

Multimodal Monitoring: Head Injury Management Using SjvO₂ and LICOX

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Abstract: Monitoring methods following severe head injury currently use indirect measurements of cerebral oxygenation and perfusion defined as intracranial pressure (ICP) and cerebral perfusion pressure (CPP) monitoring. Adequate information regarding cerebral blood flow and oxygenation is necessary to guide treatment and prevent secondary cerebral ischemia. Because of the ineffectiveness of ICP and CPP monitoring in detecting early ischemic changes in healthy, as well as compromised, brain tissue, patients' neurological outcome and recovery may be less than optimal. New technology has been recently developed to provide early detection of poor cerebral oxygenation and perfusion. The LICOX brain tissue oxygenation and jugular venous bulb catheters are two types of monitors currently available. Patients can benefit from a unique combination of multimodal monitoring when cerebral oxygenation and consumption measurements are quantified on both a local and global level. This unique combination helps reflect early changes in brain tissue viability. As with any new monitoring device, there are associated advantages and disadvantages as well as cost issues, to consider. Patients with head injuries can benefit from technology that guides decisions and prevents treatment delays.

Head trauma results in 500,000 hospital admissions (Archibald, S.J., Fleckenstein, W., Littlejohns, L., O'Grady, J., & Trimble, B., 2001) and represents more than two thirds of in-hospital deaths annually. It carries a high mortality rate among young Americans today (Sarrafzadeh et al., 2001). Brain injuries are a continuous challenge on both a national and global scale. Without adequate intervention, head injuries lead to severe physical and mental disability, as well as death. The current cost of treatment and rehabilitation of one severely brain-injured patient is more than \$300,000 (Haselman & Fox, 2000). This cost does not include the eventual long-term cost of care needed after a patient is discharged from a rehabilitation facility.

Graham, Lawrence, and Adams (1988) contend that ischemic changes were found postmortem in 70% of injured brains without previous evidence of clinical, pathological, or radiological changes indicative of

increased intracranial pressure (ICP). Adverse effects (e.g., hypoxemia, hypotension, and elevated ICP) may cause secondary cerebral ischemia. Graham et al. (1998) suggest these insults remain undetected with the use of current technology and may worsen patient outcomes. In addition, concomitant multiple trauma places the patient at risk for cerebral ischemia because of other complications, including hyperglycemia secondary to physiologic stress, hypovolemia, and shock. The brain consumes close to 20% of the body's oxygen (Maloney-Wilensky et al., 2002). It does not contain an oxygen reservoir from which to draw during acute illness. This further emphasizes the fact that the brain is extremely fragile, because of its inability to rely on a steady blood flow after a traumatic injury. Therefore, it is important that initial care is directed toward life-threatening injuries that may affect brain oxygen supply and demand.

Multimodal intracranial monitoring provides useful observation of specific changes as well as an opportunity for early intervention. LICOX[®], which evaluates local ICP, brain temperature, and brain tissue oxygenation (PbtO₂) values, as well as the jugular venous bulb (SjvO₂), which monitors global brain tissue oxygen consumption, are the two monitors in use today. Multimodal, or multitechnique, monitoring (van den Brink, et al. 1998) involves the use of LICOX together with the SjvO₂ monitors to synergize beneficial information regarding treatment for a particular patient suffering from a traumatic brain injury. Other current technology exists to gather information and guide treatment in brain-injured patients such as ICP monitors, ventriculostomy drainage systems, as well as continuous bedside electroencephalograms (EEGs). By including the use of PbtO₂ and SjvO₂ monitoring, intracranial hemodynamics status can be accurately obtained. This article explores the capabilities of both the LICOX and the SjvO₂ catheters in detecting cerebral ischemia, as well as implications for practice and future potential in critical care management.

Current Cerebral Function Monitoring and Management

Head Trauma Complications

Enhancing cerebral blood flow (CBF) in a traumatic brain-injured patient becomes complicated because of continuous metabolic demands and changes. Hypoxia, for example, secondary to hypoperfusion or an underlying pulmonary process, may indirectly affect the

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oxygenation of brain tissue due to changes in CBF. Vasodilation decreases CBF and perfusion when decreased oxygen and increased carbon dioxide levels result from poor ventilation. In addition to hypoxia and hypoperfusion, cerebral edema may develop and cause an elevation in ICP that may further impede oxygen delivery to the brain. For accurate management, these adverse events are best monitored invasively in a traumatic brain injury.

ICP Versus CPP

Cerebral perfusion pressure (CPP), a parameter that is a calculated value from ICP and mean arterial pressure (MAP) values, is an indicator of general cerebral perfusion, as well as CBF. The debate is whether to use ICP or CPP targeted management to maintain adequate CBF, because CPP is a more reliable indicator of cerebral oxygenation than ICP alone. According to van den Brink et al. (1998), CPP is affected as fluctuations in MAP and ICP occur, regardless of whether these values are within normal range for the patient. Treatments can be used to maintain both the ICP and CPP to support adequate CBF. This is important to provide adequate oxygenation and CBF to the brain (Robertson et al., 2001).

ICP and CPP values are currently used to evaluate and guide treatment in head trauma. Although they provide useful information, these parameters provide only limited information about brain tissue perfusion and oxygenation. For this reason, both LICOX and S_{jv}O₂ monitors are available to provide up-to-date information regarding the status of brain tissue oxygenation and to indicate the need for early intervention and treatment. It is important to note that over time, immediate and long-term management varies depending on the stability of the brain injury. Brain oxygenation and perfusion values such as CPP, ICP, PbtO₂, and S_{jv}O₂ may eventually normalize without a change in neurological function. At that point, it is important to prevent overtreatment of the initial brain insult and begin appropriate rehabilitation therapy.

ICP and calculated CPP are currently used to manage patients with head injuries. These are less specific, as well as less sensitive, indicators of adequate cerebral perfusion and oxygenation. With the use of LICOX and S_{jv}O₂ monitors, information regarding intracranial hemodynamics not only includes ICP measurements but also actual brain tissue oxygenation and consumption via continuous bedside monitor readings. This allows efficient management of viable brain tissue to optimize a patient's future recovery.

Multimodal Monitoring

LICOX (PbtO₂) Monitor

Changes in ICP values alone do not accurately depict poor cerebral blood flow or oxygenation deficits to brain tissue. PbtO₂ monitoring via the LICOX system is a valuable assessment tool, because it provides early detection

of impending ischemic events (Meixensberger, Dings, Kuhnigk, & Roosen, 1993). LICOX provides information on early changes in cerebral oxygenation, perfusion, and temperature before ICP changes. LICOX measures regional rather than global areas of arterial oxygenation and perfusion when placed in the penumbra surrounding the injury. Normal PbtO₂ values range from 37 to 47 mm Hg. A value below 37 mm Hg is indicative of a swollen cortex and possible ischemia that could lead to permanent neuronal injury (Valadka, Gopinath, Contant, Uzura, & Robertson, 1998).

When PbtO₂ values decrease, areas of brain tissue may be deprived of adequate oxygenation. Causes of low PbtO₂ values include seizures, cerebral edema, intracranial hemorrhage, as well as intracranial hypertension. A critical low range is considered to be between 5 and 10 mm Hg (Valadka et al., 1998). Valadka and colleagues found that a PbtO₂ of ≤ 5 mm Hg within the first 24 hours results in death as compared with a PbtO₂ of > 5 mm Hg during the first 24 hours. Van den Brink et al. (2000) state that a PbtO₂ < 5 mm Hg for 30 minutes poses a 50% risk of death. Moderately hypoxic levels of < 10 mm Hg are also associated with a 50% mortality risk if this level persists for more than 1 hour and 45 minutes. Patients with PbtO₂ levels of < 15 mm Hg in the presence of mild hypoxia have a 50% chance of death if left untreated up to 4 hours or more. Overall, the longer the patient's PbtO₂ levels are < 15 mm Hg, the greater the chance of a fatal outcome. In contrast, a PbtO₂ of > 35 mm Hg is associated with a good recovery, whereas those patients with levels of PbtO₂ 26–35 mm Hg remained disabled, and lastly those patients with a PbtO₂ of ≤ 25 mm Hg were linked to an early death or persistent vegetative state (Zauner et al., 1998).

LICOX plays an important role in the implementation of multimodality monitoring in intensive care units. The LICOX system is supplied with a temperature, oxygenation, and ICP probe—all three monitors are incorporated into one device and inserted at the same time, and in the same place. The temperature probe is considered by the LICOX manufacturer to have an accuracy rate of up to 0.2°C of the current temperature of any tissue. In head trauma, the probe can read in Celsius up to 0.8° higher than the core temperature. This temperature probe is considered to be accurate for up to 5 days. LICOX is also capable of obtaining extensive and precise PbtO₂ values from regional areas of the brain, as well as a global ICP measurement.

LICOX Management

ICP and brain tissue oxygenation readings may be followed simultaneously. However, these numbers may not correlate, which is why the oxygenation probe is a valuable tool in the head injured population. For example, a low or fluctuating PbtO₂ may or may not be associated with an increase in ICP. This reinforces the fact that

changes are noted earlier with the LICOX probe versus an ICP monitor alone and allows for a comparison between early versus late detection of cerebral changes in oxygenation. However, in the case of an elevated ICP, values in the face of a normal PbtO₂ reading may imply a malfunction of the autoregulatory system within the brain and a hyperemic state that increases CBF such as when a patient is febrile (Stiefel et al., 2003). In this case, an elevated ICP may not reflect impending cerebral ischemia. Normal monitoring parameters, as well as brain tissue values detected by the LICOX monitor, are found in Table 1.

LICOX Placement Issues

Accurate placement of LICOX catheters is crucial to obtain reliable readings. Catheters are usually placed on the right side in the subarachnoid space of the frontal lobe with the oxygen probe extending into the white matter. Placement on the left may cause injury to the speech center of the brain. A computed tomography (CT) scan can be used to locate the area of damage, and then the LICOX catheter may be placed within the penumbra of the injury. If the area of vasospasm or blood clot is thicker on the right, then the catheter will be placed on the right, and if the area of thicker blood is on the left, then it is placed on the left. If the area of vasospasm or injury cannot be accurately determined, such as with diffuse axonal injuries, then the LICOX is placed on the right (Maloney-Wilensky et al., 2003). The LICOX probe should be placed 14 mm from other probes such as a ventricular drain. Brain tissue may take up to 120 minutes to settle before readings may be obtained. Next, an oxygen challenge test should be performed to check the reliability of the LICOX. This is achieved by setting the ventilator to deliver 100% oxygen to the patient, and if the probe is working correctly, the PbtO₂ will respond by increasing in value. If no response occurs, a head CT scan should be completed to confirm placement (Maloney-Wilensky et al.). A probe rarely produces false values because the oxygen sensor is located <5 mm from the distal end of the probe (Valadka, Gopinath, Contant, Uzura, & Robertson, 1998).

PbtO₂ values vary greatly depending on placement

of the LICOX, as well as insults during placement into brain tissue. A patient may have a good recovery because viable tissue may exist in surrounding areas despite low PbtO₂ readings secondary to localized placement in the penumbra of the injury, because viable tissue may exist in surrounding areas. Low readings may result when placement of a probe causes microvascular compression within the injury penumbra (Bruzzzone, Dionigi, Bellinzona, Imberti, & Stocchetti, 1998). Furthermore, placement in contusions, infarcts, and hemorrhagic areas may adversely affect oxygen measurements and may cause display of low readings that may be refractory to treatment. As a result, local placement by LICOX may potentially lead to either overtreatment or undertreatment of viable brain tissue because of the limited information provided on surrounding areas.

Conversely, placement with an undamaged area is beneficial as it reflects changes in global oxygenation. PbtO₂ readings are indicative and influenced by oxygen exchange within the capillary beds and by the diameter of these vessels that surround the catheter (van den Brink et al., 1998). After placement, the probe takes up to 80 minutes to adapt to brain tissue and record accurate data (van Santbrink, Maas, & Avezaat, 1996). A malfunctioning catheter may provide low readings for 6 hours from the time of initial insertion and calibration despite increasing oxygen delivery via ventilator (Pollina & Gibbons, 1999). It is fortunate that the LICOX is stable in brain tissue for up to 16 days, and therefore recalibration is unnecessary (Pollina & Gibbons, 1999). Absolute

Table 1. Relationship Between LICOX and SjvO₂

Indications (Average CBF: 50–55 ml/100 gm brain tissue/min)	SjvO ₂ (global/venous)	PbtO ₂ (local/arterial)
Normal values	55%–75%	37–47 mm Hg
Critical values	< 50%	< 25 mm Hg
Global decrease in CBF (anemia, ischemia, hypotension, hypovolemia, hypoxia, ICP)	Decrease	Decrease
Regional decrease in CBF and O ₂ delivery (ischemia)	Normal or increase	Decrease
Cerebral O ₂ demand > supply (seizures, ICH, pain, agitation, fever)	Decrease (54%) (Due to increased O ₂ extraction)	Decrease (< 20 mm Hg) (Before increase O ₂ delivery via ventilator, FiO ₂)
Cerebral O ₂ supply > demand (hyperemia, increase in PaCO ₂ , increase in CBF, hyperoxia, anesthesia, paralysis)	Increase (> 90%)	Increase (> 40 mm Hg)
Hyperventilation management	Increase	Increase (depends on location of LICOX)
Sensitivity for ischemia	63.5%	69.7%

Sources: Archibald, Fleckenstein, Littlejohns, O'Grady, J., & Trimble, 2001; Bader, Littlejohns, & March, 2003; Clay, 2000; Littlejohns, Bader, & March, 2003; Maloney-Wilensky et al., 2003; Meixensberger, Dings, Kuhnigk, & Roosen, 1993; Meixensberger, Jager, Dings, Baunach, & Roosen, 1997; Schell & Cole, 2000; Van den Brink et al., 2000.

contraindications for placement of LICOX are coagulopathy, defined as a platelet count of <50,000, and insertion through a craniotomy bone flap as it is without stability. Fortunately, the actual risk of infection or a contusion produced by the placement of a LICOX monitor is relatively low at about < 2% (Maloney-Wilensky et al., 2003).

SjvO₂ Monitor

Schell and Cole (2000) contend that monitoring of oxygenation of cerebral venous outflow via the jugular vein has been a topic under investigation for more than 50 years. SjvO₂ monitoring was first developed and applied to the care of head-injured patients in the 1980s (Gopinath, Valadka, Uzura, & Robertson, 1999). It measures global cerebral oxygenation supply and demand by measuring total venous brain tissue extraction of oxygen. Normal SjvO₂ values are 60%–80%, whereas a reading of <50%–55% is considered critical. SjvO₂ monitors are able to detect desaturations within the first 48 hours of a brain injury as they are mainly used on a short-term basis (Clay, 2000). Fluctuating SjvO₂ values can guide the management of sedation, fluid administration, a barbiturate-induced coma, as well as hyperventilation strategies. Over time, effective implementation of the SjvO₂ monitor could prevent adverse outcomes in patients who require continuous life support.

SjvO₂ Management

Cerebral extraction of oxygen, or SjvO₂ monitoring, has been proven to be beneficial in managing head injuries in the acute phase. The acute phase, according to Bouma et al. (1991 as cited in Cruz, 1998), is the first 12 hours since the initial brain injury occurred. After 12 hours, acute intracranial hypertension (ICH) may develop, reflected as low SjvO₂ values (i.e., <50%). Values obtained by the SjvO₂ catheter are relied on over a short time period usually less than 5 days. (Kiening, Unterberg, Bardt, Schneider, & Lanksol, 1996). However, the initial management, 24 to 48 hours following a head injury, is crucial to outcome and survival. In multiple trauma patients, hypotension secondary to blood loss complicates management. In patients without a significant extracranial blood loss, ICH may develop because of intracranial hemorrhage and may be refractory to medical management. These patients usually require maximum medical therapy and hyperventilation strategies or an emergent craniotomy to prevent cerebral ischemia.

Cruz states that the SjvO₂ monitor, along with a calculated CPP, is useful in guiding management of acute ICH (1998). Without an SjvO₂ catheter, monitoring CPP alone does not reflect adequate brain oxygenation needs as well as impending ischemia (Cruz). ICP and CPP are valuable indicators of cerebral blood flow; however, these numbers alone do not accurately interpret true cerebral hemodynamics during the acute stages of a

brain injury. Cruz indicates that when ICP is >30 mm Hg and CPP is optimized, the brain tissue oxygenation values continue to decline despite supportive vasopressor and metabolic therapy. Furthermore, there were patients in the CPP or control group who required at least 10 days versus those in the SjvO₂ and CPP group, who required only 6 days or less of acute ICH management. Those in the control group without SjvO₂ monitoring could not benefit from hyperventilation because brain oxygenation values were not monitored and cerebral ischemia would be less detectable. The optimization of hyperventilation assisted in normalizing SjvO₂ values, as well as cerebral glycemic and oxygenation needs. Monitoring SjvO₂ improves overall patient outcomes by decreasing the need for long-term vasopressors and allowing the use of hyperventilation therapy to prevent recurrence of unstable episodes of ICH and potential cerebral ischemia.

Placement of SjvO₂

Optimal placement of the SjvO₂ catheter is based on several factors. The left and right jugular veins are asymmetrical because the right internal jugular is larger in diameter. As a result, venous blood from both cerebral hemispheres drains unequally through the jugular veins with up to a 10% difference in readings. Most patients' head injuries are monitored with the SjvO₂ catheter inserted on the ipsilateral side of the head injury. However, the larger right jugular vein facilitates insertion on that side. In the case of a bilateral insult, the SjvO₂ is placed in the right internal jugular vein. Normal monitoring parameters as well as indications for treatment for SjvO₂ are found in Table 1.

Advantages and Disadvantages of SjvO₂ Placement

The accuracy of the SjvO₂ catheter is dependent on several factors. These factors are the position of the catheter and patient, as well as attention to light intensity levels of the catheter, which all lead to the need for continuous recalibration. It is placed above C1 via a large intravenous catheter and is checked by X ray for correct placement to minimize extracerebral contamination (Fig 1). The Trendelenburg position is necessary for placement, and therefore patients with unstable ICPs may not tolerate insertion unless placed under appropriate sedation or in surgery. At times, a Doppler is used (Clay, 2000) to minimize placement difficulty. Arterial blood gases are drawn to calibrate the SjvO₂ catheter, which is connected to a bedside monitoring device similar to a continuous cardiac output monitor. The catheter frequently lodges against the vessel, or the light at the end of the probe may malfunction and display low readings. Contraindications include cervical spine injury, brainstem injury, neck trauma, presence of a tracheostomy, coagulopathy, as well as jugular venous occlusion or malformation. Complications from placement include pneumothorax, carotid artery puncture, nerve injury,



Fig 1. X ray confirming placement of the jugular venous bulb infection, thrombosis, and increased ICP secondary to obstruction of venous return (Schell & Cole, 2000). Troubleshooting complications associated with both the S_{iv}O₂ and the LICOX monitor are found in Table 2.

Costs

LICOX

The total package to place a PbtO₂ (LICOX) monitor including the access tray is approximately \$16,000. The cost of the catheter alone is about \$900. Each of the catheters may be ordered separately as well, if needed. Separate access trays that are used by neurosurgeons, as well as intensivists, to place a LICOX at the bedside cost \$194. These trays include the sheaths, introducers, and a mechanical drilling device to place the PbtO₂ probe. The bedside monitors cost approximately \$15,000 and the cables that connect the intracranial oxygen and temperature probes to the monitor are included in this cost. These cables may be replaced separately from the box if displaced or malfunctioning but are costly as well. There are no daily charges except for maintenance care of the site with daily dressing change kits that are relatively inexpensive. LICOX has been approved by the FDA to remain in place for up to 5 days. A head CT scan is necessary to confirm placement, which is an added cost to the use of LICOX.

S_{iv}O₂

The S_{iv}O₂ monitor is less expensive than LICOX, and the total price for the catheter is about \$200–250. This does not factor in the use of a central-line kit as well as

the transducer, bedside oxygenation monitor, occlusive dressing, and the use of several arterial blood gas syringes frequently used on a daily basis to calibrate the machine. Although, the S_{iv}O₂ monitor is less expensive than the LICOX monitor, the two monitors are most beneficial when used together. They provide synergistic information when managing any patient with a severe brain injury.

CPP/CBF Management Using S_{iv}O₂ and LICOX

Both the LICOX (PbtO₂) and the S_{iv}O₂ monitor are indicators of cerebral oxygenation and consumption. However, S_{iv}O₂ is considered a more sensitive indicator of cerebral oxygenation and CBF than PbtO₂ due to its capability to monitor global cerebral tissue oxygenation (Schell & Cole, 2000). In many cases, LICOX is the preferred choice for monitoring head injuries if it is impossible to place both monitors. At times, S_{iv}O₂ catheters display at or above normal values during hypoxic events, which indicates increased extracerebral contamination and displays falsely elevated S_{iv}O₂ values that reflect an increased systemic versus cerebral influence secondary to decreased oxygen extraction in areas of ischemia.

PbtO₂ is considered an acceptable measurement of cerebral oxygenation for guiding CPP management to optimize CBF as well. Although studies continue to determine the correlation among ICP, CPP, and PbtO₂ levels, LICOX detects poor oxygen perfusion in brain tissue before changes in ICP occur (Artru, Perret-Liaudet, Charlot, & Mottolese, 1998). For example, LICOX monitors placed in brain tissue away from the site of injury have detected a decrease in oxygen levels even when the ICP is within normal limits (Artru et al.). A second study by Hartl and colleagues (1997) showed that when CPPs are low and ICPs are decreased by administering mannitol IV, PbtO₂ levels remain unchanged. A case report by Stiefel et al. (2003) also states that elevated ICPs may also be correlated with normal PbtO₂ values; therefore aggressive treatment is not necessary as signs of cerebral hypoxia are not apparent.

S_{iv}O₂ and LICOX probes are invaluable tools when used together to depict a comprehensive and realistic outlook of a patient's cerebral oxygenation and metabolism in the acute phase of head trauma. A correlation was found between CBF (CPP), the S_{iv}O₂, and the PbtO₂ monitors in the prevention of cerebral hypoxia (Kiening, et al. 1996). This study found that when the CPP is <60, both the S_{iv}O₂ and LICOX readings would begin to decline as well. Whereas at a CPP of >60, both PbtO₂ and S_{iv}O₂ correlated indicating adequate cerebral oxygenation and perfusion. However, as previously stated by Cruz (1998), CPP may be optimized in the face of an elevated ICP, yet brain tissue oxygenation is compromised because of increased CBF and persistent ICP.

Table 2. PbtO₂ and SjvO₂ Monitoring: Troubleshooting Tips

PbtO ₂ and SjvO ₂	Troubleshooting Tips
Equipment Malfunction <ul style="list-style-type: none"> Broken cable/catheter/light sensor malfunction Inaccurate calibration <i>in vitro</i> or <i>in vivo</i> Artifact Power supply interruption: LICOX restarts automatically, no recalibration necessary 	<ul style="list-style-type: none"> Obtain backup equipment. Check calibration machine. Recalibrate SjvO₂ with ABG. May take up to 6 hours to calibrate. Minimize excessive movement, use appropriate sedation. Reposition head/neck (if spine cleared). Reposition catheter (may be against vessel wall).
Catheter Dislodgement	<ul style="list-style-type: none"> Secure catheter at site/stabilize for transports. Reconfirm placement with head CT for LICOX or head/neck X ray for jugular venous bulb
Placement Complications	Treatment/Management
SjvO₂ <ul style="list-style-type: none"> Hemorrhage with placement in carotid artery; hematoma at site. Puncture through jugular vein, obstruction of venous return (increase in ICP), thrombosis, nerve injury, infection 	<ul style="list-style-type: none"> Optimally, correct coagulation times before placement, platelet count < 50,000 contraindicates placement of intracerebral catheters Administer clotting factors as needed. Protect airway and draw ABGs to recalibrate with CCO monitor (if SjvO₂).
PbtO₂ <ul style="list-style-type: none"> Inaccurate or low readings after placement, infection 	<ul style="list-style-type: none"> Increase FiO₂ on vent to 100% x 5 minutes, if no improvement; head CT scan to check placement.
Infection	<ul style="list-style-type: none"> Maintain exit site of catheter with dressing, clean site. Remove and replace in new area if needed.

Sources: Archibald, Fleckenstein, Littlejohns, O'Grady, & Trimble, 2001; Bader, Littlejohns, & March, 2003; Littlejohns, Bader, & March, 2003; Maloney-Wilensky et al., 2003; Meixensberger, Dings, Kuhnigk, & Roosen, 1993; Meixensberger, Jager, Dings, Baunach, & Roosen, 1997; Van den Brink et al., 2000; Can Santbrink & Maas, 1996.

Implications for Nurses

The first 24 hours after a traumatic brain injury or intracranial bleed is the most critical time to monitor changes in brain tissue oxygenation. Conventional treatment without SjvO₂ and LICOX monitoring includes sedation, muscle paralysis, hypothermia, cerebral spinal fluid drainage via a ventriculostomy, hyperventilation, monitoring arterial blood gases to keep PaCO₂ at 35 mm Hg or less; administering diuretics; and lastly, using vasopressors to maintain a CPP of >70. For the treatment of refractory elevated ICPs, a patient may be placed into a barbiturate coma using a bedside EEG. In addition to vasopressors, judicious use of fluid replacement may be an important tactic as well to keep a patient closer to euvolemic than hypervolemic. This may be achieved through the administration of blood products as well as hypertonic or normal saline and other plasma expanders such as hetastarch. Nurses, along with respiratory therapists, can collaborate at the bedside to directly enhance brain tissue oxygenation content by increasing the amount of inspired oxygen on

the ventilator as well. In general, the use of both the LICOX and SjvO₂ monitors provides immediate feedback at the bedside from which nurses can effect changes in treatment.

Current and Future Research

LICOX

Several issues remain regarding the LICOX monitor. Global changes are less frequently detected with LICOX because of local placement of the catheter. However, if this monitor is placed in healthy tissue versus the injured areas, the oxygenation levels will reflect potential secondary insults to intact brain tissue. Placement in an injured area may result in overtreatment of a known ischemic area that could place surrounding healthy tissue at risk. Hyperventilation with 100% oxygen for an elevated ICP can cause a decrease in the PbtO₂ reading and this in turn may cause secondary ischemic events in viable

brain tissue (Fandino, Stocker, & Prokop, 1999). In contrast, hyperoxygenation alone drastically improves brain tissue oxygenation in the setting of a low CPP (Fandino et al.). Bardt et al. 1998 state that hypoxic episodes of less than 30 minutes defined as PbtO₂ levels <10 mm Hg with a Glasgow coma scale (GCS) of >4-5 had more favorable outcomes than those who had hypoxic episodes of greater than 30 minutes with a PbtO₂ of <10. These patients either died or had unfavorable outcomes at the time of discharge and at 6 months. Long-term outcomes such as improved neurological functioning using LICOX are relatively unknown in severe head injuries as well as other pathologies.

SjvO₂

Future research in the use of SjvO₂ is required. Despite short-term use of SjvO₂, long-term morbidity and mortality associated with this monitoring in critical care units have not been proven except when SjvO₂ is used in conjunction with hyperventilation strategies or the LICOX to prevent cerebral ischemia (Schell & Cole, 2000). New

uses for SjvO₂ monitoring include adult and pediatric cardiac surgery patients. Neurological function is important to monitor during the rewarming phase of cardiac surgery; a decrease in SjvO₂ can indicate impending ischemia. Patients undergoing an intracranial aneurysm clipping benefit from SjvO₂ monitoring by monitoring response to induced hypotension at the time of clipping, thereby minimizing hypoperfusion and hypoxia to brain tissue (Schell & Cole).

Summary

Ultimately, the prevention of secondary brain tissue ischemia is the goal. Therefore, how oxygenation and perfusion consumption are best measured continues to be debated. It is not clear whether a low PbtO₂ level within the first 24 hours of injury is indicative of decreased CBF versus ischemia or is reflecting an increased uptake of oxygen in potentially viable tissue (van den Brink et al., 2000). The lower end of the threshold is 25 mm Hg; however, new limits may be set depending on future data regarding patient outcomes and survival. As a result, multimodal monitoring over time provides information on brain tissue oxygen supply and demand measured by both the LICOX and SjvO₂ catheters.

Multimodal monitoring facilitates prevention and timely management of head injuries. Each intensive care unit (ICU) patient requires unique management because of multiple underlying medical problems, which constantly challenge practitioners as they consider treatment options. Hypoxia, hypoperfusion, and infection are frequent complications and can compound patients' ICU course from hours to weeks. Aggressive treatment to reverse these problems is necessary to prevent unnecessary secondary cerebral ischemia. Together LICOX and SjvO₂ catheters provide valuable and synergistic information to evaluate, on both a local and global level, head injury hemodynamics. Appropriate management steps may then be taken to avoid future adverse neurological outcomes.

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