Traumatic Brain Injury: Advanced Multimodal Neuromonitoring From Theory to Clinical Practice

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PRIME POINTS

• Advancements in multimodal neuromonitoring involve markers of brain metabolism.

• Physiological parameters such as intracranial pressure, cerebral perfusion pressure, cerebral blood flow, brain tissue partial pressure of oxygen, blood pressure, and brain temperature affect brain monitoring.

• Aspects of multimodal monitoring and the use of specific equipment guide therapy in patients with traumatic brain injury.

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_{T_{O_2}}), blood pressure, brain temperature, and, recently, cerebral blood flow (CBF) are monitored in the intensive care unit (ICU). ©2010 American Association of Critical-Care Nurses doi: 10.4037/ccn2010226

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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. 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 \( \text{PbT} \text{O}_2 \) in brain parenchyma after acute brain injury.

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, \( \text{PbT} \text{O}_2 \), 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. As brain tissue becomes hypoxic, oxygen no longer functions as the final electron carrier in the electron transport chain. Nicotinamide adenine dinucleotide hydrogen (NADH+) 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.
ischemia, a lack of oxygen and glucose results in anaerobic respiration. In ischemic and hypoxic states, glucose is converted primarily to lactate, resulting in decreased levels of pyruvate.4,8-10 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.5,7,11,12 Under anaerobic conditions, the PBTO2 and glucose level decrease while the LPR increases.8,9 Elevated glycerol levels also indicate failure in cellular bioenergetics. Glycerol levels increase when cells do not have sufficient energy to maintain homeostasis.11,12 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.7

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.7,11 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 capillary12 (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.7,11 The metabolites recovered represent 70% of the true interstitial fluid concentrations.3

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

Figure 3 Microdialysis technique. The microdialysis catheter at the distal end acts as an artificial capillary. Extracellular substances diffuse from interstitial tissue to perfusion fluid inside the catheter membrane. Courtesy of CMA Microdialysis, Solna, Sweden.
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 pericontusional penumbra of the injury, and its position is verified by using computed tomography.\(^5\) 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.\(^8\)

### 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).\(^{3,14}\) 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.\(^15\) 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.\(^15\) 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).\(^9\) 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\(^3,14\) (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.\(^16\) 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.\(^17,18\) 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).\(^2,18\)

High ICP, a source of energy failure, is associated with a decreased CPP and lower CBF, the underlying cause of cellular energy failure.\(^19\) 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.\(^16,13,20\)

Finally, ICP monitoring is accepted
in the Brain Trauma 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.

The fiber-optic catheter, with its integrated transducer, is inserted through a burr hole space in the subdural, parenchymal, and ventricular spaces. The transducer must be zeroed before insertion and disconnected from the preamplification connector when a patient is moved. A red mark will indicate correct placement in the brain parenchyma. The subdural and ventricular catheters do not have a red line, rather they have graduated lines to calculate the depth of the catheter.

The catheter is visible on computed tomography and is not compatible with magnetic resonance imaging.

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 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 dilatation of pupils to light can be quantitatively assessed. The Neuroptics ForeSite pupillometer (Medtronic, Minneapolis, Minnesota) is a noninvasive, battery-operated, hand-held device that uses light stimulus and rapid live photography to measure maximum and minimum aperture and constriction velocity of pupils. Although the pupillometer is a relatively new device, preliminary testing suggests that constriction velocities less than 0.8 mm/s indicate increased brain volume and velocities less than 0.6 mm/s suggest elevated and problematic ICP. Similarly, pupil reactivity less than 10% after light stimulus suggests elevated ICP and should be considered in conjunction with the results of other monitoring systems for ICP.

We have found that using the 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 healthcare 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 \(P_{O_2}\) and \(P_{CO_2}\) levels, administering diuretics, or elevating the head of the bed to an optimal 30° 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 craniec- tomy. 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, \(P_{CO_2}\), \(P_{O_2}\), and autoregulation. Increases in CPP can increase CBF during ischemic conditions. Autoregulation of this change in CPP and CBF includes vasodilatation and vasoconstriction (Figure 6). The 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

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 umbil-
cical cord and monitor to begin cali-
bration. 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 param-
eters within a temperature range of
25°C to 39.5°C. Cooling the patient
should be considered if brain tem-
perature 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 hemody-
namic changes and complications
such as hyperemia, vasospasm,
decreased CBF, and intracranial
hypertension. Transcranial Doppler
sonography provides a real-time
assessment of changes in flow

Figure 6 Vasodilatation (left) and vasoconstriction (right) cascades protect the brain. The dynamics of cerebral blood flow are best encompassed by the patterns of intact autoregulation. The vasodilatation cascade occurs when cerebral perfusion pressure (CPP) decreases, leading to increases in cerebral blood volume (CBV) and intracranial pressure (ICP), which can lead to edema. If CPP increases, vasoconstriction occurs, reducing CBV and decreasing edema by decreasing ICP.

Figure 7 Hemedex catheter. Probe can be tunneled or bolted. Probe is embedded 2 to 2.5 cm below the dura.

Courtesy Hemedex Inc, Cambridge, Massachusetts.
velocity that reflect changes in CBF when cardiac output and blood pressure remain constant.32-34

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.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.35 Blood pressure is measured by using a cuff or an arterial catheter.

Normal CBF is 18 to 35 mL/100 g per minute.30 The threshold value is 15 mL/100 g per minute36 (Table 2). Because of the synergistic relationship between CBF and arterial blood pressure, both parameters must be considered in therapeutic decision making.26 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 nitropresside. Suboptimal MAP can be increased by using phenylephrine, norepinephrine, vasopressin, or dopamine. Maintaining optimal CPP in order to maximize CBF 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.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.25-27 Systemic hypoxia, hypotension, and intracranial hypertension can lead to oxygen deprivation. If autoregulation is functional, low PO2 can be resolved by vasodilatation. When autoregulation is impaired, low oxygen flow easily disrupts brain metabolism.25-28 The effects of manipulations of ICP, CPP, and PCO2 on PbTO2 have been reviewed extensively, stressing that high ICP and low CPP correlate with low PbTO2 and poor neurological outcome.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 PbTO2 monitoring have been developed. Examples include indirect systems such as near-infrared spectroscopy and more invasive fiber-optic catheter technology1; however, a direct monitoring system has recently been introduced.

The Licox PbTO2 monitoring system (Integra NeuroSciences, Plainsboro, New Jersey) measures PO2 and temperature in the brain. PO2 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 PO2 in blood of tissue.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. PbTO2 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 PbTO2 value is greater than 20 mm Hg; the ideal is 30 mm Hg. Lower values may
Temperature and Hypothermia

Reduction of Brain Temperature

In order to improve $P_{BTO_2}$ during ischemic conditions, CBF can be maximized by decreasing ICP via barbiturates, CSF drainage, and/or craniotomy. If the decreased $P_{BTO_2}$ 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, antiinflammatory agents, and cooling devices are used. In contrast, $P_{BTO_2}$ may reach luxury perfusion levels because of hyperemia or excessive cerebral blood flow, which increases ICP. High $P_{BTO_2}$ 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 $P_{BTO_2}$.

**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 postischemic 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.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.

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