Traumatic brain injury (TBI) accounts for 1.4 million reported injuries and 52,000 deaths annually in the United States. Intensive bedside neuromonitoring is critical in preventing secondary ischemic and hypoxic injury common to patients with traumatic brain injury in the days following trauma. Advancements in multimodal neuromonitoring have allowed the evaluation of changes in markers of brain metabolism (eg, glucose, lactate, pyruvate, and glycerol) and other physiological parameters such as intracranial pressure, cerebral perfusion pressure, cerebral blood flow, partial pressure of oxygen in brain tissue, blood pressure, and brain temperature. This article highlights the use of multimodal monitoring in the intensive care unit at a level I trauma center in the Pacific Northwest. The trends in and significance of metabolic, physiological, and hemodynamic factors in traumatic brain injury are reviewed, the technical aspects of the specific equipment used to monitor these parameters are described, and how multimodal monitoring may guide therapy is demonstrated. As a clinical practice, multimodal neuromonitoring shows great promise in improving bedside therapy in patients with traumatic brain injury, ultimately leading to improved neurological outcomes. (Critical Care Nurse. 2011;31:25-37)

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This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Describe the interrelationships among intracranial pressure, cerebral perfusion pressure, brain tissue partial pressure of oxygen, blood pressure, and brain temperature
2. Identify aberrations in cerebral metabolites indicative of cerebral ischemia
3. Discuss common neuromonitoring parameters and the threshold values and appropriate nursing interventions associated with each

Traumatic brain injury (TBI) accounts for 1.4 million reported injuries and 52,000 deaths each year in the United States. TBI is the leading cause of death and disability in patients from ages 1 to 44 years. The main causes of TBI are motor vehicle crashes, falls, and assaults. Secondary neurological damage, the damage that occurs in the ensuing hours and days after the primary injury, contributes markedly to poor neurological outcome and mortality. Signs of secondary neurological damage include brain swelling (Figure 1), somnolence, abnormal motor function, and pupillary changes. Nevertheless, the onset and extent of secondary injury are still difficult to detect. Intensive neuromonitoring is therefore critical in improving neurological prognosis in patients with TBI.

Changes in intracranial pressure (ICP), cerebral perfusion pressure (CPP), brain tissue partial pressure
of oxygen (P_{BO_2}), blood pressure, brain temperature, and, recently, cerebral blood flow (CBF) are monitored in the intensive care unit (ICU).\textsuperscript{2} Cerebral microdialysis is a technique increasingly used as a bedside method for measuring glucose, lactate, pyruvate, and glycerol levels in the brain of patients with severe head trauma.\textsuperscript{3-5} Cerebral ischemia may be detected on the basis of aberrations in cerebral metabolites. Similarly, minimizing secondary ischemic injury common in TBI may be possible with the manipulation of ICP, CPP, CBF, blood pressure, brain temperature, and P_{BO_2} in brain parenchyma after acute brain injury.\textsuperscript{2,4-7}

In this article, we describe the successful use of multimodal neuromonitoring to guide therapy in our ICU. First, we provide an overview of the significance of changes in glucose, lactate, pyruvate, and glycerol levels in traumatically injured brain and review the importance and interrelationships between ICP, CPP, CBF, P_{BO_2}, blood pressure, and brain temperature. We also describe the background and important clinical aspects of specific current equipment used in our ICU to measure all the changes. Finally, we indicate threshold values that may change the treatment of patients with severe brain injury. Our emphasis is on cerebral microdialysis and CBF monitoring, which are relatively new monitoring techniques.

**Metabolic and Cellular Energy**

**Metabolic Trends of Microdialysis Markers**

Understanding the bioenergetics of hypoxic and/or ischemic brain is important.\textsuperscript{6-7} As brain tissue becomes hypoxic, oxygen no longer functions as the final electron carrier in the electron transport chain. Nicotinamide adenine dinucleotide hydrogen (NADH\textsuperscript{+}) is produced in aerobic glycolysis. These products are used in the Krebs cycle and electron transport chain to generate 32 molecules of ATP (left side). In ischemia and hypoxia, glucose and oxygen levels are reduced. Lack of oxygen disables the electron transport chain, causing cells to begin anaerobic respiration (right side), in which pyruvate is converted to lactate. Only 2 molecules of ATP are produced, resulting in brain tissue death and the release of glycerol.
and hypoxic states, glucose is converted primarily to lactate, resulting in decreased levels of pyruvate. These alterations in the Krebs cycle make lactate and pyruvate excellent indicators of energy failure in the brain. Because the levels of these compounds naturally fluctuate, detecting the changes in lactate levels in relation to pyruvate levels, a relationship known as the lactate to pyruvate ratio (LPR), is desirable. Extensive research has shown that the LPR is a good indicator of ischemic and hypoxic conditions as well as possible mitochondrial damage.

Under anaerobic conditions, the $P_{O_2}$ and glucose level decrease while the LPR increases. Elevated glycerol levels also indicate failure in cellular bioenergetics. Glycerol levels increase when cells do not have sufficient energy to maintain homeostasis. Because of a lack of ATP, calcium ion channels can no longer be maintained, and cellular influx of calcium occurs. The influx activates phospholipases, causing the phospholipids within cellular membranes to be enzymatically cleaved, yielding abnormally high glycerol levels.

**Microdialysis Technology**

Microdialysis enables measurement of the metabolic markers (glucose, pyruvate, lactate, and glycerol). The technique was first described in 1966, when Bito and colleagues successfully placed dextran membrane-lined sacks in the cerebral hemispheres of dogs to collect amino acids. This technique was refined to its present-day form in the 1970s by Tossman and Ungerstedt.

Microdialysis involves a catheter with a 10-mm semipermeable distal-end membrane that is placed into the brain parenchyma. The catheter is pumped with fluid isotonic to tissue interstitium. In short, the catheter acts as an artificial blood capillary (Figure 3). Through diffusion, molecules related to the production of ATP (glucose, pyruvate, lactate, glycerol) are collected from the interstitial fluid and are analyzed hourly. The metabolites recovered represent 70% of the true interstitial fluid concentrations.

**Microdialysis**

We use 2 devices to perform cerebral microdialysis. The CMA 600 (CMA Microdialysis, Solna, Sweden) is used for bedside analysis of metabolic indicators. This device was approved for use in the United States by the Food and Drug Administration in 2005. A second type of analyzer is the ISCUSflex (CMA Microdialysis), which was approved by the Food and Drug Administration in July 2009. At the time this article was written, our facility was the only one involved in beta tests of the ISCUS in the United States. We have clinical experience with more than 100 patients with both analyzers. The CMA 600 can be used to monitor up to 3 patients and 4 reagents. In contrast, the ISCUSflex can be used to monitor up to 8 patients simultaneously, with a total of 16 catheters and 5 reagents. Calibration of both analyzers is performed automatically every 6 hours, and controls are run every 24 hours.

The microdialysis catheter is placed through a bolt or burr hole or is implanted during an open craniotomy. The catheter is then attached to a microdialysis syringe filled with sterile perfusion fluid (artificial CSF) and placed in the CMA 106 pump, which is precalibrated to...
pump the perfusion fluid at a rate of 0.3 µL/min. In TBI patients, the catheter is ideally placed in the peri-contusional penumbra of the injury, and its position is verified by using computed tomography. In addition, we place a second microdialysis catheter in an area of undamaged tissue for comparison or reference. Each metabolite marker has a separate reagent for detection. The contents of the buffer solution are mixed in the reagent bottle.

We run samples every hour. Samples can be run every 20 minutes, as indicated by changes in a patient’s condition. Increased attention to a patient’s catheters will decrease the risk of dislodgement of the microdialysis catheters. Of note, the Clinical Laboratory Improvement Act requires control testing for this point-of-care device with low, normal, and abnormally high concentrations as controls. Known concentrations along the linear range of each analyte are run every 24 hours during monitoring and after a reagent change.

**Therapeutic Interventions for Abnormal Levels of Brain Metabolites**

Normal brain glucose levels are 30.6 (SD, 16.2) mg/dL (to convert to millimoles per liter, multiply by 0.0555). On the basis of the lowest level within the standard deviation (Table 1), the ischemic threshold for brain glucose is 14.4 mg/dL. In our patients, strict adherence to tight glycemic control (blood glucose levels 80-110 mg/dL) often leads to dangerously low brain glucose levels that can be detected only with cerebral microdialysis. To avoid cerebral hypoglycemia, we adjusted our blood glucose ranges to 110 to 180 mg/dL, resulting in brain glucose levels of 2.5 mg/dL or higher. This experience suggests that preventing systemic hypoglycemia most likely is key to preventing metabolic crisis and, ultimately, secondary brain injury. Therefore, earlier than usual nutritional support as a means of maintaining higher brain glucose levels may be important.

The normal LPR is 23 (SD, 4). Normal glycerol levels are 184 to 460 mg/dL (to convert to millimoles per liter, multiply by 0.1086). An LPR near a threshold value of 30, in conjunction with a low glucose level, requires intervention to prevent cellular energy failure. Similarly, glycerol levels near 921 mg/dL indicate cellular energy failure (Table 1). Baseline LPR and glycerol levels are recorded and watched for changes and trends toward threshold ischemic values. Routine interventions to lower the LPR, by preventing anaerobic respiration that leads to abnormally elevated glycerol levels, include increasing glucose levels by adjusting insulin infusions for a permissive blood glucose level of 110 to 180 mg/dL, elevating and adjusting the patient’s head and position, and augmenting CPP with vasopressors (Figure 4).

**Intracranial Monitoring**

**Physiology of ICP and CPP**

Elevated ICP is a common neurosurgical concern after trauma because it impedes CBF and is associated with ischemia and hypoxia. Common causes of elevated ICP include development of mass lesions such as subdural hematoma, epidural hematoma, and intracerebral contusions. Additionally, cerebral edema and communicating and noncommunicating hydrocephalus are treatable causes of increased ICP. ICP is the target parameter for many treatment algorithms. ICP is used to calculate CPP, the pressure gradient for blood perfusion in the brain measured in millimeters of mercury and used to calculate CBF (CPP/cerebral vascular resistance).

High ICP, a source of energy failure, is associated with a decreased CPP and lower CBF, the underlying cause of cellular energy failure. Increased ICP and low CPP often result in marked neurological morbidity and poor outcome. The threshold values of ICP, CPP, and CBF, which vary markedly according to body position, age, and body surface area, are widely debated. Finally, ICP monitoring is accepted in the Brain Trauma

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**Table 1** Normal and threshold values of metabolic parameters important in neuromonitoring of patients with traumatic brain injury

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal levels</th>
<th>Threshold value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose, mean (SD), mg/dL</td>
<td>30.6 (16.2)</td>
<td>14.4</td>
</tr>
<tr>
<td>Pyruvate, mean (SD), mg/dL</td>
<td>1.46 (0.33)</td>
<td>0.27 (fatal)</td>
</tr>
<tr>
<td>Lactate mean (SD), mg/dL</td>
<td>26.1 (8.1)</td>
<td>80.2 (fatal)</td>
</tr>
<tr>
<td>Lactate to pyruvate ratio</td>
<td>23 (4)</td>
<td>30</td>
</tr>
<tr>
<td>Glycerol, mg/dL</td>
<td>184-460</td>
<td>21</td>
</tr>
</tbody>
</table>

SI conversion factors: To convert glucose to mmol/L, multiply by 0.0555; pyruvate to µmol/L, multiply by 113.56; lactate to mmol/L, multiply by 0.111; glycerol to mmol/L, multiply by 0.1086.
Foundation guidelines as the gold standard for TBI monitoring to guide intervention.

Cerebral Pressure and Perfusion Monitoring Systems

Unlike microdialysis monitoring, intracerebral technology and monitors vary in concept and design. Bedside physiological monitors are used to measure ICP and calculate CPP. Pressure transduction varies between monitors and involves different mechanisms, including catheter-tip strain gauge, external strain, and fiber optics. Pupillometers provide an alternative method of evaluating ICP levels by giving quantitative evaluation of pupillary function.

Camino ICP Monitor

The Camino ICP monitor (Integra NeuroSciences, Plainsboro, NJ) consists of a patented fiber-optic transducer-tipped pressure-temperature catheter that is placed via a burr hole and can be used to measure ICP in the subdural, parenchymal, and ventricular spaces. The device measures ICP and brain temperature and displays ICP waveforms and the calculated CPP. The Camino catheter has a miniaturized transducer at the distal end. The device has no fluid-filled system, thus eliminating the problems associated with an external transducer, pressure dome, and pressure tubing. The monitor provides continuous information and does not require recalibration.

Elevated lactate to pyruvate ratio and/or high glycerol

Return aerobic respiration and glucose

Figure 4 Flow chart of metabolic related therapy integrates the possible responses to low blood glucose, elevated lactate to pyruvate ratio, and glucose (left). Normothermic treatment is used to reduce brain metabolic demand (right).

CODMAN ICP Express

The CODMAN ICP Express (Codman & Shurtleff Inc, Raynham, Massachusetts) is also used for measuring ICP and calculating CPP. The ICP Express can be used to measure ICP through a tunneled catheter, a bolted catheter, or an intraventricular approach. The ICP Express allows both continuous readings and CSF drainage via ventriculostomy. The ICP Express can be implanted in the subdural space or the intraparenchymal space and then secured to the skull.

Ventriculostomy

A ventriculostomy catheter provides a method for monitoring ICP while simultaneously reducing ICP through therapeutic CSF drainage. Using a ventriculostomy is particularly helpful in treating obstructive hydrocephalus. If an excessive amount of CSF accumulates in the ventricles after TBI, the fluid can be externally
drained through a ventricular catheter secured to the head.

ICP monitoring via a ventricular drain is accomplished by using a transducer system. Ventriculostomies are leveled at the tragus and open to drainage at the prescribed centimeters of water by the neurosurgeon orders. Documenting the amount of CSF drained hourly is important. Troubleshooting measures if drainage stops include lowering the ventriculostomy, flushing away from the head in case of clot in the tubing, and flushing 0.1 mL of preservative-free normal saline toward the head. The stopcock to the transducer must be turned in the direction of flow for continuous ICP monitoring or for drainage of CSF. During repositioning of the patient, the stopcock is turned to the off position to prevent overdrainage of CSF.

**Pupillometry.** The pupil check with a flashlight has always been a standard subjective measurement of pupil reactivity and status of the nervous system and brain. Now changes in constriction and dilation of pupils to light can be quantitatively assessed. The Neuroptics ForeSite pupillometer (Medtronic, Minneapolis, Minnesota) is a noninvasive, battery-operated, handheld device that uses light stimulus to assessably assess pupil reactivity. The Neuroptics pupillometer is an easy and quick adjunct to assessing neurological changes of patients with TBI.

In order to use the pupillometer, the head rest of the device must be fitted correctly. The head rest is disposable and should be changed for each patient. Awake patients are instructed to look straight ahead and focus their untested eye on a distant object. Manually and gently holding the patient’s eye open may be necessary. The green pupil boundary circle must be centered on the pupil for measurement. Exact measurement of each pupil and constriction is then obtained. This measurement is more reliable and consistent than the subjective assessment of a health care provider.

ICP monitoring and catheters have become the standard of care for measuring ICP. Baseline normal ICP levels range from 0 to 10 mm Hg; treatment threshold values are usually 20 to 25 mm Hg. Ideal CPP is approximately 60 mm Hg; the treatment threshold value is about 50 mm Hg (Table 2). Current TBI guidelines include first- and second-tier interventions to reduce ICP if it increases beyond the threshold value. First-tier interventions may involve draining CSF, increasing PaO₂ and PaCO₂ levels, administering diuretics, or elevating the head of the bed to an optimal 30º angle. Second-tier interventions involve administering medications, such as mannitol, furosemide (to reduce intravascular volume), hypertonic saline, or barbiturates, to reduce ICP. Patients who do not respond to these therapeutic interventions require computed tomography and, possibly, craniotomy or craniectomy. Finally, a brief trial of hyperventilation may be used as a temporary measure to control high ICP (Figure 5).

**Cerebral Blood Flow**

CBF is a complex and essential variable in determining whether the brain experiences posttraumatic secondary damage. Acute brain trauma causes a decrease in CBF while increasing the demand for blood and oxygen. Many variables affect blood flow in the brain, including metabolic regulation, PaCO₂, PaO₂, and autoregulation.

Increases in CPP can increase CBF during ischemic conditions. Autoregulation of this change in CPP and CBF includes vasoconstriction (Figure 6).
vasodilatation cascade occurs when CPP decreases, cyclically increasing vasodilatation. In response, ICP and cerebral vascular resistance increase, aggravating brain edema. In contrast, the vasoconstriction cascade occurs when CPP increases, causing constriction of vessels to reduce cerebral blood volume and CBF. If autoregulation is ineffective, CBF is determined by blood pressure. Hypotension may then cause ischemia. Similarly, hypertension may cause hyperemia.²⁶-²⁸

**CBF Monitoring Systems**

Direct measurement of CBF is relatively new in neurointensive care. Accordingly, real-time perfusion measuring devices and technology are still being developed and refined. Monitoring CBF could play an important role in neurological care, because the brain depends on continuous blood flow to supply glucose and oxygen. Regional CBF is considered an important upstream monitoring parameter indicative of tissue viability.²⁹

*Hemedex System.* The Hemedex CBF monitoring system (Codman & Shurtleff, Inc) is approved by the Food and Drug Administration for the bedside monitoring of tissue blood flow and circulation. With this device, CBF is measured by calculating real-time tissue perfusion at the capillary level (in milliliters per 100 grams per minute) with an attached probe. The probe is minimally invasive and includes a heated distal thermister and a proximal thermister to track baseline temperature. The monitor and probe measure tissue perfusion by measuring the ability of the tissue to carry heat through thermal conduction, represented as the K value by thermal convection from blood flow. The monitoring system calculates tissue perfusion by calculating thermal convection and total dissipated initial power. The probe can be viewed on computed tomography and radiography. It is not compatible with magnetic resonance imaging.³⁰

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**Abbreviations:** CPP, cerebral perfusion pressure; CSF, cerebral spinal fluid; CT, computed tomography; ICP, intracranial pressure; P<sub>BTO</sub>₂, brain tissue partial pressure of oxygen.
The probe is inserted through a burr hole or is placed 2 to 2.5 cm below the dura into brain white matter (Figure 7). The probe is secured via fixation disc or a single- or double-lumen bolt. In patients with TBI, the probe is placed either in noninjured brain white matter ipsilateral or contralateral to the injury or in the ischemic penumbra surrounding injured brain tissue. For comparison, a probe can be placed in uninjured brain tissue. Once the probe is placed by a neurosurgeon, a nurse attaches the probe to an umbilical cord and monitor to begin calibration. The proper K value for white brain matter is 4.9 to 5.8 mW/cm per degree Celsius. The probe can be retracted or advanced accordingly if the K value is not within range.

The monitor provides CBF parameters within a temperature range of 25°C to 39.5°C. Cooling the patient should be considered if brain temperature is greater than 38.5°C.

The monitor does not run on battery power, so the probe must be disconnected from the umbilical cord before the patient is transported to other departments for procedures or tests. The probe should be secured to the patient’s head dressing to prevent dislodging the probe. If the probe is used in conjunction with a microdialysis catheter, the 2 catheters must be separated by 2.0 mm for accurate results.

Transcranial Doppler Sonography. Although we do not routinely use transcranial Doppler sonography for patients who do not have an aneurysm, this technique is being investigated in patients with TBI. With this technique, a probe with a low-frequency ultrasonic signal is used on thin areas of cranium to measure velocity and direction of blood flow in the intracranial arteries. Although most commonly used to detect vasospasm after cerebral aneurysms, Doppler imaging can be used to detect posttraumatic cerebral hemodynamic changes and complications such as hyperemia.
vasospasm, decreased CBF, and intracranial hypertension.\textsuperscript{31,32} Transcranial Doppler sonography provides a real-time assessment of changes in flow velocity that reflect changes in CBF when cardiac output and blood pressure remain constant.\textsuperscript{33-34}

**Blood Pressure**

Mean arterial pressure (MAP) and ICP are important in calculating CPP (CPP = ICP − MAP). CPP is directly proportional to CBF. Drastic decreases in CPP result in decreased CBF. Autoregulation (Figure 6) protects the brain from variation in blood pressure. When autoregulation is functional, large changes in MAP do not lead to significant changes in CBF.\textsuperscript{35} If autoregulation is impaired, uncontrolled blood pressure directly causes changes in ICP, CPP, and CBF. In patients with impaired autoregulation, reducing blood pressure reduces CBF and aggravates ischemia. In contrast, in patients with impaired cerebral autoregulation, hypertension can cause increases in ICP and CBF.\textsuperscript{35} Blood pressure is measured by using a cuff or an arterial catheter. Normal CBF is 18 to 35 mL/100 g per minute.\textsuperscript{36} The threshold value is 15 mL/100 g per minute\textsuperscript{36} (Table 2). Because of the synergistic relationship between CBF and arterial blood pressure, both parameters must be considered in therapeutic decision making.\textsuperscript{28} MAP is monitored at least hourly; the goal is to maintain an optimal MAP to achieve a CPP greater than 60 mm Hg (Table 2). MAP can be controlled by using fluids and vasoactive agents. Medications that decrease MAP include metoprolol, nicardipine, enalapril, nitroglycerin, and nitroprusside. Suboptimal MAP can be increased by using phentylephrine, norepinephrine, vasopressin, or dopamine. Maintaining optimal CPP in order to maximize CPP may also require interventions that decrease ICP (Figure 5).

**Brain Tissue Oxygenation**

**Mechanisms**

Maintaining appropriate oxygen flow to satisfy the metabolic demands of the brain is critical to ensuring good neurological outcome. This concept is emphasized more generally in the overall physiological resuscitation of injured patients.\textsuperscript{1,17} Establishing a patent airway and restoring circulating blood volume and oxygenation are all attempts to maintain normal oxygenation of brain tissue.

The principal cause of secondary brain damage and poor neurological outcome is cerebral hypoxia triggered during the ischemic cascade.\textsuperscript{17,37} Systemic hypoxia, hypotension, and intracranial hypertension can lead to oxygen deprivation. If autoregulation is functional, low \( P_{\text{O}_2} \) can be resolved by vasodilatation. When autoregulation is impaired, low oxygen flow easily disrupts brain metabolism.\textsuperscript{17,28} The effects of manipulations of ICP, CPP, and \( P_{\text{CO}_2} \) on \( P_{\text{BTO}_2} \) have been reviewed extensively, stressing that high ICP and low CPP correlate with low \( P_{\text{BTO}_2} \) and poor neurological outcome.\textsuperscript{37}

**Monitoring Systems**

The Brain Trauma Foundation recommends oxygen monitoring because a significant number of patients with TBI have hypoxemia and hypotension. As with ICP technology, various techniques for \( P_{\text{BTO}_2} \) monitoring have been developed. Examples include indirect systems such as near-infrared spectroscopy and more invasive fiber-optic catheter technology; however, a direct monitoring system has recently been introduced.

The Licox \( P_{\text{BTO}_2} \) monitoring system (Integra NeuroSciences, Plainsboro, New Jersey) measures \( P_{\text{O}_2} \) and temperature in the brain. \( P_{\text{O}_2} \) is an established marker of cerebral ischemia and secondary brain injury. The triple-lumen introducer kit, with a 7-mm–long oxygen-sensing area at the distal tip, measures regional oxygenation, with separate probes to measure ICP and temperature. The most recent device provides the option to bolt or tunnel the catheter and has a sensor that measures temperature and oxygen integrated into the same catheter. The Licox catheter uses Clark-type electrode technology to measure \( P_{\text{O}_2} \) in blood of tissue.\textsuperscript{38}

The catheter is placed 25 to 35 mm into the brain. The oxygen sensor is located in the white matter of the brain, preferably in the penumbra of the injured area. The catheter is inserted via the triple- or double-lumen bolt. The optimal location for normal brain measurements is uninjured brain. Setup and calibration are minimal. After the brain has adjusted to the new catheter, an oxygen challenge test should be performed by setting the ventilator fraction of inspired oxygen at 100% for 2 to 5 minutes. \( P_{\text{BTO}_2} \) should increase. A neurosurgeon can adjust the probe as needed.

Although measuring ICP and CPP is key in patients with TBI, monitoring cerebral oxygenation can indicate hypoxic events earlier than monitoring ICP and CPP can and thus may improve neurological
outcome. The goal \( P_{\text{BtO}_2} \) value is greater than 20 mm Hg; the ideal is 30 mm Hg. Lower values may indicate impending hypoxia. A \( P_{\text{BtO}_2} \) of 55 mm Hg suggests a threshold value defined as “luxury perfusion.” In order to improve \( P_{\text{BtO}_2} \) during ischemic conditions, CBF can be maximized by decreasing ICP via barbiturates, CSF drainage, and/or craniotomy. If the decreased \( P_{\text{BtO}_2} \) is due to lower oxygen delivery, increasing CPP and avoiding hypotension, hypovolemia, and hypoxia will be important. Common interventions to improve cerebral oxygen delivery include administration of isotonic solutions, vasopressors, and blood transfusions and increases in the fraction of inspired oxygen. Because pain, shivering, agitation, and fever further increase cerebral metabolism, sedatives, anti-inflammatory agents, and cooling devices are used. In contrast, \( P_{\text{BtO}_2} \) may reach luxury perfusion levels because of hyperemia or excessive cerebral blood flow, which increases ICP. High \( P_{\text{BtO}_2} \) and hyperemia can be temporarily reduced with carefully guided prophylactic hyperventilation, although this intervention may cause secondary injury. If hyperventilation is used, brain oxygenation should be monitored with either a tissue oxygenation monitor or a jugular bulb catheter. Decreasing body temperature and inducing heavy sedation can further decrease the demand of the brain tissue and in turn increase \( P_{\text{BtO}_2} \) (Figure 5).

**Brain Temperature and Hypothermia**

**Reduction of Brain Temperature**

In humans, brain temperature is an important marker of brain metabolism and cellular injury. In initial studies on ischemic brain in animals, slight changes in brain temperature accounted for fluctuations in histological changes in brain tissue. Normothermia and moderate hypothermia in rats (33°C) resulted in a marked decrease in brain glutamate levels, the metabolite uncontrollably released during tissue energy failure. Although lowering brain temperature in humans with TBI is still debated and scientifically unproved, the intervention is a neuroprotective strategy that in theory reduces the metabolic demand of the brain, possibly decreasing secondary neuronal injury and improving behavioral outcomes.

The mechanisms of moderate hypothermia (32°C-33°C) and normothermia (36°C-37°C) on postschismic tissue, although complex, are multifunctional. At the cellular level, hypothermia and reduction of brain temperature in general can block excitatory neurotransmitters. Prevention of toxic calcium overload allows continued proper amino acid folding by replacing ubiquitin and results in improved oxygen delivery and CBF and depresses the immune response. Increased brain temperature has been associated with longer ICU stays and thus extended intensive care, as well as higher mortality. Finally, smaller nonrandom and class 2 clinical studies have indicated the successful use of hypothermia in neuroprotection. Similarly the Brain Trauma Foundation has recommended that induced hypothermia within the first 48 hours of injury may reduce mortality, further stressing the importance and merit of temperature monitoring in TBI.

**Monitoring Brain Temperature**

Monitoring brain temperature is relatively easy because it is an integral part of multiple systems. In our ICU, the Camino bolt system and the Hemedex and Licox systems can all be used to monitor brain temperature.

Brain hyperthermia, a temperature of 38.5°C or greater, can be prevented by multiple methods. Of note, brain hyperthermia must be monitored simultaneously with body temperature to ensure that cooling interventions are adequately affecting the temperature of the injured brain tissue. Administration of antipyretics such as acetaminophen or ibuprofen is a common initial therapeutic intervention. Passive cooling measures such as cooling blankets and/or ice packs can be used. Invasive cooling measures are considered if the noninvasive methods are ineffective. We use the Thermogard XP system (Zoll Medical Corporation, Chelmsford, Massachusetts), an intravascular, multilumen catheter. We prevent hyperthermia in patients with TBI by starting use of the Thermogard XP system if brain temperature is greater than 38.5°C. The Hemedex monitor for CBF functions within the temperature ranges of 25°C to 39.5°C. The goal of using the Thermogard XP system is to reduce brain temperature to a normothermic range of 36°C to 37°C. Our hyperthermia orders require frequent laboratory tests for levels of potassium, phosphates, and magnesium; prothrombin and partial thromboplastin times, and platelet counts to prevent coagulopathies.

Treatment with the Thermogard may cause shivering, a normal
thermoregulatory response to hypothermia. Shivering increases oxygen consumption in skeletal muscles, diverting valuable oxygen away from the injured brain. Refractory shivering may require deep sedation and/or the administration of paralytic agents to facilitate the induction and maintenance of hypothermia and minimize oxygen consumption.\textsuperscript{43-45}

**Conclusion**

Advancements in neuromonitoring have improved the bedside care of patients with TBI. These developments have provided the possibility of true multimodal monitoring for effective therapy. As described in this article, we have taken steps to turn this possibility into a routine standard of practice. Neuromonitoring traditionally has been used as a method of detecting problems as the problems emerge. Yet, many of these technologies can be used to detect problems before the problems become major, thus creating the opportunity for more timely interventions. The nursing staff in our ICU realize that caring for patients with complex brain injuries requires vigilant monitoring of multiple parameters in hopes of preventing secondary injury. In addition to the conventional placement of a ventriculostomy in a patient with TBI, we routinely use microdialysis to evaluate metabolic changes (glucose, pyruvate, lactate, glycerol) and various monitoring systems to assess ICP, CPP, CBF, blood pressure, and brain temperature. Figure 8 shows placement of the devices in a typical patient in our ICU. In this article, we have detailed our practice by explaining the background of the parameters monitored in TBI patients, the technical aspects of each machine or device used, and related therapeutic interventions. Our use of multimodal monitoring to provide comprehensive care has great potential to improve the outcomes of our patients who have marked neurological injury. \textsuperscript{46}

![Figure 8: Placement of multimodal catheters and monitoring probes.](Image)

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