

Gizmos and Gadgets for the Neuroscience Intensive Care Unit

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Abstract: Managing the critical neuroscience patient population challenges practitioners because of both the devastating injury involved and the complexity of care required. Emerging technology provides the neuroscience intensive care unit team with better information on the intricate physiology and dynamics inside the cranium. In particular, the team is better able to detect changes in pressure, oxygen, and blood flow. With improved data in hand, the team can intervene to optimize intracranial dynamics, possibly reducing disability and death among such patients.

New technologies for the neuroscience intensive care unit (NICU) provide practitioners vital information on intracranial dynamics. For years, the gold standard of monitoring has been the intracranial pressure (ICP) monitor. Although helpful in differentiating normal and abnormal pressures inside the cranial vault, ICP technology is limited because it measures just one parameter. The injured brain sustains structural and chemical derangement as well as alterations in cerebral blood flow (CBF) and oxygen delivery. Detecting these derangements and instituting interventions to normalize pressure and optimize blood and oxygen delivery are two goals for the NICU team. The use of emerging technologies may help the NICU team meet these goals and optimize outcomes in this critically ill patient population. This article identifies various technologies designed to measure ICP and brain oxygen and covers indications, normal and abnormal values, and nursing considerations involved in their use with critical NICU patients. Applications of the various technologies are illustrated by case vignettes. New technologies for measuring CBF and cerebral metabolites, which are in transition from development to clinical use, are introduced.

Intracranial Dynamics

Critically ill neuro patients may have sustained brain injury from trauma or stroke or as a consequence of another pathology. The primary injury creates chaos inside the cranial vault. Understanding the basic dynamics of the vault is essential to determining the consequences of such injuries and monitoring their impact.

The cranial vault contains three main components: The brain occupies 80% of the space, and blood and

cerebrospinal fluid (CSF) each occupy 10%. The Monroe-Kellie doctrine treats the cranial vault as a closed box; the three components exert pressure inside their closed compartment. If one of the three components increases in volume, then the other two must decrease in volume to maintain a stable pressure. A certain amount of compensation occurs inside the vault; this situation is known as compliance. For instance, when a patient sustains an acute subdural hematoma (SDH) and blood rapidly accumulates to 20% of the space occupied, the brain matter will compress and shift away from the SDH. Likewise, CSF shunts to the lumbar subarachnoid space, and venous blood normally inside the skull compensates by returning to the heart (Bader & Littlejohns, 2004). These compensatory mechanisms function to maintain the steady relationship of volume to pressure inside the skull. Once these mechanisms are exhausted, the pressure inside the cranial vault will increase, possibly decreasing the delivery of blood to the brain.

Normal blood delivery to the brain is measured in various ways. The brain receives 15%–20% of the cardiac output via CBF. Normal CBF ranges from 20 to 50 ml per 100 g of brain tissue per minute, with white-matter CBF rates around 20 ml per 100 g per min and gray-matter rates approximately 50 ml per 100 g per min (Bader & Littlejohns, 2004). The brain consumes 20% of the body's oxygen and 25% of the body's glucose and cannot store either. Therefore, it requires a constant supply of blood for its nutrients. When pressure inside the cranial vault increases beyond the normal range, the heart has more difficulty delivering blood and its nutrients to the brain.

Other mechanisms can contribute to intracranial chaos after brain injury. Secondary injury is a phenomenon in which either intracranial or extracranial events, or both, contribute to further injury to the brain. Ghajar (2000) identifies secondary brain injury as the leading cause of in-hospital deaths after traumatic brain injury (TBI). Intracranial causes of secondary brain injury include cerebral edema, cerebral blood flow abnormalities, local inflammatory changes, calcium and excitatory amino acid imbalances, vasospasm, excitotoxic injury, and apoptosis (Chesnut, 2004; Dutton & McCunn, 2003; Mulvey, Dorsch, Mudaliar, & Lang, 2004; Springborg, Frederiksen, Eskesen, & Olsen, 2005). Extracranial causes include events such as hypotension, hypoxia, hypocapnia, and hyperglycemia (Chesnut et al., 1998; Jeremitsky, Omert, Dunham, Protetch, & Rodriguez, 2003; Lam, Winn, Cullen, & Sundling, 1991; Roylias & Kotsou, 2000). Two recent studies identify only hypotension as significantly increasing mortality in TBI (Barton, Hemphill, Morabito,

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& Manley, 2005; Manley et al., 2001). If the brain sustains any of these secondary injuries, the delivery of oxygen and other nutrients will be compromised.

To reduce morbidity and mortality, the NICU team must be vigilant in its efforts to recognize and treat secondary brain injury. Recognition of these insults can be improved through the use of monitoring devices.

Technology in the NICU

Because of the complex physiology of brain injury, practitioners must explore multiple ways to monitor these patients. The monitoring devices available for use in the NICU focus on different aspects of intracranial dynamics. Each contributes data integral to choosing the best intervention to maximize outcomes. The following sections discuss devices for monitoring ICP, oxygen, CBF, and neurological chemicals.

Technology Related to ICP

Since Lundberg's landmark 1960 publication, the ICP monitor has become the standard of care for monitoring pressure inside the cranial vault. Measures that control ICP reduce the volume of the skull's contents and enhance delivery of blood to the brain. In current practice, ICP is measured by placing a device into the brain. In an emerging approach, a handheld pupillometer is used to assess pupillary responses as an indirect measure of ICP. This method is noninvasive and is being tested for its correlation to ICP. These two monitoring methods are described and contrasted.

ICP Monitor

ICP monitoring is accomplished by placing a catheter or similar device into or around the brain and connecting it to a pressure transducer. The device may be placed in the lateral ventricle, brain parenchyma, subarachnoid space, subdural space, or epidural space (Bader & Littlejohns, 2004). Ventriculostomy and intraparenchymal monitors are shown in Fig. 1. Various transducer systems exist: external strain-gauge (pneumatic; i.e., standard fluid-filled), internal-strain gauge or microchip, fiberoptic, and air-pouch (Bader & Littlejohns). The

preferred method of monitoring is the ventriculostomy, which allows practitioners to both monitor ICP and drain CSF. It is also the most accurate system (Bader & Littlejohns; Bullock et al., 2000). Less accurate and more limited is a parenchymal catheter inserted into the brain. It is easy to insert but allows only the monitoring of ICP. The other methods are used less frequently.

Patients with a decreased level of consciousness or a Glasgow Coma Scale (GCS) score between 3 and 8 are candidates for ICP monitoring (Bader & Littlejohns, 2004; Bullock et al., 2000). Diagnostic indications for ICP monitoring include TBI, aneurysmal subarachnoid hemorrhage (SAH), spontaneous intracerebral hemorrhage, ischemic stroke accompanied by significant cerebral edema, brain tumor, neuroinfectious processes, decompensated hydrocephalus, cerebral hypoxia or anoxia producing edema, or Reyes syndrome (Bader & Littlejohns; Dunn, 2002). Once the monitor is placed, the ICP is measured. The normal ICP is 0–10 mm Hg (in the literature, values up to 15 mm Hg are cited), with treatment thresholds at 20–25 mm Hg (Bader & Littlejohns; Bullock et al., 2000).

One drawback is that ICP monitoring is invasive. As an alternative, technology that is noninvasive yet still capable of sensing responses to pressure changes inside the cranial vault is being developed. One such technology, pupillometry, is described here.

Pupillometer

The Neuroptics ForeSite pupillometer (Medtronic, Minneapolis, MN) is a handheld, battery-operated device, similar in size to a portable telephone (Fig. 2). A plastic disposable headrest ensures accurate position and distance from the pupil. A microprocessor-based optical scanner captures and analyzes pupil dynamics. When the unit is held over a pupil, a 3-sec targeting phase allows the practitioner to observe a live image of the pupil in the LCD on the pupillometer face. A single 0.8-sec flash stimulus is followed by a 2.2-sec acquisition phase that concludes with a beep. During this time, 124 pictures of the pupil are recorded. During the targeting and acquisition phase the patient's eye must be open and no blinking can occur. As the images are analyzed, the pupillometer displays the maximum and minimum aperture of the pupil before and after the light stimulus, the percentage change in the pupil, and several velocities. The key velocity to be noted is the constriction velocity. This is the speed at which the pupil constricts after the light stimulus, measured in millimeters per second (Medtronic, 2002).



Fig 1. (Left) Artist's depiction of intraparenchymal ICP catheter and ventriculostomy ICP catheter; (middle) placed through bolt system; (right) tunneled.

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Fig 2. (Left) Pupillometer; (right) taking of a pupillometer reading.

Taylor et al. (2003) studied 300 healthy volunteers and 26 adult patients with TBI and ICP monitors in place. In the healthy volunteer group, 2,432 paired measurements were taken. The maximum aperture (MaxA) was 4.1 ± 0.34 mm, the minimum aperture (MinA) was 2.7 ± 0.21 mm, and the average pupil-change percentage was 34%. The average constriction velocity in the healthy group was 1.48 ± 0.33 mm per sec. When this group was compared with the TBI patients, the researchers found the TBI patients on average had smaller pupils. In the TBI group, the MaxA was 2.1 ± 0.16 mm, the MinA was 1.7 ± 0.1 mm, the constriction velocity averaged 1.18 ± 0.18 mm per sec, and the pupil-change percentage was smaller (Taylor et al.). For patients with ICP levels greater than 20 mm Hg for 15 min and a midline shift of more than 3 mm, the constriction velocities were slower on the side of the mass effect (Taylor et al.). The average constriction velocity fell to below 0.6 mm per sec in this group. In a group of five patients with diffuse brain swelling, the constriction velocities fell when the ICP was higher than 30 mm Hg. Significant findings included a trend toward ICPs greater than 20–30 mm Hg when the constriction velocities were less than 0.6 mm per sec and the pupil change was less than 10% (Taylor et al.).

Applying pupillometer technology in a clinical setting is a challenge, because limited data are available. At Mission Hospital, Mission Viejo, CA, the pupillometer has been in use for 2½ years. The NICU nurses and physicians have found a trend toward lower constriction velocities (less than 0.8 mm per sec) and smaller pupil changes (usually less than 10%) in patients with ICPs higher than 20 mm Hg. The pupillometer is an important adjunct to the clinical exam for patients with minor head injury as well as for those with an ICP monitor in place. Case vignette 1 illustrates the potential use of the pupillometer and concurrent data collected with the pupillometer and ICP monitor.

Case Vignette 1. T. B., an 18-year-old male, fell from a skateboard and sustained a brief loss of consciousness. When paramedics arrived, his GCS was 13. As he was transported to the trauma center, T. B. became agitated and combative. His GCS on arrival at the emergency department was 10. The primary survey and initial resuscitation were initiated in the trauma room. Part of the secondary survey included a pupillometer assessment. The first values obtained demonstrated that the right pupil had a MaxA of 2.6 mm, MinA of 2.1 mm, pupil change of 16%, and constriction velocity of 1.11 mm per sec. The values for the left pupil were 2.7 mm, 2.3 mm,

14%, and 0.98 mm per sec, respectively. T. B.'s computed tomography (CT) scan of the brain revealed bilateral cerebral contusions, more on the left than right. There was no midline shift. When the diagnostic tests were completed, T. B. was admitted to the ICU with orders to monitor his neurologic status closely, trend the pupillometer readings hourly, and repeat the CT scan of the brain in 4 hours. After 3 hours, T. B. became more difficult to arouse. His GCS decreased to 9. The third pupillometer check revealed a marked decrease in all values for both eyes. The reading showed for the left pupil a MaxA of 2.1 mm, MinA of 2.0 mm, pupil change of 6%, and constriction velocity of 0.49 mm per sec and, for the right pupil, 2.1 mm, 1.9 mm, 8%, and 0.8 mm per sec respectively. The repeat CT scan was moved up 1 hr and revealed blossoming of the contusions more on the left than right, a midline shift of 5 mm from left to right, and diffuse cerebral edema. T. B. was taken to the operating room where he was immediately intubated to control the airway. The neurosurgeon placed ICP and brain tissue oxygen monitors. The initial ICP reading was 25 mm Hg.

Nursing Considerations for ICP Technologies

By using different technologies to monitor ICP, practitioners can continuously assess pressure dynamics and intervene as indicated. In T. B.'s case, the pupillometer measurements supplemented the neurologic exam. The team found the falling constriction velocities and pupil change disturbing enough that they took action earlier than anticipated. The placement of a ventriculostomy ICP monitor gave the team a direct, continuous measurement of ICP, and thereafter the team's focus was to control the ICP.

Interventions to reduce ICP include first- and second-tier interventions. First-tier interventions include raising the head of the bed 30°, maintaining the neck in the

PbtO₂ >20–40 mm Hg/SjO₂ >55% ICP >20 mm Hg	PbtO₂ <20 mm Hg*/SjO₂ <55% ICP >20 mm Hg	PbtO₂ <20 mm Hg / SjO₂ <55% ICP <20 mm Hg
<ul style="list-style-type: none"> • Drain cerebrospinal fluid • Decrease PaCO₂ until ICP < 20; stop when PbtO₂ <20 • Optimize CPP for patient. Use fluids to optimize CVP/PCWP and vasopressors if CPP <60 mm Hg • Check analgesia/sedation: morphine/lorazepam drips • Give mannitol 0.25–1.0 grams/kg IV • Call physician • Begin propofol drip for refractory increased ICP • CT scan if ICP >20 mm Hg <p>Physician decision: Pentobarbital coma versus craniectomy</p>	<ul style="list-style-type: none"> • Drain cerebrospinal fluid • Place patient on 100% FiO₂ x 5–15 minutes • Optimize CPP for patient: Use fluids to optimize CVP/PCWP and packed RBCs if hematocrit <33 and PbtO₂ < 20 mm Hg. Start vasopressors if CPP < 60 • Check analgesia: Increase morphine if needed • Give mannitol 0.25–1.0 grams/kg IV • Begin propofol drip for refractory increased ICP • Call physician • CT scan if ICP >20 mm Hg <p>Physician decision: Pentobarbital coma versus craniectomy</p> <p>Temperature: Keep 36–37 °C; institute cooling measures as needed</p> <p><i>*Refer to decision tree in ICP < 20 and PbtO₂ < 20 for critical analysis of hemodynamic vs. pulmonary causes</i></p>	<ul style="list-style-type: none"> • Check PaCO₂: if low then increase to 40–45 mm Hg as long as ICP maintained within range • If still low, place patient on 100% FiO₂ x 5–15 minutes • Check lung sounds/ventilator setting and hemodynamic profile <p>Hemodynamic evaluation Check PCWP/CVP: if ↓, give fluids Check Hct: transfuse if <33% Check MAP/SVR/SV: (a) If ↓ MAP and SVR <1000, start vasopressors (b) If SVR >1400, decrease vasopressors (c) If ↓ SV abruptly, check 12-lead EKG</p> <p>Pulmonary Evaluation Consult respiratory therapist: Check ventilator settings Check need for sedation Check need for paralytic Check need for nebulizer treatments: (a) Change ventilator settings instead of high FiO₂ (b) Evaluate I:E status</p> <p>Fluid overload related to pulmonary edema/volume overload Check I/O balance Check chest X ray Consider Lasix</p> <p>Temperature: Keep 36–37 °C; use cooling measures.</p>
<p><i>Note. PbtO₂ = partial pressure of brain oxygen; SjO₂ = jugular oxygen saturation; MAP = mean arterial pressure; ICP = intracranial pressure; CPP = cerebral perfusion pressure; CVP = central venous pressure; PCWP = pulmonary capillary wedge pressure; PaCO₂ = partial pressure of carbon dioxide in arterial blood; FiO₂ = fraction inspired oxygen; ↓ = decrease; I = intake; O = output; I:E = inspiratory/expiratory ratio; Hct = hematocrit; SVR = systemic vascular resistance; SV = stroke volume; RBCs = red blood cells; IV = intravenous.</i></p> <p><i>Copyright © 2005 Mission Hospital. Used with permission.</i></p>		

Fig 3. Algorithms for management of severely brain-injured patient

neutral position, draining CSF if a ventriculostomy is in place, optimizing the partial pressure of arterial oxygen (PaO₂) to higher than 60 mm Hg and partial pressure of carbon dioxide (PaCO₂) to 35–45 mm Hg, maintaining cerebral perfusion pressure (CPP) at least 60 mm Hg (calculated by taking the mean arterial blood pressure [MAP] minus the ICP), administering sedation and analgesic medications to reduce anxiety and pain, administering osmotic diuretics or hypertonic saline, and controlling environmental stimulation (Bader & Littlejohns, 2004; Chesnut, 2004). Second-tier interventions include medications such as propofol (for ICP control) or pentobarbital (to induce barbiturate coma) and hemicraniectomy (Bader & Littlejohns; Chesnut). An algorithm identifying first- and second-tier interventions can give the team direction and help them prioritize care. An example of such an algorithm is given in Fig. 3.

Case vignette 1, Part 2. T. B. returned to the ICU intubated on a ventilator with an ICP monitor, brain tis-

sue oxygen monitor, and pulmonary artery catheter in place. The pulmonary artery catheter helped the team measure appropriate fluid volume levels, which guided the titration of vasopressors to support CPP. The team goals were to keep T. B.'s ICP less than 20 mm Hg while maintaining normal brain oxygen levels. His orders included maintaining PaCO₂ at 35–45 mm Hg and CPP greater than 60 mm Hg with fluids and vasopressors, draining CSF for an ICP higher than 20 mm Hg, initiating an intravenous (IV) lorazepam drip at 3 mg per hour and an IV morphine sulfate drip at 5 mg per hour, and using mannitol as needed for refractory increased ICP. During the first 4 hours after surgery, T. B.'s ICP did not decrease below 20 mm Hg, and all first-tier interventions were exhausted. Second-tier therapy was initiated with pentobarbital. T. B. received a loading dose of 10 mg per kg over 30 minutes, followed by three doses of 5 mg per kg over 60 minutes each. Following the loading doses, he was maintained on a 1–3 mg per kg per hour drip,

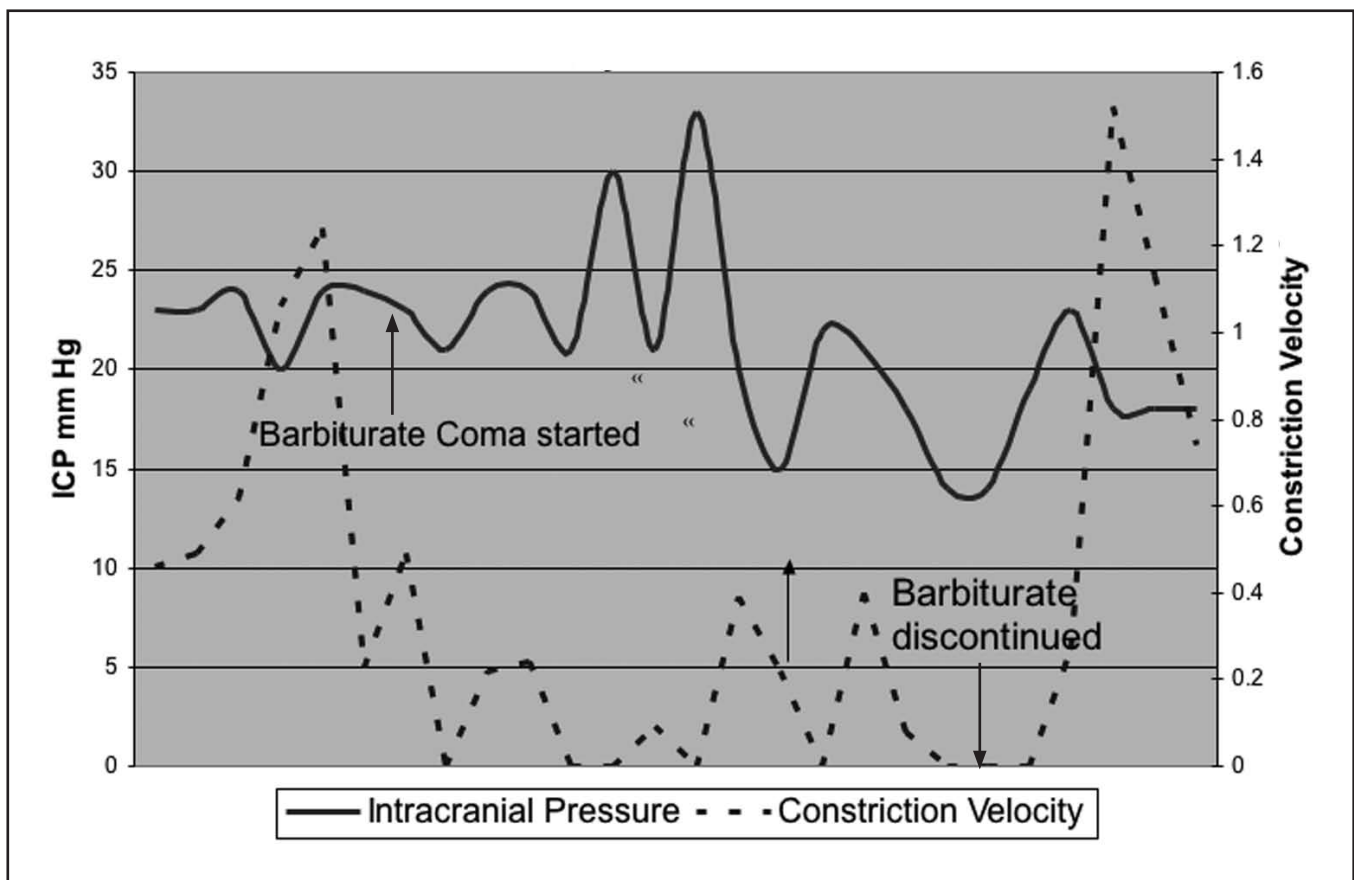


Fig 4. Intracranial pressure and constriction velocity in Patient T. B.

achieving burst suppression on the electroencephalogram (EEG) and control of elevated ICP. The constriction velocities decreased as the barbiturate-induced coma ensued, with loss of pupillary reaction during this time (Fig. 4). The ICP was maintained at less than 20 mm Hg for several days. The barbiturate coma was discontinued on day 5. The constriction velocities recovered within 12 hours after the barbiturates were turned off.

T. B.'s case illustrates first- and second-tier interventions based on ICP and provides data on the relationship between pupil response and ICP. The NICU team employed multiple interventions in an effort to reduce ICP, which was only one of several parameters used to manage the intracranial dynamics. The team was challenged on days 1–7 not only with ICP control but also with maintaining adequate brain oxygen levels. Despite ICP reduction, the team discovered the brain oxygen fluctuated to critically low levels during the intensive management phase. The team realized that they had to focus on multiple parameters to achieve the right balance in the intracranial vault. Managing the cerebral oxygenation was a key factor in the plan to maximize T. B.'s outcome.

Technology Related to Brain Oxygen

The delivery of oxygen is critical to the survival of the brain. When caring for a critically ill neuro patient, the team must be aware of the impact that various interven-

tions have on blood flow and brain oxygen. To intervene appropriately, practitioners must be able to relate the dynamics of CBF to oxygen delivery in the brain.

Oxygen and CBF Dynamics

Oxygen is picked up in the lungs and carried in the blood mostly bound to hemoglobin (SaO_2) with a small percentage dissolved in the plasma of arterial blood (PaO_2). Many factors influence blood flow and oxygen delivery to the brain, including CBF, CPP, and cerebrovascular resistance (CVR). CBF is calculated by dividing CPP by CVR. CPP equals the MAP minus the ICP. Optimal CPP has a physiologic basis, but the rationale and theories vary (Robertson, 2001). The Brain Trauma Foundation's *Guidelines for the Management of Severe Head Injury* suggests a minimum threshold of CPP at approximately 60 mm Hg (Bullock et al., 2003). It is important to remember that maintaining a normal CPP does not ensure that CBF is adequate to meet the metabolic needs of the brain, because many variables can affect CBF (Robertson, 2001).

CPP thresholds vary from patient to patient, depending on whether autoregulation is intact or disrupted (Robertson, 2001; Vespa, 2003). Autoregulation, a major factor in the regulation of CBF, is influenced by vasomotor factors, flow-metabolism mechanisms, and metabolic substances (Bader & Littlejohns, 2004).

Vasomotor control of CBF includes myogenic and

adrenergic influences. Given intact autoregulation, CBF will be maintained in a constant flow at pressures between 50 and 150 mm Hg through changes in the diameter of the blood vessels. When blood pressure (BP) increases, the brain arteries vasoconstrict, increasing the resistance of the cerebrovasculature. This change maintains a constant CBF. As BP drops, the arteries vasodilate, and resistance in the cerebrovasculature decreases, once again maintaining CBF (Bader & Littlejohns, 2004).

Flow metabolism is the relationship between CBF and changes in metabolism. As metabolism in the brain increases, CBF increases. When metabolism decreases, CBF decreases. The metabolic factors that greatly influence CBF are carbon dioxide (CO₂), oxygen (O₂), and pH. Carbon dioxide is a powerful vasoconstrictor or vasodilator of cerebral arteries. When the percentage of CO₂ dissolved in the plasma of arterial blood (PaCO₂) decreases below 35 mm Hg, the cerebral arteries vasoconstrict. For every decline of 1 mm Hg in PaCO₂, there is a 2%–3% reduction in CBF. If PaCO₂ increases, the cerebral arteries vasodilate, causing an increase in CBF. Oxygen in the normal range has minimal impact on the cerebral arteries. When PaO₂ is less than 50 mm Hg, the arteries vasodilate, increasing CBF. As for pH, in the presence of acidosis, the arteries dilate; when alkalosis occurs, the arteries constrict (Bader & Littlejohns, 2004). Once these factors are understood, CBF and oxygen delivery can be manipulated.

Oxygenation in the brain can be measured several ways. The following section discusses three monitoring technologies that have been studied and used in the management of neuro patients: noninvasive transcutaneous cerebral oximeters, intravascular jugular bulb venous oxygen saturation catheters, and implanted brain tissue oxygen catheters.

Noninvasive Transcutaneous Cerebral Oximetrics

Brain oxygenation can be measured indirectly by using near-infrared spectroscopy. One such monitoring system is called INVOS (In-Vitro Optical Spectroscopy; Somanetics Corporation, 2001). A disposable transducer probe is applied to the forehead, and the monitor attached to the probe detects wavelengths of light at distances 3–4 cm from the source. The transducer converts the wavelengths to a measurement that represents regional oxygen saturation, which is known as the rSO₂ index (Somanetics Corporation, 2001). The rSO₂ index is the oxygen saturation of a mixture of arterial and venous blood in the brain parenchyma. By applying sensors to both sides of the forehead, practitioners can measure the rSO₂ in both frontal hemispheres.

The INVOS unit can be used in adult and pediatric patients with age-appropriate sensors. Normal rSO₂ has not been validated in studies. One study cites an optimal measurement of approximately 75% in cardiac surgery patients (Murkin, Adams, Schaefer, Irwin and Fox, 2004). Papadimos and Marco (2004) cite a 20% decrease in rSO₂ as being significant with a minimal acceptable

reading of 45% in cardiac surgery patients. Monitoring of rSO₂ has been studied in patients undergoing carotid endarterectomy, TBI, SAH, stroke, cardiac arrest, and cardiac surgery (Bar-Yosef, Sanders, & Grocott, 2003; Brawanski, Faltermeier, Rothoerl, & Woertgen, 2002; Dunham, Ransom, Flowers, Siegal, & Kohli, 2004; Dunham, Sosnowski, Porter, Siegal, & Kohli, 2002; Edmonds, Granzel, & Austin, 2004; Mille et al., 2004; Murkin et al., 2004; Nemoto, Yonas, & Kassam, 2000). In the cardiac and carotid endarterectomy patient populations, the device has been helpful in determining episodes of low perfusion, as evidenced by a decrease in rSO₂ (Murkin et al., 2004; Edmonds et al., 2004). Murkin et al. found that maintaining rSO₂ above 75% reduced the number of strokes after cardiac bypass surgery. One citation maintains that a level below 50% may be correlated with poorer neurologic outcomes in cardiac bypass patients (Somanetics Corporation, 2003).

In the neuro population, a pilot study found a correlation between CPP levels and rSO₂ levels (Dunham et al., 2002). There was a trend toward lower rSO₂ levels as CPP decreased. Dunham et al. (2004) also found a correlation between lower rSO₂ levels and lower GCS scores on admission. Other work has explored the use of the noninvasive cerebral oximeter in SAH patients, but no large studies have been published. The reliability and accuracy of the system has yet to be determined. Research into the use of noninvasive cerebral oximetry in the neuro population is ongoing.

Intravascular Jugular Bulb Venous Oxygen Saturation

Another method of measuring cerebral oxygenation, jugular venous oxygen saturation (SjO₂) monitoring, has been used since the 1980s. Considerable research has been conducted with the SjO₂ monitoring system. Monitoring of SjO₂ involves use of a fiber-optic catheter, typically 5.5 french in size, placed retrograde in one of the internal jugular (IJ) veins (Fig. 5). It is tunneled up the IJ vein against blood flow toward the brain. The catheter is placed so the tip of the catheter is level with the mastoid air cells or approximately at the jugular bulb of the IJ vein (Coplin et al., 1998; Feldman & Robertson, 1997). The fiber-optic catheter measures the oxygen saturation of hemoglobin as it exits the cerebral circulation in the IJ vein and provides a continuous reading of venous oxygen saturation.

Monitoring of SjO₂ is indicated for neuro patients with GCS scores less than or equal to 8 (Cruz, 1998; Feldman & Robertson, 1997; Gopinath, Valadka, Uzura, and Robertson, 1999; Robertson et al., 1995). Nemani & Manley (2004) noted that the SjO₂ monitoring system measures only global oxygenation in the brain. The system does require a high level of maintenance; practitioners must ensure that the neck stays midline, the catheter remains free from clots, and the system is frequently recalibrated.

The SjO₂ monitoring approach has been fairly well studied. The normal range of SjO₂ confirmed in a study

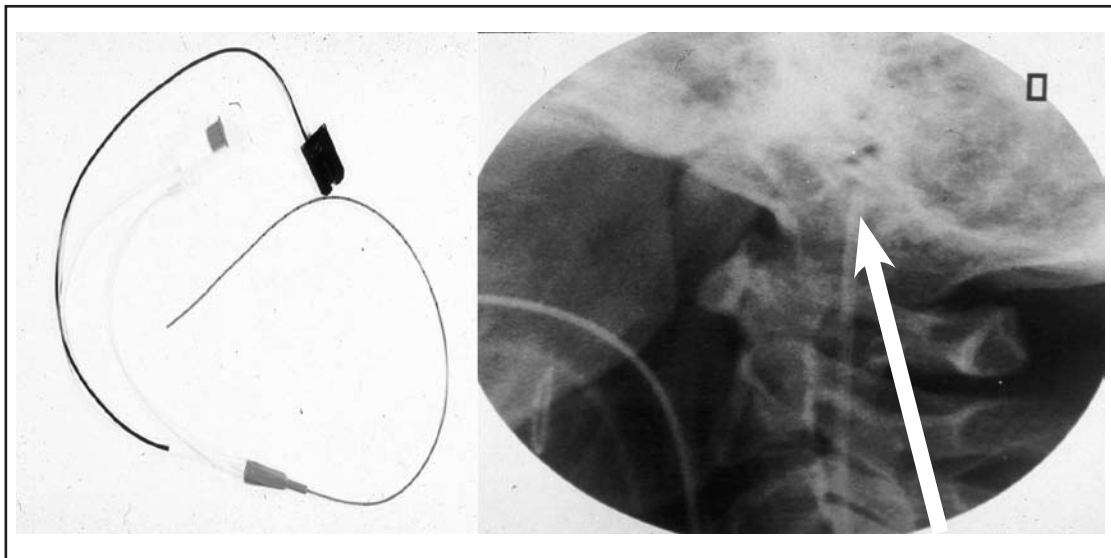


Fig 5. (Left) Jugular bulb 5.5 french oxygen saturation fiber-optic catheter; (right) correct placement of the catheter, shown on a lateral skull X ray, with the tip of the catheter in the jugular bulb

of healthy young men, is 55%–71% (Feldman & Robertson, 1997). Most users establish the normal SjO_2 range at 55%–75%. Lower SjO_2 is a cause for concern; a value below 55% reflects a state of ischemia in which oxygen delivery is compromised or oxygen consumption exceeds the supply of oxygen delivered to the brain. The low state must be treated promptly to avoid poor outcome (Nemani & Manley, 2004). The ischemic threshold has been reported to be an SjO_2 of less than 50% for at least 10 minutes, with multiple episodes contributing to poor outcome (Robertson et al., 1995). A group of patients managed by targeting optimal SjO_2 levels and CPP enhancement had better outcomes than a group of patients managed with CPP-targeted therapy alone (Cruz, 1998).

The SjO_2 monitoring system provides a global assessment of brain oxygen. Integrating this parameter into the nursing care of the complex brain-injured patient allows the team to differentiate care priorities independent of increased ICP.

Nursing Considerations Related to SjO_2

Monitoring of SjO_2 has been valuable in helping practitioners target appropriate treatment strategies in brain injury to minimize ischemic episodes in the brain (Palmer et al., 2001). Strategies to maintain SjO_2 in the normal range or raise a low SjO_2 include increasing the delivery of inspired oxygen; increasing $PaCO_2$ levels; administering packed red blood cells; raising CPP; reducing ICP by draining CSF, administering mannitol, and providing adequate sedation; and administering fluids for vasospasm (Bader & Palmer, 2000).

Case vignette 2 illustrates the application of SjO_2 monitoring. Maintenance of the catheter system is essential to obtaining meaningful data. The SjO_2 catheter should be connected to a heparin saline flush similar to other hemodynamic lines to maintain patency. Samples of venous blood must be obtained every 12–24

hours and sent to the respiratory lab to calibrate the fiber-optic catheter. The neck needs to be straight to prevent the catheter from lying on the side of the IJ vein. Light intensity from the fiber-optic catheter displayed on the monitor is diminished when the catheter is kinked or a clot forms on the end of the catheter. Care

should be taken to reduce or prevent infection.

Case vignette 2. K. A., a 47-year-old female, was critically injured following a traffic collision in which the point of impact was on her side of the car. She was transported to the trauma center by prehospital personnel. On admission to the emergency department, her GCS was 1-2-1 (4); pupils were 4 and nonreactive bilaterally; and vital signs were as follows: BP 140 over 100 mm Hg, pulse 98 beats per minute, and respirations assisted. K. A. was intubated, and intravenous lines, a urinary catheter, and oral gastric tubes were placed. A CT scan done 20 min after arrival revealed a right SDH, cerebral contusions in the right temporal and parietal region, SAH, and left basilar skull fracture. Her chest X ray showed a second-rib fracture and bilateral pneumothoraces. K. A. was taken to the operating room for evacuation of the SDH, insertion of bilateral chest tubes, and placement of ICP, SjO_2 , and pulmonary artery catheters. On admission to the NICU, the surgeon ordered ICP to be kept below 20 mm Hg and SjO_2 to be kept higher than 55%.

K. A. was managed by the NICU team (Table 1). On day 1, ICP increased to above 20 mm Hg with an adequate SjO_2 of 74%. The team increased the ventilator rate, causing the $PaCO_2$ to decrease. This action also lowered ICP and SjO_2 , but both variables were within the zone of control set by the neurosurgeon. On day 2, the ICP increased again above 20 mm Hg, causing the team to explore interventions to lower the ICP. The first intervention was to lower the $PaCO_2$, which causes the ICP and SjO_2 to decrease, but the ICP was still above 20 mm Hg. Mannitol was given, CSF was drained, and the vasopressor was increased to raise the MAP. These interventions lowered the ICP below 20 mm Hg and ensured an adequate SjO_2 . K. A.'s ICP increased to 30 mm Hg on day 4. Her pulmonary status challenged the team

because of her lung injuries. She was placed on 100% oxygen and inverse inspiratory per expiratory ratio ventilation. The team gave K. A. albumin and increased the phenylphrine (neosynephrine) drip. The neurosurgeon ordered the initiation of a pentobarbital drip to help lower the ICP. With these interventions, K. A. was brought back into the zone of control. The barbiturate coma was continued through day 8.

On day 7, K. A. sustained a crisis. The team was unable to maintain an adequate CPP. As the CPP dropped, SjO₂ decreased to 30% despite an ICP in the normal range. Interventions were employed to raise SjO₂, PaCO₂ and FIO₂ were increased, and all five vasopressors were maximized. A check of arterial blood gas revealed acidosis, so K. A. received sodium bicarbonate. SjO₂ returned to the normal range, and CPP rose to an optimal level once again. Once SjO₂ was in the normal range, FIO₂ was decreased.

K. A. stabilized by the end of day 8. The barbiturate

coma was withdrawn. The vasopressors were tapered off by day 14. The team's interventions were successful in getting K. A. through a very difficult period.

Brain Tissue Oxygen Monitoring

Whereas SjO₂ is a reflection of global cerebral oxygen, another form of brain oxygen monitoring measures oxygen in a regional method. In this method, a small 0.8-mm probe placed into the white matter of the brain parenchyma provides a direct measurement of the partial pressure of brain tissue oxygen (PbtO₂). This method has been in use in Europe since the 1990s and in research trials in the United States during the same period. Two types of monitoring systems are currently approved for use in the United States. Both systems use small catheters placed into the brain to measure brain tissue oxygen regionally and are based on similar Clark-type electrode technology. However, they differ in the actual

Table 1. Neuroscience Intensive Care Unit Management of Patient K. A.

Day 1. ICP increase at 1800: CO₂ decreased and CSF drained to reduce ICP

Time	ET CO ₂	MAP	ICP	CPP	SjO ₂	Interventions
1600	34	108	18	90	72	Drain CSF; family at bedside
1800	35	113	26	87	74	Drain CSF; increase ventilator rate
1900	30	107	19	88	65	Maintain parameters

Day 2. CO₂ decreased to lower ICP; drop in ICP triggers mannitol administration

Time	ET CO ₂	MAP	ICP	CPP	SjO ₂	Interventions
1400	35	92	26	66	68	Drain CSF, increase rate; family at bedside
1430	30	88	22	67	58	Administer mannitol; drain CSF; increase phenylphrine
1500	31	95	15	80	64	In the zone of control

Day 4. MAP increased to decrease ICP

Time	ET CO ₂	MAP	ICP	CPP	SjO ₂	Interventions
2300	35	76	30	46	53	Increase FIO ₂ to 100% due to pulmonary issues; 250 ml albumin to increase wedge pressure to 12 mm Hg; increase phenylphrine drip; initiate barbiturate coma
0030	35	85	17	68	92	Vasopressors maximum; family at bedside

Day 7: Neuro crisis: SjO₂ drops to ischemic levels; MAP, CO₂, and FiO₂ increased to raise SjO₂

Time	ET CO ₂	MAP	ICP	CPP	SjO ₂	Interventions
1900	35	64	19	44	44	Increase vasopressor to increase MAP; decrease ventilator rate; norepinephrine and dopamine at maximum dose
1920	40	58	19	38	40	Increase phenylphrine to 300 mcg per minute
1940	41	46	19	27	30	Place on 100% FIO ₂ ; patient acidotic: sodium bicarbonate given
2000	41	77	14	63	55	Fluid bolus
2030	40	96	16	80	59	Decrease FIO ₂ to 80%

Note. ICP = intracranial pressure; ET CO₂ = end-tidal carbon dioxide; MAP = mean arterial pressure; CPP = cerebral perfusion pressure; SjO₂ = jugular oxygen saturation; CSF = cerebrospinal fluid; FiO₂ = fraction inspired oxygen

oxygen-sensing length on the catheter, which changes the threshold values of measured tissue oxygen.

The Paratrend/Neurotrend system (Codman; Raynam, MA) catheter measures three variables: oxygen, pH, and CO₂. Originally designed as an intravascular catheter, it has been adapted for use in the brain. The actual sensing area that monitors the oxygen is 1.4 mm long (Nemani & Manley, 2004).

The LICOX system (Integra Neurosciences; Plainsboro, NJ) measures two variables on its catheter: oxygen and temperature. The oxygen-sensing area on the latest catheter, called the PMO catheter, is 7 mm long and is located at the distal tip of the catheter; the temperature-sensing area is located on a more proximal area of the catheter (Integra Neurosciences, 2004).

The indications for placing a PbtO₂ catheter are identical to those for patients requiring ICP or SjO₂ monitoring. Patients sustaining TBI, SAH, stroke with mass effect, or any pathology that results in a GCS less than or equal to 8 are candidates for this type of monitoring.

Optimal placement of the catheter by the neurosurgeon is generally 25–35 mm into the brain (Littlejohns, Bader, and March, 2003). The oxygen-sensing area should be in the white matter of the brain, because it is considered more metabolically stable than the gray matter. Because the catheter measures regional oxygenation, the neurosurgeon must decide into which side and region of the brain to place the catheter. If the physician is interested in monitoring the injured side of the brain, the catheter is placed near the injury or penumbra. So placed, it will function as a regional measure of brain oxygen. If the catheter lies within a contusion or clot, its measured value will be quite low and will fail to signal the patient's response to interventions. If the physician is interested in monitoring the side opposite the injury, the values may be less region specific. According to Nemani and Manley (2004), placing the catheter in the contralateral hemisphere in a normal brain produces values more reflective of global brain oxygen even though it is still a regional measurement.

In both systems, the PbtO₂ values measure millimeters of mercury; however, the normal and abnormal thresholds are different. Nemani and Manley (2004) point out that true "normal" values have never been determined, because the catheters have not been placed in healthy volunteers; the values obtained have been in critically ill neuro patients. The Paratrend/Neurotrend normal values are probably higher than 35 mm Hg and higher than the LICOX system values. (Doppenberg, Zauner, Watson, and Bullock, 1998; Nemani and Manley; Zauner, Doppenberg, Young, & Bullock, 1996). Studies using the LICOX catheter have found normal levels in critically ill neuro patients ranging between 20 and 40 mm Hg (Hlatky, Valadka, and Roberston, 2003; Maas, Fleckenstein, de Jong, & van Santbrink, 1993; Sarrafzadeh et al., 1998;). Critical threshold levels where poorer outcomes have been identified differ between the two systems. In two studies using the

Paratrend system, vegetative state or death resulted when PbtO₂ was less than 25 mm Hg or 31 mm Hg, respectively (Doppenberg et al., 1998; Zauner et al., 1996). In studies using the LICOX system, critical thresholds where poor outcomes occurred varied. The PbtO₂ threshold levels identified were below 15 mm Hg, below 10 mm Hg, and any occurrence below 6 mm Hg (van den Brink et al., 2000; Bardt et al., 1998; Valadka, Gopinath, Contant, Uzura, & Robertson, 1998). The length of time under the particular threshold measured also influenced poor outcomes.

Once placed, the PbtO₂ monitor will provide essential data to help the team make decisions. Nursing care focuses on keeping the oxygen in the normal zone while balancing all of the other variables.

Nursing Considerations Related to PbtO₂ Technology

Setup and calibration vary, depending on the type of brain oxygen monitor used. The Neurotrend monitor has an extensive calibration process that requires as much as 30 min to complete. The LICOX monitor requires minimal setup (i.e., placement of a card into the front slot of the monitor).

Many factors influence brain tissue oxygen (Bader, Littlejohns, & March, 2003). Low pulmonary variables, such as low PaO₂ or PaCO₂, cause a decrease in oxygen available to the brain. Low hemodynamic variables, such as a decrease in CPP or hemoglobin concentration, lower PbtO₂. An elevated ICP reduces CBF and oxygen delivery to the brain (Littlejohns et al., 2003). The NICU team must know how to detect low PbtO₂ values, determine the probable cause, and intervene with appropriate measures to optimize oxygen delivery in the brain.

Interventions to raise a low PbtO₂ focus on countering the cause. If the PaO₂ is low, increasing the FIO₂ will increase the supply of oxygen. If the PaCO₂ is less than 35 mm Hg, raising it might raise the PbtO₂. If the patient has pulmonary issues and lung compliance has decreased, administering a neuromuscular blocker may improve the ability to oxygenate and ventilate the patient. Achieving optimal CPP and using strategies to reduce ICP will enhance brain oxygenation. Inducing a barbiturate coma helps decrease ICP and brain metabolism. Choosing the correct intervention may assist the team in optimizing oxygen delivery and consumption. Returning to T. B.'s case illustrates the use of PbtO₂ monitoring.

Case vignette 1, Part 3. The team struggled to keep T. B.'s ICP under control. Primary- and secondary-tier interventions were employed to manage ICP. On day 1, the team began by decreasing PaCO₂ to reduce ICP, but this caused PbtO₂ to drop to 16 mm Hg and ICP to increase to 29 mm Hg (Table 2). Mannitol was given, and CO₂ was increased to reduce ICP and increase PbtO₂.

The team was challenged on day 3 when a pulmonary crisis ensued. PaO₂ decreased to 66 mm Hg, which caused PbtO₂ to decrease to 17.4 mm Hg. A bronchoscope done to remove secretions from the lungs caused ICP to rise above 30 mm Hg. Intravenous sedation, analgesia,

and lidocaine were given. At the conclusion of the bronchoscopy, T. B. received a nebulizer treatment and was given an IV neuromuscular blocker. This intervention improved his pulmonary compliance and his PbtO₂.

T. B.'s ICP issues resolved by day 7, but his pulmonary problems returned. He experienced an acute drop in his PbtO₂ to 12.7 mm Hg. To raise the PbtO₂, the ventilator rate was reduced and the CO₂ was increased. T. B. was temporarily put on 100% FiO₂. Thirty minutes later his PbtO₂ was 18 mm Hg. After checking the hematocrit and noting that it was below 33 mg %, the team called the neurosurgeon, who ordered a unit of packed red blood cells. After the transfusion, T. B.'s PbtO₂ increased to 23 mm Hg. Four hours later, T. B. sustained another acute drop in PbtO₂, to 63 mm Hg. The intensivist noted that T. B.'s lungs were nearly whited out on his chest X ray, indicating adult respiratory distress syndrome. The NICU nurse suggested placing the patient in the prone position to open the closed alveoli. This was done, and PbtO₂ and PaO₂ improved dramatically. The proning regimen (6 hr prone followed by 6 hr supine) continued for 48 hours.

T. B. improved steadily over the next 14 days. His ICP and PbtO₂ catheters were removed on day 10. He was

extubated on day 16. He emerged from his coma and went to acute rehabilitation on day 24. He returned to school 4 months after his injury.

The multiple parameters the team used to manage T. B.'s care included interventions to raise PbtO₂, but sometimes this strategy increased the ICP. When this occurred, the team was challenged to intervene again to find the balance in his care.

Bedside CBF Monitoring

In the past few years, new technology for bedside CBF monitoring has been examined for use in the NICU. For the past two decades, CBF has been measured by using radioactive tracers such as xenon. This CBF technology requires the patient to be moved to a large machine, where injection or inhalation of xenon enables practitioners to measure global CBF at that specific moment (Springborg et al., 2005). This technology permits measurement (i.e., intermittent assessment of the parameter) but not monitoring (i.e., constant observation of a parameter; Steiner & Czonsnyka, 2002). Another limiting factor is that xenon is only available in research centers.

The ultimate goal is to develop CBF technology that

Table 2. Cerebral Hemodynamic Profile of Patient T. B.

Day 1. Increased ICP: First- and second-tier interventions					
ET CO ₂	MAP	ICP	CPP	PbtO ₂	Interventions
35	90	25	65	26.9	Drain CSF; increase ventilator rate
31	85	21	64	22	Drain CSF; increase phenylphrine drip
31	90	29	61	16	Mannitol given; decrease ventilator rate
34	89	23	66	20.2	Initiate barbiturate coma: bolus/drip
Day 3. Pulmonary crisis					
ET CO ₂	MAP	ICP	CPP	PbtO ₂	Interventions
33	92	20	72	28.4	FiO ₂ at 100% due to lungs; increase phenylphrine drip
32	85	26	59	17.4	Problem ventilating; PaO ₂ = 66 mm Hg
34	84	32	52	17	Bronchoscopy in progress; premedicated with IV lidocaine, morphine sulfate, and lorazepam
34	90	28	64	19.9	Albuterol treatment; give neuromuscular blocker
36	90	18	72	26	In the zone of control
Day 7. Pulmonary crisis					
ET CO ₂	MAP	ICP	CPP	PbtO ₂	Interventions
35	86	14	72	12.7	Increase FiO ₂ from 75% to 100%; decreased rate
41	77	17	60	18	Hematocrit 29; give one unit packed red blood cells
41	85	14	71	23	Four hours go by
45	75	17	68	12.6	PaO ₂ 63; give nebulizer treatment; patient in prone position for 6 hr
42	70	15	55	19	PaO ₂ 80 1 hr later
38	72	15	67	25.5	Prone position for total of 6 hr; PaO ₂ 152

Note. ICP = intracranial pressure; ET CO₂ = end-tidal carbon dioxide; MAP = mean arterial pressure; CPP = cerebral perfusion pressure; PbtO₂ = brain tissue oxygen; CSF = cerebrospinal fluid; FiO₂ = fraction inspired oxygen; IV = intravenous; PaO₂ = partial pressure of arterial oxygen

operates continuously at the bedside. In recent years, two types of bedside CBF monitoring have been developed and marketed. This section does not discuss transcranial Dopplers (TCDs), which some may be considered an indirect method of assessing CBF. Technically, the TCD measures blood flow velocity in certain cerebral arteries, not CBF (Steiner & Czosnyka, 2002). The TCD technology is not within the scope of this article. Each technology measures CBF differently.

The first bedside CBF monitor released for clinical use in the United States was the Anspach CBF monitor (Hemadex; Hilliard, OH). CBF is measured by using a microprobe that is inserted into the brain parenchyma, typically into white matter. This type of CBF measurement is known as thermal diffusion flowmetry (TDF). The probe is a thermodilution probe with two thermister sensors located 5 mm apart near the tip of the catheter (Vajkoczy et al., 2000). Once implanted into the brain and connected to the machine, the proximal thermister measures the temperature in the brain. The distal thermister is heated up to 2 °C higher than the patient's measured temperature (Steiner & Czosnyka, 2002). The monitoring system uses a series of data-reduction algorithms that factor in quantification of tissue perfusion using conductive properties of the tissue (Vajkoczy et al., 2000). The result is a measurement of local CBF in milliliters per 100 g of brain tissue per minute. The accuracy and reliability of this system have been challenged (Springborg et al., 2005). Another cited limitation is that the system measures CBF in only one small area of the brain, although differences in global and regional CBF often occur (Steiner & Czosnyka, 2002).

The second bedside CBF monitor introduced in the United States was the Novus Neurosensor (Integra Neurosciences; Plainsboro NJ). This CBF monitoring system uses a combination ICP per CBF probe that is based on laser Doppler flowmetry (LDF) technology. Like the Anspach bedside CBF monitor, the Novus Neurosensor probe must be inserted into the brain parenchyma through an introducer. The LDF measures regional CBF (Integra Neurosciences, 2005) and is considered to have excellent dynamic resolution and to be fairly reliable for local cortical non-directional CBF (Springborg et al.; Vajkoczy et al.). Some challenges for this system are that the patient must remain immobile to limit interference with data acquisition, artifacts are created by a number of external causes, and the reliability of data depends on the probe position (Springborg et al., 2005; Vajkoczy et al., 2000). The area of measurement is only 1–2 mm², so the ability to generalize to CBF rates for the entire brain is limited (Steiner & Czosnyka, 2002). The small measurement area is the primary limitation of this promising new technology.

At Mission Hospital, the TDF-type CBF monitor has been used occasionally for the past 2 years in TBI and aneurysmal SAH patients. The initial challenge centered on inserting the catheter far enough into the brain, because it does not come with its own introducer. Once inserted, the monitor was fairly reliable. In the aneurysmal

SAH population, the CBF recordings revealed dramatic falls in CBF after nimodipine administration, with MAP decreases, and when severe vasospasm occurred. The simultaneous use of PbtO₂ monitoring gave the team insight into the relationship between CBF and PbtO₂. In the SAH population, decreases in CBF corresponded with decreases in PbtO₂. In the TBI population, when both CBF and PbtO₂ probes were used, the two variables had similar increases and decreases—some of the time. In other instances, the data gathered showed normal CBF with low PbtO₂ and low CBF with normal PbtO₂. This discrepancy has led the team to question the usefulness of the CBF probe in the TBI patient population, especially if CBF is the only parameter to be measured.

Further research is needed to improve the measuring capabilities of bedside CBF monitoring. As the technology improves, the ability to quantify CBF and incorporate this parameter into clinical decision making at the bedside is likely to be enhanced.

Cerebral Microdialysis for Neurochemicals

Another technology that has been studied for its usefulness in the NICU is a cerebral microdialysis system. Researchers are exploring the use of cerebral microdialysis technology as a means to measure extracellular levels of cerebral metabolites. When the brain suffers ischemic episodes, changes are produced in energy metabolites, such as glucose, pyruvate, lactate, the excitatory amino acids glutamate and aspartate, and membrane-degrading substances such as glycerol (Sarrafzadeh et al., 2002; Springborg et al., 2005). Through a tiny microcatheter inserted into the brain parenchyma, a small pump delivers a 0.3-ml per min flow of dialysate fluid into the brain. After passing through a semipermeable membrane, the dialysate fluid is pumped into a small vial and then analyzed. The substances measured include glucose, lactate, and pyruvate from the extracellular fluid (CMA Microdialysis, 2003). The dialysate fluid is recovered at a set time, generally every 60 min, and then processed through an analyzer that reports the levels of neurochemicals. This technology is approved for use in the United States but few centers have the capabilities or personnel to interpret and use the data. It has been studied in TBI, SAH, epilepsy, and stroke (Hillered & Persson, 2003; Sarrafzadeh et al., 2002). It does not produce continuous readings; data are obtained intermittently, only as often as sampling is scheduled.

Sarrafzadeh et al. (2002) reported that the bedside microdialysis system was safe and could detect changes in the chemicals indicative of early ischemia. Hillered & Persson (2003) were advocates for the use of microdialysis in certain centers in conjunction with other monitoring systems, such as the ICP, S_jO₂, PbtO₂, and CBF monitors and continuous EEG, to detect ischemia in the brain.

Normal levels of cerebral chemicals include a lactate-to-pyruvate ratio of 15–20, a glutamate level of 10 mcm, and a glycerol level of 50–100 mcm with a 10-mm dialysis

membrane (Ungerstedt & Rostami, 2004). Certain neurochemicals change when there is damage to brain cells or when cerebral ischemia is present, or both. One marker of ischemia is a lactate-to-pyruvate ratio greater than 25 (Ungerstedt & Rostami). Target glutamate and glycerol levels are more difficult to quantify because of other factors. Springborg et al. (2005) cited a study in SAH patients that found increasing concentrations of glutamate as an early sign of vasospasm. Later, elevations of lactate, lactate-to-pyruvate ratio, and glycerol were found as indexes of ischemia (Springborg et al., 2005). In a study of 44 patients with aneurysmal SAH, researchers found a correlation between the development of acute focal neurological deficits and increases in lactate, glutamate, lactate-to-pyruvate ratio, and glycerol (Sarrafzadeh et al., 2003).

Microdialysis is a promising technology, but widespread use has not occurred. More research is needed to explore the uses of this technology in the NICU.

Summary

The care provided to critical neuro patients can be enhanced by integrating technology into the clinical decision making that occurs at the bedside. Current monitoring systems capable of measuring pressure and oxygen contribute significant data to be factored in along with clinical and diagnostic information. Further development and improvement of CBF and micro-dialysis technologies might provide practitioners with a more comprehensive view of the intracranial chaos produced in brain injury.

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