Postanesthesia Care of the Patient Suffering From Traumatic Brain Injury

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Abstract:

Each year 1.5 million people in the United States suffer a traumatic brain injury (7BI), and many of these patients require immediate surgery. 7BI patients provide additional assessment and technological challenges for perianesthesia nursing care. A major goal of PACU nursing is the prevention of secondary head injury during the post- anesthesia period.

Objectives—Based on the content of this article, the reader should be able to (1) define the goal of PACU care for the neurotrauma patient; (2) calculate the cerebral perfusion pressure from data obtained from an arterial line and intracranial pressure monitor; (3) describe 4 nursing interventions that increase cerebral perfusion pressure; and (4) describe interventions designed to improve cerebral blood flow.

Article:

WHILE RIDING A BICYCLE without a helmet on a busy street, a 17-year-old male is struck by a car. He is found semiconscious by emergency rescue personnel and transported to the trauma center. On arrival, the patient responds only to deep pain, is unable to follow commands, and is intubated. Pupils are equal and reactive bilaterally. His Glasgow Coma Scale (GCS) score is 8. A computed tomography (CT) scan identifies a brain contusion with hemorrhage and subdural hematoma. The injury is consistent with a coup-coutrecoup injury in which the brain is injured directly beneath the site of the striking force. He is transferred to the OR for a craniotomy for evacuation of the hematoma. The PACU manager is informed that a neurosurgical ICU (NICU) bed is not available for direct admission, and the patient is sent to the PACU for care.

On admission to the PACU, the patient remains intubated and is placed on a ventilator: tidal volume 700, rate of 12/min, FiO_2 of 100%. There are no spontaneous respirations. Vital signs are a blood pressure of 160/94 with a mean arterial pressure of 108, a heart rate of 72, normal sinus rhythm, axillary temperature of 97.6°F, and oxygen saturation of 100%. An end tidal (ET) CO_2 monitor, connected to the endotracheal tube, indicates an ETCO2 of 36 mm Hg. An intraventicular catheter for cerebral spinal fluid (CSF) drainage and intracranial pressure (ICP) monitoring is in place. The ICP is 15, and the cerebral perfusion pressure is calculated as 89. A jugular venous catheter has been inserted for monitoring and the S_jO_2 is 659,66. Physical assessment reveals clear breath sounds. The patient is not responsive but is under full sedation with propofol and chemically paralyzed with vecuronium. A Foley catheter is draining clear,

pale yellow urine. The PACU nurse positions the patient with a 300 head-of-bed elevation and ensures head-torso alignment.

After 45 minutes in the PACU, the SjO2 level decreases to 589,66 with an increase in ICP pressure to 19 mm HG. After troubleshooting the SjO_2 monitor for a false low reading, the monitor is recalibrated and the patient's SjO_2 returns to 629,66. The PACU nurse continues to monitor the patient over the next 3 hours, minimizing external stimulation by clustering nursing interventions. The patient is transferred to the NICU in stable, but critical, condition for continued care. Together, the PACU and NICU nurses complete a neurological assessment to ensure continuity of care.

Traumatic Head Injury

Head injuries, including any trauma to the scalp, skull, or brain tissue, are described as traumatic brain injury (TBI), acute head injury, or closed head injury. A GCS of 8 or less indicates severe head injury.[1] The primary causes of TBI are motor vehicle crashes and accidents involving a bicycle, pedestrian, or recreational vehicle.[2] Head injuries are the most likely injury to cause death or permanent disability in the United States. Males are twice as likely as females to sustain a TBI, with persons age 15 to 24 and those over 75 at highest risk.[3] Each year 1.5 million people in the United States suffer a TBI, which is 8 times the number of people diagnosed with breast cancer.[4] Of the 1.5 million persons per year with a TBI, 50,000 die, 230,000 are hospitalized and survive, and 80,000 to 90,000 will experience long-term disability.[5] The traumatic event causing the head injury is referred to as the primary injury. Secondary brain injury can occur as a result of ischemia, secondary hemorrhage, and cerebral edema .6 The goal of nursing care is the prevention of secondary injury. PACU Standards of Care as well as evidence-based Guidelines for the Management of Severe Head Injury[1,7] provide a framework for nursing actions.

Respiratory Care

TBI patients are mechanically ventilated and assisted by a chemically induced coma to ensure low $PaCO_2$ levels. Hypocarbia promotes cerebral vasoconstriction. Careful monitoring of the PaCO2 can be accomplished by obtaining arterial blood gases, through continuous monitoring using end tidal capnography, or invasively via sensors placed on the cortex of the brain surface. $ETCO_2$ measurements are generally 5 mm Hg lower than a $PaCO_2$ derived from the arterial blood gas. If hyperventilation is necessary, the goal is to maintain the $PaCO_2$ between 30 and 35 mm Hg. A $PaCO_2$ less than 35 mm Hg can compromise cerebral perfusion during the first 24 hours and must be avoided.

Hypoxemia, a PaO_2 level less than 60 mm Hg, must be prevented to avoid cerebral ischemia.[7,8] Although the airway must be kept free of secretions, suctioning, known to increase ICP, must be kept to a minimum. Preoxygenating with 1009,66 oxygen for 1 minute before suctioning and limiting suctioning to 1 or 2 passes of less than 10 seconds each is recommended.

Neurological Care

The Monro–Kellie hypothesis describes the skull as a closed system with 3 components: blood ($109{\cdot}6^6$), CSF ($109{\cdot}6^6$), and brain tissue ($809{\cdot}6^6$). Normal ICP is 0 to 15 mm Hg and can be assessed invasively with intraventricular catheters. ICP can be monitored by epidural, subarachnoid, parenchymal, or intraventricular devices. Intraventricular devices are considered the

gold standard. There are 3 types of intraventricular devices used to measure ICP: external fluid-filled strain-gauge transducers, fiber-optic sensors, and microstrain-gauge sensors.[9] Most

Table 1. Actions That Increase Intracranial Pressure

Respiratory
Suctioning
Intubation
Coughing
Body positioning
Trendelenburg
Prone
Extreme hip flexion
Neck flexion
Activity
Valsalva maneuver
Isometric exercises
Seizures
REM sleep or arousal from sleep
Noxious stimuli
Emotional distress

Table 2. Interpreting Cerebral Perfusion Pressure

Normal CPP	80-100 mm Hg
Recommended minimal level in TBI	>70 mm Hg
Minimally adequate blood supply	60 mm Hg
Autoregulation begins to fail	<50 mm Hg
Cerebral blood flow decreased by 25%	<40 mm Hg
Neuronal hypoxia and cell death	<30 mm Hg

NOTE. CPP = mean arterial pressure (MAP) − intracranial pressure (ICP).

patients will have an intraventricular catheter attached to an external strain-gauge transducer, the most accurate, low-cost, and reliable method of monitoring ICP. The intraventricular catheter allows for CSF sampling, calculating of CPP, and administration of medications. The intraventricular catheter can also be used as a ventriculostomy to drain CSF in an effort to decrease ICP.

In a traumatized brain, even small increases in cerebral volume can cause elevations in ICP. ICP is increased with any abnormal production, circulation, or absorption of CSF, any increase in intracranial blood volume, or any increase in brain tissue volume. Specific nursing activities can also increase ICP (Table 1).

Through autoregulatory mechanisms the cerebral circulation seeks to maintain a constant blood flow and a constant blood pressure necessary to provide oxygen and nutrients to the brain. There is evidence that this autoregulation remains intact in many cases of TBI. Cerebral perfusion

pressure (CPP) determines cerebral blood flow. CPP can be calculated by subtracting the ICP from the mean arterial pressure (MAP). CPP calculation, monitoring, and interpretation are included in a PACU neurological assessment (Table 2).

Caring for a TBI patient requires attention to elevations in ICP, especially as they are related to patient care activities. If the ICP level rises, the surgeon is notified immediately. Excessive ICP can result in brain herniation, ischemia, and infarction. Specific measures may be ordered to lower ICP, including hyperventilation, controlled drainage of CSF, and administration of osmotic diuretics such as mannitol. Glucocorticoid therapy, once used in severe head injury, is no longer recommended.[1]

Monitoring of ICP and CPP does not always provide reliable indicators of cerebral ischemia.[10] The TBI patient may require a jugular venous catheter for SjO_2 monitoring. This catheter is threaded through the internal jugular vein into the jugular bulb at the point where venous blood leaves the intracranial vault. SjO_2 provides a more accurate measure of cerebral metabolism. SjO_2 monitoring, in conjunction with CPP, demonstrates better patient outcomes than when CPP, MAP, or ICP alone are

Table 3. Monitoring SjO_2 Values

Normal	55-80% mm Hg
Critical	<50% mm Hg
Hyperemia	>75% mm Hg

Data from references 7, 8, and 11.

Table 4. Interventions for SjO_2 Desaturation

Increasing $PaCO_2$
Increasing hemoglobin level
Increasing MAP
Fluid administration
Vasopressors: dopamine and phenylephrine
Decreasing ICP
CSF drainage
Resuming normothermia
Pharmacology
Sedatives: midazolam and lorazepam
Propofol
Barbiturates

used to determine treatment.[11] Levels of S_jO_2 over 80% can indicate hyperemia (Table 3). Hyperemia is the result of the brain receiving more blood flow than needed or the presence of dead tissue, which does not extract oxygen. If SjO2 levels become too low, less than 50%, critical intervention is required (Table 4). Increased ICP is a frequent cause of jugular bulb oxygen desaturation.

Research has found that 15% to 50% of S_jO_2 desaturation readings may be false, [12] so equipment troubleshooting is necessary before further actions are taken. False readings can be due to low intensity from the light source. If the light intensity meter is working properly, the next step is to

flush the SjO_2 catheter. Two to 3 mL of isotonic sodium chloride solution is used. The patient's head and neck can be repositioned to move the tip of the catheter to an area of the internal jugular vein with greater blood flow. A blood sample via the jugular catheter can be obtained and analyzed to check the accuracy of the oximeter. A difference of more than 4% between blood sample oxygen saturation and S_jO_2 monitor readings indicates a need to recalibrate the monitor.

Neurological assessments and vital signs must be documented at least every 15 minutes in the postoperative TBI patient. Neurological assessments are a priority because vital signs are the last parameters to change in a head injury. Because the patient is fully sedated to decrease cerebral metabolism and lower ICP, a level of consciousness cannot be determined. Pupils are assessed for size, shape, equality, and reaction to light. Anesthetic and preoperative medications, noted to have an effect on pupil size and reactivity, must be considered when evaluating pupil findings (Table 5).

The assessment data are analyzed for Cushing's triad: increased systolic blood pressure, widening pulse pressure, and decreased pulse rate. The triad indicates an increase in ICP. The physician should be notified immediately and a treatment plan instituted.

Cardiac Care

Historically, ICP was managed in patients with head injuries by using dehydration and preventing the ICP from rising above 20 mm Hg. Current guidelines call for reducing systemic hypertension while avoiding systemic hypotension. Hypotension, a systolic blood pressure less than 90 mm Hg, must be avoided because research has determined that prolonged hypotension has a significant detrimental effect on outcomes in severe head injury patients.[1] The MAP should be maintained at 90 mm Hg to allow for a CPP greater than 70 mm Hg, which provides adequate perfusion of the brain and prevention of tissue ischemia.[11]

Cardiovascular alterations are common after head injury and can include increased blood

Table 5. Common Perianesthesia Drugs That Influence Pupil Assessment

Miotics = Constrictors	Mydriatrics = Dilators
Remifentanil	Atropine sulfate
Fentanyl	Glycopyrrolate
Sufentanil	Scopolamine
Morphine sulfate	Epinephrine
Neostigmine bromide	Ephedrine
Edrophonium chloride	Ketamine
Pyridostigmine bromide	Metaraminol
	Trimethaphan

pressure, increased cardiac output, and tachycardia.[13] The management of the patient's blood pressure requires a delicate balance between the risk of hypoperfusion, worsening edema, and increased ICP. Ideal agents for maintaining blood pressure in TBI patients are easily titratable intravenous drugs that have minimal side effects and a short duration of action. Intravenous neosynephrine is frequently ordered to regulate the MAP within 90 to 115 mm Hg.

Normothermia
Temperature monitoring is especially important for the brain-injured patient. Hypothermia that causes shivering will increase oxygen demand and increase ICP. Hyperthermia will also increase ICP and may be a symptom of hypothalamic damage. To prevent shivering, neuromuscular blocking agents such as pancuronium and vecuronium are often used. [14] In addition, the use of antipyretics and cooling blankets can be used to maintain normothermia.

Positioning
The TBI patient is always positioned to promote venous drainage from the brain. Elevation of the head above the level of the heart will reduce ICP but may compromise CPP. A 30° head-of-bed elevation has been found to reduce ICP while maintaining CPP.[15] Attention to the patient's head, neck, and chest alignment will prevent obstruction of venous drainage. Cervical collars and endotracheal or tracheostomy tube ties require careful monitoring so that venous drainage is not limited. Paralyzed limbs must be properly aligned and supported in functional positions with pillows and wedges.

Safety
The corneal or blink reflex of the TBI patient may be absent either because of injury or chemical paralysis. The eyes must be irrigated with sterile saline or a lubricant applied. The eyes are not taped shut to avoid corneal abrasions.

To prevent infection, aseptic technique must always be used when dealing with invasive monitoring devices. While performing routine site care for the ICP catheter, observe for CSF drainage. A halo or ring sign, which is drainage with a blood-tinged center surrounded by lighter colored concentric rings, on bed linens or dressing can indicate CSF. If drainage is noted, it can be checked with a dextrose stick. CSF and blood contain glucose; mucus does not.

The patient is placed on seizure precautions and anticonvulsants will likely be ordered. Although overt seizures in the paralyzed patient are difficult to assess, seizure precautions and careful assessment for even small changes in the patient's condition are needed. No two head- injured patients behave or respond in the same way. [16]

Conclusion
TBI patients provide additional assessment and technological challenges for postanesthesia nursing care. The PACU nurse plays a critical role in the prevention of secondary head injury. Excellence and diligence in neurological assessment skills and an understanding of the complex care needs for maintaining adequate cerebral perfusion pressure are required. With appropriate knowledge and skilled nursing care, the TBI patient will be provided an opportunity for a full recovery.

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