elastance increases, compliance decreases and compensatory mechanisms fail. thus small increases in volume can lead to large increases in ICP (Hudak & Gallo p.676).

Clinically compliance can be assessed through the addition of small incremental fluid volumes (approx. 1 ml) into a ventricular catheter. As fluid volumes are added the ICP is

observed. An increase of 1-2 mmHg indicates an adequate compliance whereas a rise greater than 2 -5 mmHg indicates a low compliance.

Clinical Significance:
An understanding of compliance is important because it relates to a patients ability to compensate for increased intracranial volumes. Although two patients may have the same ICP a small increase in volume in one patient may not affect ICP but may cause a dramatic rise in ICP for another. Whilst we never insert a ml of saline into the head to measure compliance we clinically can observe a patient with decreased compliance by the way they respond to our nursing cares. For instance if two patients both have ICP's of 15mmHg and then we suction both patients and both patients ICP's rise to 25mmhg, this is a normal response. If however one patients ICP falls back to 15mmHg within 3 minutes whilst the second patients ICP takes 15 minutes to return to 15mmHg then the second patient has decreased compliance and should alert us to the need to modify our nursing procedures with this second patient.

Activity 5

Indicate on the "Volume Pressure Curve" figure where compensation and decompensation are occurring

ICP can be reduced by decreasing any of the components that contribute to the volume of the cranium, ie CSF, blood volume or brain tissue.

Cerebrospinal Fluid (CSF)

Intracranial pressure can be effected by the balance between the rate of secretion and absorption of CSF. Although CSF only accounts for 10% of the total intracranial volume, CSF translocation is the major means of buffering for expanding intracranial masses. CSF formation is primarily the function of the choroid plexus located in the lateral, third and fourth ventricles. The rate and direction of CSF flow is dependent on the structure of the subarachnoid space, pressure gradients secondary to arterial and respiratory pulsations and sudden changes in position. The rate of CSF formation is constant, the rate of CSF absorption however increases with an elevated intracranial pressure. Reabsorption is dependent on the hydrostatic pressure difference between the subarachnoid space and dural sinuses. Any factor that interferes with the absorption of CSF will alter ICP. Changes in the volume of the intracranial content can lead to an increased pressure depending on the compliance of the craniospinal compartment (Hudak & Gallo p.676).

Clinical Significance*:*

CSF functions as a means to buffer the brain from injury during trauma. In incidences of raised ICP, translocation of CSF is the major means of compensation.

The development of mass lesions, such as blood in the subarachnoid space, may impede the absorption of CSF, causing hydrocephalus and an increase in ICP. This is why most Subarachnoid haemorrhage patients require a ventricular drain at least in the short term and may require a permanent VP shunt.

Read
"Composition of CSF" McCance & Heuther (2002) p. 380, or any other anatomy & physiology book.

Activity 6

Label a diagram, outlining the formation and absorption of CSF

AND

Describe the flow of CSF

CEREBRAL BLOOD FLOW (CBF):

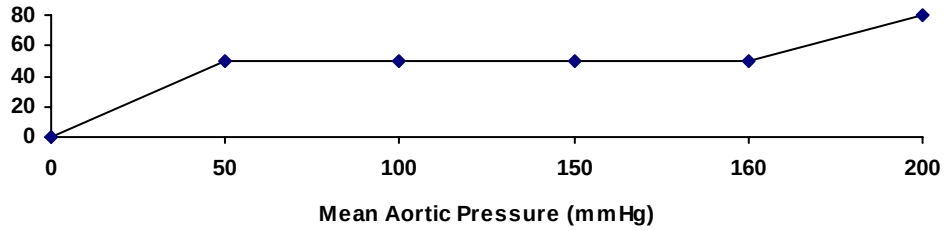
Read “Alterations in Cerebral Haemodynamics”
McCance & Heuther p. 466

A generalised increase in ICP will have a significant effect on cerebral blood flow throughout the brain. The true driving force of CBF is the perfusion pressure, or the difference between CBF and CVP, which in the intracranial space is essentially the same as ICP. Hence cerebral perfusion pressure may be calculated using the following equation:

CPP = MAP - ICP

Oxygen delivery to the central nervous system (CNS) is the product of CBF and arterial oxygen content. Due to the high metabolic rate of the brain and the low reserves of oxygen and substrates, the CNS relies on a generous, well oxygenated blood supply to support normal function. The amount of blood supplied to the brain is approximately 17-20% of the bodies resting cardiac output. In order to maintain an adequate cerebral blood flow the cerebro-vasculature must dilate and constrict in response to changes in blood pressure. Under normal circumstances cerebral blood flow remains relatively constant despite a wide range of mean arterial pressures (see figure).

Figure 6. CPP accomodation with varying MAPs



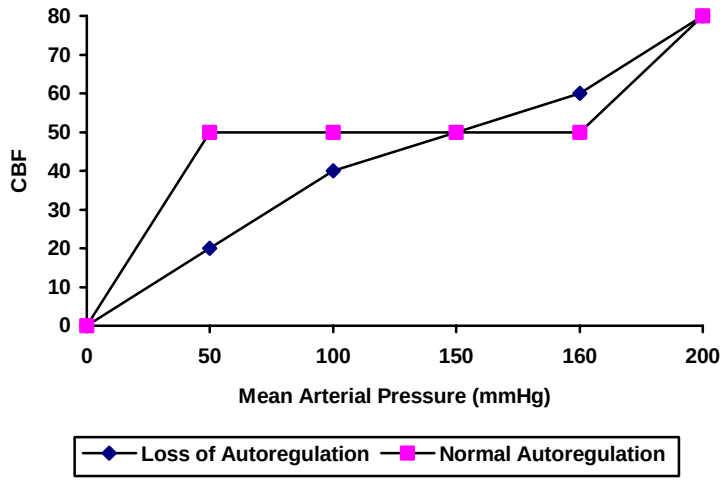
Alterations in mean aortic blood pressure between 50 and 160 mmHg will generally only cause a 1- 2 ml/100gm/min change in cerebral blood flow. The mechanism by which cerebral blood flow is maintained is known as ***autoregulation***. Autoregulation refers to the ability of the cerebral blood vessels to constrict and dilate, thus altering resistance, in order to maintain a constant blood flow despite a wide range of mean aortic pressures. As mean aortic pressure decreases the cerebral vasculature dilates, thus lowering resistance to flow and maintaining CBF. Conversely as blood pressure increases the cerebral arteries constrict, increasing resistance to flow. CBF is determined by the cerebral perfusion pressure of the CNS and the resistance of the cerebral vessels to flow:

$$CBF = \frac{(MAP - ICP)}{R}$$

where MAP is mean aortic pressure, ICP is intracranial pressure and R is cerebral vascular resistance. Under normal circumstances ICP is low (typically < 10 mmHg) and MAP varies over a considerable range. Autoregulation of the vasculature circuit, by varying R, achieves nearly constant CBF, as described above. Under pathologic conditions, ICP may rise dramatically to a level that impairs CBF. Brain injury often affects vessel reactivity and autoregulation may be impaired (see following diagram). The degree of loss of

autoregulation is directly proportional to the severity of the head injury. When autoregulation is impaired, CBF becomes passively dependent on changes on blood pressure. Autoregulatory mechanisms become nonfunctional with a sustained rise in ICP, focal or diffuse cerebral injury, loss of blood brain barrier or a mean arterial pressure of less than 50 mmHg or exceeding 160 mmHg. The combination of dysfunctional autoregulation and increased ICP further increases the patients risk for cerebral ischaemia .

Figure 7 Relationship between CBF and autoregulation



Activity

What is the normal CPP and how can it be measured?

Cerebral Metabolism

CBF is closely linked to the regional cerebral metabolic rate for oxygen ($CMRO_2$). Increased neuronal metabolic activity usually accompanies increased neurotransmission. Increased neurotransmission is associated with increased frequency of action potentials with release of potassium from the nerve cell and consequent increases in extracellular potassium. Elevation of extracellular potassium results in dilation of cerebral vessels causing increased blood flow.

Temperature

Temperature exerts a potent effect upon regional cerebral metabolic rate for O_2 ($CMRO_2$). A decrease in temperature by 1^0C will decrease the oxygen requirements by approximately 10%. This mechanism has been proposed to explain functional preservation of the brain after cold water near drowning. Above normal body temperature $CMRO_2$ rises sharply until approximately 42 or 43 ^{o} C when the metabolic rate ceases to climb and may actually fall.

Activity

*What effect will an elevated temperature have on a patient with an elevated ICP? Explain***.**

ICP and venous return

Due to the absence of valves in the jugular veins, the mechanism for blood flow out of the head remains passive and gravity dependant. This being the case any increase in venous resistance such as raised intrathoracic pressures or obstructed neck veins will decrease the amount of venous drainage from the head. The lack of venous drainage results in increased venous blood volume and therefore increased ICP.

Activity

How is head positioned if it is said to be in a neutral head position?

Describe the rationale for maintaining a neutral head position for a patient with an elevated ICP.

Identify any nursing actions which may be undertaken in attempts to reduce adverse effects of venous congestion

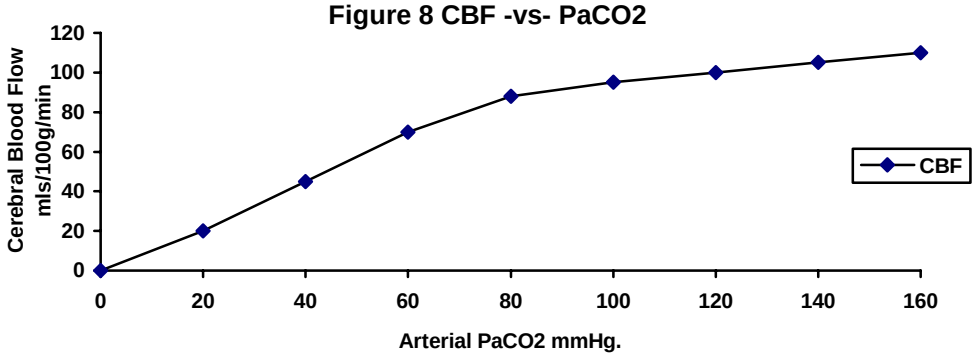
Activity

Describe the rationale for careful consideration of the level of Positive End Expiratory Pressure (PEEP) on patients with raised ICP.

Cerebral Blood Flow and $PaCo_2$

The level of carbon dioxide has an important effect on both CBF (see following diagram) and ICP. CO_2 is a potent vasodilator, causing increased CBF during hypercarbia and decreased CBF during hypocarbia. CBF changes by 2 to 3 percent for each change of 1 mmHg in $PaCO_2$ from the normal level of 40 mmHg. An increase in blood volume will have little effect on ICP if the pressure is low. If the ICP is already elevated however an increase in CBF can bring about a major rise in pressure. Conversely hypocarbia can constrict arterioles and reduce cerebral blood volume.

Figure 8 CBF -vs- PaCO2



Activity

What is the rationale for preventing hypercarbia in a patient who has a head injury?

Activity

How could alteration in minute volume influence ICP?

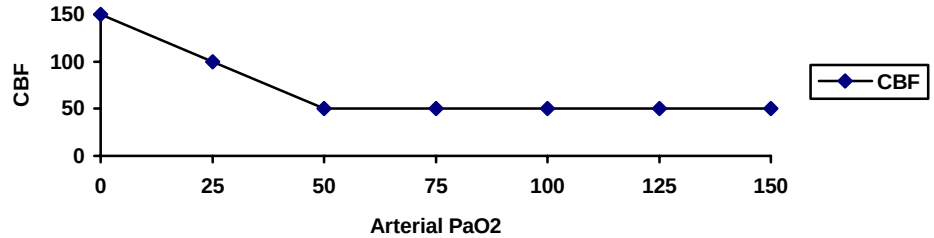
Activity

Describe the potential complications of hyperventilating a patient to a CO_2 of < 25 mmHg.

Cerebral Blood Flow and PaO_2:

During periods of decreased arterial oxygen content cerebral blood flow will rise in an attempt to maintain oxygen delivery to the brain. Mild to moderate degrees of hypoxia do not significantly alter CBF. At normal values of $PaCO_2$ and blood pressure, PaO_2 values below 60 mmHg are associated with relatively large increases in CBF and hence cerebral blood volume (see following diagram)

Figure 9 CBF -vs- Arterial PaO2 mmHg



Activity

What would be the result if arterial oxygenation fell below normal levels in a patient with raised ICP?

Cerebral Oedema

Cerebral oedema is an abnormal accumulation of water or fluid, either local or generalised, in the intracellular space, the extracellular space or both and is associated with an ***increase in brain tissue volume***. Cerebral oedema may cause deterioration in brain function through the compression of brain tissue and blood vessels. the compression of blood vessels may cause a deterioration in brain function by reducing oxygen delivery to the brain. Cerebral oedema may also block the flow of CSF, contributing to a rise in ICP. Cerebral oedema often reaches its maximum 48-72 hours post injury. The three main types of cerebral oedema are vasogenic, cytotoxic and interstitial.

Vasogenic oedema is the most common type of oedema encountered. Vasogenic oedema is an increase in the brain water content that occurs as the result of disruption of the blood brain barrier. In vasogenic oedema there is extravasation of electrolytes, proteins and even blood, which in turn pulls water out of the intravascular space and into brain tissue. The development of vasogenic oedema is influenced by systemic blood pressure. An increase in blood pressure will increase vasogenic oedema whereas hypotension will retard its development. Once vasogenic oedema is established the volume of brain tissue increases and there is a rise in ICP. An elevated ICP will decrease CBF and CPP. The reduction of oxygenated blood causes an increase CO_2 and lactic acid, which in turn may cause an impairment in autoregulation. The impairment of autoregulation may lead to a cycle of events that further increases vasogenic oedema, which if left untreated may cause a further increase in the volume of brain tissue, leading to herniation and death. Conditions producing vasogenic oedema include tumours, abscesses. meningitis, trauma, prolonged ischaemia and contusions (Hickey 2003 pp 290).

Learning Task

Review "Cycle of malignant progressive cerebral oedema,"Hickey (2003 p.290)

Cytotoxic oedema is an intracellular accumulation of fluid, supposedly caused by the inhibition of the sodium potassium pump. If the sodium potassium pump is not functioning correctly sodium may accumulate in the brain tissue. An increase in sodium in the extravascular space will draw fluid into the brain through osmosis. Cytotoxic oedema is often associated with an increase in lactic acid which contributes to a rapid deterioration of cellular function. Cytotoxic oedema is caused by toxic or metabolic insults to the brain, such as prolonged episodes of hypoxia.

Interstitial oedema *is* the net movement of CSF from the ventricles into the interstitial spaces. The movement of CSF into the brain tissue is caused by an increase in ventricular pressure which typically arises through a decreased absorption of CSF. During periods of raised ICP CSF flow is most likely to become obstructed at the narrow passageway known as the aqueduct of sylvius. As CSF production continues CSF pressure rises forcing CSF into brain tissue.

Activity

Describe how mannitol and frusemide decrease ICP.

Brain Herniation:
Increased intracranial pressure caused by cerebral oedema or a space occupying lesion will lead to herniation of the brain tissue if left untreated. Brain herniation can be simply defined as the protrusion of brain tissue outside of its normal compartment. Three areas within the skull predispose the brain tissue to herniation in the presence of increased intracranial pressure. These are the ***falx cerebri***, the ***tentorium cerebelli*** and the ***foramen magnum.*** The falx cerebri and tentorium are structures that describe divisions within the cranium. The foramen magnum is a hole in the base of the skull.

Cingulate Herniation
The cingulate gyrus is a deep medial area of the cerebral hemisphere that lies next to the falx cerebri. Cingulate herniation occurs when the expanding frontal portion of the cerebral hemisphere shifts laterally, forcing the cingulate gyrus under the flax cerebri. The major danger of cingulate herniation involves the compression of blood vessels, primarily the anterior ipsilateral and anterior cerebral artery. Compression of these arteries causes; ischaemia, congestion, oedema and necrosis. Cingulate herniation may cause a cycle that results in severe rises in ICP and damage to the entire brain

Central Transtentorial Herniation
Central herniation is downward displacement of the cerebral hemispheres, basal ganglia diencephalon, and midbrain through the tentorial notch. This downward movement occurs in response to increased pressure in the supratentorial region. the signs and symptoms of central herniation, in addition to an impaired level of consciousness, include disturbed eye movements with a loss of upward movement and bilateral decorticate or decerebrate posturing.

Lateral Transtentorial (Uncal) Herniation
Masses that are more laterally located will produce lateral transtentorial or uncal herniation. Compression of the third cranial nerve results in loss of its occulmotor and pupillomotor functions but is manifest clinically primarily as dilation of the ipsilateral pupil.

Tonsillar Herniation
The herniation of the cerebellar tonsils through the foramen magnum compressing the medulla and upper cervical cord is referred to as tonsillar herniation, can occur with mass lesions in the cerebellum and supratentorial mass lesions. Signs of tonsillar herniation include precipitous changes in blood pressure, heart rate, small pupils, disturbances in conjugate gaze, ataxic breathing (completely irregular breathing) and quadriparesis. This syndrome is most frequently observed in patients with cerebella haemorrhage often develops very rapidly and if left untreated will result in death.

Learning Activity
Review the types of herniation in Urdan (2002) p.711 and Hickey (2003)pp.291-296.

Signs of impending herniation
- decreased level of consciousness
- Pupillary abnormalities
- motor dysfunction (hemiplegia, decorticate or decerebrate posturing)
- impaired brain stem reflexes (corneal, gag)
- Alterations in vital signs including respiratory irregularities.

SECTION 5 SUBARACHNOID HAEMORRHAGE /CEREBRAL VASOSPASM

Subarachnoid haemorrhage (SAH) bleeding in the subarachnoid space within the cranial vault. The causes of SAH include ruptured cerebral aneurysms, ruptured arteriovenous malformation and hypertensive haemorrhage. The major cause of SAH is cerebral aneurysm.

Learning Activity
Read Subarachnoid Haemorrhage p. 499-502 in OH, T. E, (2003) .

Several systems of grading SAH have been proposed. in T.E. Oh (2003 p. 499).

After re-bleeding, cerebral vasospasm is the main complication of subarachnoid haemorrhage. Cerebral vasospasm is the narrowing of a cerebral blood vessel that results in decreased cerebral blood flow to the area that is normally perfused by that vessel. Decreased cerebral blood flow can cause ischaemia, infarction and death.

As blood enters the subarachnoid space it begins to break down and vasoactive substances, known as spasmodic agents, are released. The breakdown of blood increases the extracellular calcium which moves intracellularly, promoting smooth muscle contraction. Spasmogenic by products and other agents that may be responsible for cerebral vasospasm include throboxane A2 (TXA2), platelets, oxyhaemoglobin (OxyHb), serotonin, plasma and antithrombin III. Each of these directly or indirectly vasoconstrict vessel walls and each is found in increased concentrations within the CSF of patients with subarachnoid haemorrhage. The two most widely researched agents include OxyHb and TXA2.

OxyHb
OxyHb is released during the haemolysis of red blood cells. OxyHb is spontaneously oxidised to methaemoglobin, resulting in the release of a free oxygen radical ion. This free oxygen radical ion contacts the iron of the haemoglobin and facilitates lipid peroxidation. Lipid peroxides are known as vasoconstrictors. OxyHb also prevents the relaxation of constricted arteries and has a synergistic effects with serotonin and potassium causing further vasoconstriction.

TXA2
TXA2 is released by platelets during activation and is a potent vasoconstrictor and platelet aggregating agent. The endothelial damage after subarachnoid haemorrhage is believed to cause activation of platelets and an increased release of TXA2. The increased platelet aggregation and vasoconstriction also contribute to local thrombus formation and ischaemia.

Calcium
Arterial smooth muscle contraction is mediated and regulated by calcium. Calcium moves into the cell via calcium channels within the cell membrane, allowing myosin and actin fibers to slide over each other, and result in smooth muscle contraction. this energy maintains muscle contraction as long as calcium is present.

Blood in the subarachnoid space increases the extracellular calcium concentration which leads to further influx of calcium intracellularly. These actions reduce vascular lumen size and cause increased or prolonged smooth muscle contraction, which results in prolonged or worsened vasospasm.

In addition to vasospasm induced ischaemia, calcium disrupts basic cellular functions. Excess calcium activates membrane phospholipids to release free fatty acids particularly arachondic acid. Metabolism of arachidonic acid produces prostaglandins and leukotrienes that can further damage cell membranes.

Treatment
The goal of treatment in the management of acute vasospasm is to improve the flow of the cerebral microcirculation and elevate the CPP.

Clinical Implications
General supportive care of patients with SAH are essential if the adverse effects of raised intracranial pressure are to be minimised.

Activity
Describe the mechanisms that cause hydrocephalus in subarachnoid haemorrhage (refer to activity on page 11 of this package. How is this complication acutely managed

Nimodipine is lipid soluble thus exerting a greater effect on cerebral arteries than other calcium channel antagonists. Angiographic evidence demonstrates that nimodipine does not prevent the occurrence of deficits by abating cerebral artery spasm alone. Many believe that nimodipine promotes collateral circulation by dilating small pial arteries not visible on angiography. Nimodipine also reduces platelet aggregation, blocks calcium influx into single nerve cells. and endocrine cells thus creating an anticonvulsant effect and enhancing cardiac output, via its afterload reducing effect and compensatory sympathetic stimulation.

Learning Task

Review the Drug infusion manual for Nimodipine

Activity

When first administered intravenously, nimodipine is often slowly increased until the desired dosage is attained. What side effects should you be observing for when giving nimodipine and what drug(s) may be given to minimise this side effect? Why is Nimodipine currently given via oral/nasogastric routes in preference to intravenous?

SECTION 6 INTRACRANIAL PRESSURE MONITORING

The aim of ICP monitoring is to provide a constant and accurate measurement of ICP. There are many different sites used for monitoring ICP, some examples and the advantages and disadvantages these systems are listed below.

Site	Advantages	Disadvantages
Ventricular drains	-More accurate measurement of ICP -Able to drain CSF	-Increased risk of infection -Risk of unintentional loss of CSF -Insertion may be difficult
Intraparenchymal	- Ease of placement - Non fluid filled system	-Potential for brain injury - Risk of infection - No CSF drainage
Subarachnoid	- Ease of placement - No brain penetration - Less risk of infection	- Questionable accuracy - No CSF drainage - Fluid filled system - Brain tissue obstruction
Epidural	- Dura remains intact - Non fluid filled - Ease of insertion	- Questionable accuracy - No CSF drainage

Learning Task

Describe the methods used to monitor intracranial pressure in John Hunter Intensive Care

Activity

It is currently thought that nursing procedures should be organised to allow for adequate rest time (about 20 mins) between each one, rather that "clumping" the procedures together and allowing a longer rest period between each group. True or false. Discuss your answer.

SECTION 7 EXTERNAL VENTRICULAR DRAINS

The external ventricular drain (EVD) is a temporary system which allows drainage of cerebrospinal fluid (CSF) from the lateral ventricles of the brain. The system is commonly used in the intensive care unit for the management of patients requiring drainage of CSF in order to control raised intracranial pressure associated with head injury, subarachnoid haemorrhage and acute hydrocephalus secondary to cerebral aquedunt obstruction, posterior fossa tumours or purulent meningitis, intracranial pressure monitoring and instillation of antibiotics. (RNSH Intensive Care Guidelines 2005)

EVD's are inserted by a neurosurgeon usually in the operating theatre under sterile conditions. The scalp is shaved at the site and an incision is made and a drilled is used to create a small burr hole opening in the skull. The dura is opened and the arachnoid and pia membranes are cauterised. A stylet is used to introduce the ventricular catheter into the frontal horn of the lateral ventricle. The stylet is removed once the catheter is inserted. The catheter is sutured at the insertion site. In adults, the catheter is usually inserted in the ventricles at a depth of approximately 6cm. It is connected to the drainage system and the area covered with an occlusive dressing. (RNSH Intensive Care Guidelines 2005)

Learning Task

Find and read the P & P's relating to EVD's.

Activity

Find the zero reference point for an EVD.

Outline the method of connecting a tranducer to an EVD.

The EVD does not have a pressure valve therefore CSF drainage is dependent on gravity ie the level of the drain determines the amount of CSF drained. The zero point for the DVS system is the location of the foramen of Monro. The drain should be positioned at a prescribed distance in cm above this point. This level will determine the amount of CSF drainage, for example, if the drain is set at 15c, and the ICP is greater than 15cmH_2O, the system will drain CSF to maintain and appropriate pressure. (RNSH Intensive Care Guidelines 2005)

If the drain is placed above the level of the foramen of Monro insufficient CSF will drain and may cause an increase in ICPwith potentially devastating consequences relating to increased ICP. Excessive CSF drainage may occur if the drain is placed below the level of the forament and cause ventricular collapse. (RNSH Intensive Care Guidelines 2005)

Learning Task

Discuss three complications relating to EVD use and there management.

References

BOOKS (Available from Educators)

Hickey, J. (2003) The Clinical Practice of Neurological and Neurosurgical Nursing, 5th Edition. Lippincott Williams and Wilkins, Philadelphia.

McCance K. L. Huether, S. E. (2002) Pathophysiology The Biologic Basis for Disease in Adults and children Fourth Edition Mosby St Louis

OH, T. E, (2003) Intensive Care Manual 5th Edition, Butterworth-Heinemann Oxford.

Urdan, L. D, Stacy, K. M, and Lough, M. E. (2002) Thelan's Critical Care Nursing: Diagnosis and Management, 4th Edition. Mosby. St Louis.

Articles (included in this package)

Cairns, C and Andrews, P (2002) Management of hyperthermia in traumatic brain injury Current Opinion in Critical Care Vol. 8, No. 2, pp. 106-110

Censullo, J.L and Sebastian, S (2003) Pentobarbital sodium coma for refractory intracranial hypertension Journal of Neuroscience Nursing Vol. 35, No.5, pp. 252-262

Coles, J, etal (2002) Effect of hyperventilation on cerebral blood flow in traumatic head injury : clinical relevance and monitoring correlates Critical Care Medicine Vol. 30, No. 9, pp. 1950-1959

Doyle, J.A, Davis, D.P, Hoyt, and David, B (2001) The use of hypertonic saline in the treatment of traumatic brain injury The Journal of Trauma, Injury, Infection, and Critical Care Vol. 50, No. 2, pp. 367-383

Mayer, S.A and Chong,J.Y (2002) Critical care management of increased intracranial pressure Journal of Intensive Care Medicine Vol.17, No. 2, pp.55-67

Mazzola,C.A and Adelson,P.D (2002) Critical care management of head trauma in children Critical Care Medicine Vol. 30, No. 11 supplement, pp. S393-S401

Robertson, C.S (2001) Management of cerebral perfusion pressure after traumatic brain injury Anaesthesiology Vol. 95, No. 6, pp.1513-1517

Yanko,J.R andMitcho,K (2001) Acute care management of severe traumatic brain injuries (part 2) Critical Care Nursing Quarterly Vol. 23, No. 4, pp.1-23

Other References

Arbour, R. (1998) Aggressive management of intracranial dynamics Critical Care Nurse Vol. 18, No. 3, pp. 30-42.

Brucia, J. Rudy, E. (1996) The effects of suction catheter insertion and tracheal stimulation in adults with severe brain injury Heart & Lung, Vol. 25, No. 4, pp. 295-303.

Chudley, S. (1994) The effects of nursing activities on intracranial pressure British Journal of Nursing Vol. 3, No. 9, pp. 454-458.

Jones, B. (1995) The effects of patient repositioning on intracranial pressure The Australian Journal of Advanced Nursing Vol. 2, No, 2, pp. 32-39

Palmer, M. Wyness, M. A. (1988) Positioning and handling; important considerations in the care of severely head injured patients Journal of Neuroscience nursing, Vol. 20, No. 1, pp. 42-49.

Rising, C. J. (1993) The relationship of selected nursing activities to ICP, Journal of Neuroscience Nursing Vol. 25, No. 5, pp. 302-307.

Available on John Hunter Intensive Care Help Library

JHH Intensive Care Unit Clinical Practice Guidelines Head Injury Help Library

JHH Intensive Care Unit Clinical Practice Guidelines Sedation & Analgesia Help Library

JHH Intensive Care Unit Drug Infusion Manual

Journal Articles

Cairns, C and Andrews, P (2002). Management of hyperthermia in traumatic brain injury. Current Opinion in Critical Care ,Vol. 8, No. 2, pp. 106-110.

Censullo, J.L and Sebastian, S (2003). Pentobarbital sodium coma for refractory intracranial hypertension. Journal of Neuroscience Nursing, Vol. 35, No.5, pp. 252-262.

Coles, J, etal (2002) Effect of hyperventilation on cerebral blood flow in traumatic head injury : clinical relevance and monitoring correlates. Critical Care Medicine, Vol. 30, No. 9, pp. 1950-1959

Doyle, J.A, Davis, D.P, Hoyt, and David, B (2001). The use of hypertonic saline in the treatment of traumatic brain injury. The Journal of Trauma, Injury, Infection, and Critical Care ,Vol. 50, No. 2, pp. 367-383.

Mayer, S.A and Chong,J.Y (2002). Critical care management of increased intracranial pressure. Journal of Intensive Care Medicine, Vol.17, No. 2, pp.55-67.

Mazzola, C.A and Adelson, P.D (2002). Critical care management of head trauma in children. Critical Care Medicine, Vol. 30, No. 11 supplement, pp. S393-S401.

Robertson, C.S (2001). Management of cerebral perfusion pressure after traumatic brain injury. Anaesthesiology, Vol. 95, No. 6, pp.1513-1517.

Yanko, J.R and Mitcho, K (2001). Acute care management of severe traumatic brain injuries (part 2). Critical Care Nursing Quarterly, Vol. 23, No. 4, pp.1-23.