Severe traumatic brain injury has challenged the medical community for decades. According to the Centers for Disease Control and Prevention, a traumatic brain injury is “an injury to the head that disrupts the normal function of the brain.” Almost 1.5 million cases of traumatic brain injury—some mild, some severe—are reported each year in the United States. Approximately 50,000 of the persons who have a traumatic brain injury die, and 80,000 leave the hospital with some disability. Currently, about 5.3 million persons in the United States live with a disability caused by a traumatic brain injury. The primary injury, which happens at the time of the event, causes the disruption of axons, cell bodies, and the integrity of the cell membrane, resulting in an accelerated disintegration of cell structure and function and, eventually, cell death. Secondary injury occurs in response to unchecked cerebral edema, ischemia, and the chemical changes associated with direct trauma to or systemic effects on the brain.

Historically, the management of traumatic brain injury focused on the management of intracranial pressure (ICP) and cerebral perfusion pressure (CPP) via a variety of technologies.

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. Discuss the dynamics of brain injury related to oxygen
2. Identify the influence of interventions on brain tissue oxygen
3. Describe the use of brain tissue oxygen monitoring in severe brain injury

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ICP monitoring has never been scrutinized in a prospective randomized study. However, most clinicians agree that monitoring provides information that can improve patients’ outcomes when targeted interventions are used to control ICP and CPP. This agreement was indicated by the acceptance of the Guidelines for the Management of Severe Brain Injury, published in 2000 by the Brain Trauma Foundation, and guideline-compliant care.3,4

The concept of managing and treating only ICP in brain injury limits the ability to assess perhaps one of the most important parameters: oxygenation. The delivery and use of oxygen at the cellular level, in addition to control of the excitatory amino acids, can directly affect tissue survival. Technological advances in brain tissue oxygen monitoring provide information on the cellular dynamics of oxygenation and a better understanding of the impact of low oxygen states in the brain on patients’ outcome. This technology enables practitioners to assess levels of brain tissue oxygen associated with secondary injury and with treatment interventions.5 In this article, we provide an overview and historical perspective of the latest technology in brain tissue oxygen monitoring, present research findings on factors that affect the levels of brain tissue oxygen, and suggest interventions to maintain adequate brain oxygenation.

**Dynamics of Brain Injury Related to Oxygen**

The Monro-Kellie doctrine states that the cranium is a closed box with essentially noncompressible contents of approximately 80% brain tissue, 10% blood, and 10% cerebrospinal fluid. These percentages remain constant through intrinsic regulatory mechanisms, but an increase in any one or more of the contents requires compensation and a decrease in the others. Under normal circumstances, the brain compensates to some degree through autoregulation, shunting of cerebrospinal fluid, and compliance. Once these compensatory mechanisms are exhausted, increases in ICP occur. If the pressure increases markedly, the amount of blood flow to brain tissue is reduced, thus compromising cerebral tissue oxygenation. The causes of tissue hypoxia and ischemia may be related to intracranial events (eg, edema, structural damage, intracranial hypertension, seizures, vasospasm), systemic events (eg, hypoxemia, hypotension, hypocapnia, anemia, hyperthermia), or a combination of the two.

The brain depends on the constant uninterrupted delivery of oxygen and glucose to prevent secondary ischemic injury. Any decrease in perfusion in the brain causes additional secondary ischemia and injury and results in poor outcomes.6 Because failure to deliver oxygenated blood to injured brain tissue is thought to be detrimental, Meixensberger et al7 suggest that monitoring the partial pressure of brain tissue oxygen (PbtO2) may help prevent hypoxic events and improve patients’ outcomes. Studies in Europe indicated that measurement of ICP and CPP alone does not accurately reflect the tissue oxygenation in injured brain.8 The development of PbtO2 monitoring adds to the information needed to target therapy in patients as they respond to the changes in blood flow, decrease in energy production, alteration in cellular response, and potential ischemia.9

**Historical Perspectives in Brain Tissue Oxygen Monitoring**

Because the interest of many practitioners is the sufficient delivery and use of oxygen in brain tissues for necessary cellular function, brain tissue oxygen monitoring should enable clinicians to measure the difference between delivery and consumption of oxygen. Developing an accurate and reliable method for measuring brain tissue oxygenation has been the priority of researchers during the past 15 to 20 years.

Tissue oxygenation is heterogeneous in animal and human models and has been well defined since the 1960s by using the Clark cell method of measurement.10 A Clark cell polarographic probe is a semipermeable membrane covering 2 electrodes, 1 silver and 1 gold. In the presence of dissolved oxygen crossing the membrane, an electrical current is generated and is transferred to a monitor for interpretation. Early trials of tissue oxygen measurement were done in an animal model to ensure the usefulness and validity of the information derived from placement of the probe in both cerebrospinal fluid and tissue.11 In both animals and humans, accurate placement of the probe in the lateral ventricle is easily accomplished.12 Values obtained reflect the expected and appropriate changes during manipulation of the blood pressure and oxygenation and correlate with measurements taken in the deep white matter of the brain. A distinct drawback in measuring oxygen in the cerebrospinal fluid of the ventricles, however, is the condition of the ventricles in head injury. They are often slitlike because of secondary swelling, and monitoring in this circumstance does not provide an accu-
rate measure of oxygenation in the tissue of the brain. Therefore, measuring the oxygen level in the tissue is obviously more useful. Further testing of probe placement revealed that measurements taken from the deep white matter of the brain are the most valuable and stable because oxygen consumption is most stable in that area. Placement of the probe in a bolt with a predetermined depth allows access to the white matter of the brain.

Early evaluations of the measurement of oxygen in tissue included measurement of tissue temperature, because the temperature coefficient is needed to calculate the oxygen value. Several studies indicated that gradients exist between brain temperature and body temperature in the bladder, rectum, and jugular bulb. Although researchers have assumed that rectal temperature reflects brain temperature, Rumana et al. found that brain temperature was consistently and significantly higher than the core body temperature after brain injury. Thirty patients were evaluated and monitored by using a LICOX oxygen/temperature probe placed in the parenchyma, a rectal temperature probe, and a jugular bulb catheter. In the initial 5 days after admission, mean brain temperature was 38.9°C (SD 1.0°C), and mean rectal temperature was 37.8°C (SD 0.4°C); both rectal and jugular venous oximetric (SjvO2) temperatures were 1.1°C (SD 0.6°C) lower than mean brain temperature. The difference was more than 1°C in 18 patients and more than 2°C in 3; brain temperatures were lower than rectal temperatures in 2 patients. The temperature in the brain was also measured before and after the induction of barbiturate coma; values before and after induction did not differ significantly.

In a small sample of 8 patients, Henker et al. found that brain tissue temperature was underrepresented (ie, higher than bladder and rectal temperatures); mean brain temperature was 0.32°C to 1.9°C higher than bladder and rectal temperature. In most patients, when the bladder and rectal temperatures were outside the normal range, the difference was even greater.

Several studies indicated the importance of controlling fever in patients with severe head injury, and clinicians should be vigilant about the control of fever. Because temperatures are measured outside the cranium in most patients, the information may not necessarily be pertinent to the brain. At this time, we have not determined what temperature will have deleterious effects on patients, but we have observed a decrease in PbtO2 when patients become febrile.

Although other monitoring systems are available for tissue oxygen monitoring, most studies have been done with the LICOX system (Integra NeuroSciences/GMS, Plainsboro NJ; Figure 1). This PbtO2 system was developed by Wolfgang Fleckenstein of Kiel, Germany, and has been used in tissue oxygen monitoring since the 1980s. The LICOX system includes a monitor with a screen for the display of oxygen and temperature values; cables that connect to the monitoring probes and to the bedside monitor; and a variety of probes for use in the brain, cardiac muscle, and peripheral muscles; under skin flaps; and during CRITICAL CARE NURSE. Vol 23, No. 4, AUGUST 2003

Planning interventions at the bedside to enhance tissue oxygenation can affect patients’ outcomes.

FIGURE 1 LICOX catheter system. Left, The brain tissue oxygen catheter and monitor. Right, Placement of the catheter in brain white matter.

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The ease of use and the pre-calibrated “smart” card that accompanies the probes make this single-step “system calibration” appealing to bedside nurses. The only additional time needed before monitoring is the tissue “settling” time after the microtrauma of insertion.18

Values that indicate normal levels of tissue oxygen seem to vary between the available monitoring systems, possibly because the technology varies, causing some confusion among users. However, a consensus exists that low levels (<15 mm Hg) during the resuscitation phase of traumatic brain injury are predictive of poor outcomes. Initial (first 8-12 hours after injury) oxygen values were low in several studies.19-21 Data from at least one center indicated that early intervention and targeted protocol-driven management can positively affect the outcomes of patients with severe traumatic brain injury.22

Debate over where to place the Pbto2 catheter is ongoing, with advantages and challenges in each application. Placement in the penumbra of an injury (Figure 2) is useful in assessing an increase in swelling and regional oxygenation. The challenge with this measurement is that the values reflect the local area and may not be indicative of the remainder of the uninjured brain. Placement in the so-called undamaged or normal tissue allows clinicians to use the values as a representation of somewhat normal global oxygen delivery (Figure 3) but may not reflect the subtleties of regional hypoxia.23-26

With the development of the LICOX system, critical thresholds (ie, normal and abnormal levels of Pbto2) had to be determined. Additionally, information on the influence of various parameters on Pbto2, and the impact of oxygen monitoring on patients’ outcomes was needed.

Measurement and Safety of Brain Tissue Oxygen Monitoring

Numerous researchers in the 1990s sought to determine normal and abnormal levels of brain tissue oxygenation in both animal and human models. The researchers discovered that use of 2 different monitoring systems led to different normal and abnormal values.25,27-29 Our review of the literature on normal and abnormal Pbto2 here is confined to the LICOX monitoring system because the preponderance
of research has been done with this system. It is also the system currently in use by clinicians at the bedside in the United States.

Maas et al\textsuperscript{11} placed the oxygen sensor in the white matter of the frontal lobe and determined that baseline PbtO\textsubscript{2} values were 25 to 30 mm Hg. Sarrafzadeh et al\textsuperscript{28} found that PbtO\textsubscript{2} was between 20 and 35 mm Hg in uninjured brain tissue.

Valadka et al\textsuperscript{25} studied 39 patients and found that extended periods with PbtO\textsubscript{2} less than 15 mm Hg correlated with a greater chance of death. Any occurrence of a PbtO\textsubscript{2} less than 6 mm Hg (no matter how long the time below this level) was associated with an increased risk of death. Therefore, Valadka et al considered a PbtO\textsubscript{2} less than 15 mm Hg as a significant threshold. Bardt et al\textsuperscript{29} found that PbtO\textsubscript{2} less than 10 mm Hg was associated with poor outcomes (ie, patients were severely disabled or died). Patients who experienced a PbtO\textsubscript{2} of less than 10 mm Hg for longer than 30 minutes had the worst outcomes at discharge: 50% died and 50% were severely disabled. At 6-month follow-up, 22.2% of these patients had a favorable outcome, 22.2% were severely disabled, and 55.6% had died. In the group of patients who experienced a PbtO\textsubscript{2} less than 10 mm Hg for less than 30 minutes, 80% were severely disabled or in a persistent vegetative state at discharge, yet their outcome at 6 months improved; 70% had a favorable outcome, 20% were severely disabled, and 10% had died.

In a prospective randomized trial by van den Brink et al\textsuperscript{26} 101 comatose patients with head injury were evaluated after placement of the brain oxygen monitor. They were treated according to the European guidelines for the management of severe traumatic brain injury.\textsuperscript{32} Outcome at 6 months was determined by the score on the Glasgow Outcome Scale, which is used in early prediction of gross outcome after traumatic brain injury. Scores range from 1 to 5, with 1 indicating death and 5 indicating a good recovery.\textsuperscript{35} Despite aggressive management of ICP and CPP, numerous episodes of brain tissue hypoxia occurred. In the first 24 hours after injury, PbtO\textsubscript{2} was lower than 15 mm Hg for longer than 30 minutes in 57 patients, lower than 10 mm Hg in 42, and lower than 5 mm Hg in 22. The severity and duration of tissue hypoxia were directly related to poor outcome and an increased risk of death and were an independent predictor of outcome.

Practitioners must be cognizant of normal PbtO\textsubscript{2} values and appreciate the effect that abnormally low values have on patients’ outcomes. With the LICOX monitoring system, the normal value is thought to be 20 mm Hg or higher. Striving to maintain a PbtO\textsubscript{2} of 20 mm Hg or higher is an acceptable starting point. Practitioners should be concerned and act quickly when the PbtO\textsubscript{2} decreases to less than 15 mm Hg.\textsuperscript{27} In one study,\textsuperscript{25} 4 of 5 patients with PbtO\textsubscript{2} less than 5 mm Hg died.

**Risks of Brain Tissue Oxygen Monitoring**

The risk of brain tissue oxygen monitoring has been detailed in a number of articles. In 9 studies,\textsuperscript{3,6,21,26,28,30,32,35} from 1996 through 2000, in which investigators examined safety parameters, infection, and/or hematoma in a total of 250 patients, only 2 adverse events occurred. No infections were reported, and the adverse events were related to small hematomas that occurred after catheter placement.\textsuperscript{26} Dings et al\textsuperscript{26} stated that because 3 probes were inserted through a bolt (ICP, temperature, and oxygen probes), determining which of the probes caused the bleeding was difficult. Because ICP monitoring was done in the reported cases\textsuperscript{26} through a bolt, the prevalence of hematoma (4.95%) was within the range associated with this type of ICP monitoring (ie, via a bolt).\textsuperscript{36}

**Influence of Interventions on PbtO\textsubscript{2}**

Directly monitoring PbtO\textsubscript{2} provides vital information on the effect of interventions in individual patients. During the past decade, brain tissue oxygen monitoring has been described by numerous authors,\textsuperscript{5,21,37} predominantly in Europe, where multimodality monitoring has been implemented in many intensive care units. Early monitoring of critical parameters in patients with traumatic brain injury can provide useful clinical information. Correlations of PbtO\textsubscript{2} with parameters such as ICP, CPP, SjvO\textsubscript{2}, and end-tidal carbon dioxide have been reported.\textsuperscript{3,21,37} Brain tissue oxygen monitoring can be used to provide information at the bedside about responses to clinical interventions and the success of the interventions.

In the following sections, we address common interventions in the management of patients with traumatic brain injury. Measures are usually taken to ensure adequate perfusion, and the results are measured as the reduction in ICP and the increase in CPP. Strong evidence
exists that hypotension and hypoxia should be avoided and CPP-guided therapy should be used in patients with severe brain injury.\textsuperscript{38,39} However, these measures are often unrelated to the delivery and utilization of oxygen in the brain.

**Influence of Interventions to Treat Traumatic Brain Injury**

The administration of oxygen or titration of the fraction of inspired oxygen (FIO\textsubscript{2}) and its impact on PbtO\textsubscript{2} has been investigated in several studies. In 22 patients with severe head injury, LICOX monitoring indicated that increasing the FIO\textsubscript{2} led to an increase in PbtO\textsubscript{2}.\textsuperscript{33} Van den Brink et al\textsuperscript{35} examined the effects of preoxygenation before suctioning on PbtO\textsubscript{2} in 82 patients with head injury. In 7 patients, a decrease in PbtO\textsubscript{2} occurred during suctioning in 16 episodes when preoxygenation was omitted. In 142 episodes in which preoxygenation before suctioning was used, PbtO\textsubscript{2} levels increased.\textsuperscript{35} In 9 patients with grade IV gliomas, increasing the FIO\textsubscript{2} to 100% concomitantly increased the low PbtO\textsubscript{2} 2.5- to 4-fold.\textsuperscript{40}

Menzel et al\textsuperscript{41} studied the effects of increasing PaO\textsubscript{2} to levels higher than the level necessary for hemoglobin saturation during the early phase after severe brain injury in a randomized sample of 24 patients. In the 12 patients in whom FIO\textsubscript{2} was increased, mean PaO\textsubscript{2} levels increased to 359% (SD 39%) of the baseline in a 6-hour enhancement session, and the dialysate levels of lactate decreased by 40%. This decrease in lactate levels may indicate the prevention of anaerobic metabolism in the injured brain due to supranormal levels of oxygen.\textsuperscript{41}

The proposed reason for the increase in PbtO\textsubscript{2} associated with an increase in FIO\textsubscript{2} is the enhancement of the dissolved oxygen in plasma. Although the dissolved oxygen in plasma makes up only 2% to 3% of the total arterial oxygen content, PaO\textsubscript{2} appears to be the driving force of oxygen movement from plasma to tissue.\textsuperscript{30} Bedside clinicians have resisted using oxygen therapy in patients with severe brain injury because of perceived damage due to free radicals associated with use of the therapy, but more aggressive management may have a place in the treatment of patients with severe brain injury.

**Influence of CPP and ICP**

Enhancing CPP by decreasing ICP or increasing mean arterial blood pressure can increase PbtO\textsubscript{2}. CPP can be enhanced by using vasoconstriction of the cerebral blood vessels, thus reducing blood flow and, ultimately, oxygen delivery.

**Influence of Carbon Dioxide**

Hyperventilation continues to be a treatment for intracranial hypertension in some institutions even though it has deleterious effects in patients with severe brain injury when used injudiciously.\textsuperscript{35,42} Schneider et al\textsuperscript{42} found that although hyperventilation dramatically decreased ICP and increased CPP, brain tissue oxygenation could decrease to as low as 10 mm Hg during hyperventilation. Van den Brink\textsuperscript{35} found that in 17 of 23 patients, PbtO\textsubscript{2} decreased during the first 24 hours after injury because of decreases in PaCO\textsubscript{2} associated with hyperventilation. It is well documented that hyperventilation in the first 24 hours after brain injury can cause hypoxia in the tissue at risk.\textsuperscript{37,39}

Meixensberger et al\textsuperscript{7} placed an oxygen probe in the cortex during craniotomy and observed patients who had no secondary swelling and patients who had pathological changes and swelling. Both groups had an increase in PbtO\textsubscript{2} in brain tissue when they were breathing 100% oxygen. However, although the group with no swelling had no correlation between tissue PbtO\textsubscript{2} and arterial PbtO\textsubscript{2}, the group with pathological changes did. In addition, low tissue PbtO\textsubscript{2} occurred in both groups after hyperventilation, suggesting that some patients are at risk for hypoxia during this intervention. Lowering the PaCO\textsubscript{2} produces vasoconstriction of the cerebral blood vessels, thus reducing blood flow and, ultimately, oxygen delivery.
A positive correlation between decreases in PbtO$_2$ and CPP less than 60 mm Hg. Stocchetti et al$^1$ refuted the 2 previous claims that increasing CPP to more than 60 mm Hg had no effect on PbtO$_2$. In their study$^2$, when CPP was increased from a mean of 77 mm Hg (SD 9 mm Hg) to 96 mm Hg (SD 11 mm Hg), the PbtO$_2$ increased from 24 mm Hg (SD 13 mm Hg) to 31 mm Hg (SD 13 mm Hg).

Van den Brink$^3$ found that changing the tubing used to administer vasopressor solution caused the CPP to decrease, with a resulting decrease in PbtO$_2$. Changing tubing is common in the intensive care unit, with an accompanying decrease in blood pressure and CPP. Artru et al$^4$ reported that despite increasing CPP to normal levels and greater, patients experienced hypoxic episodes during measurement of brain tissue oxygen. Using transcranial Doppler imaging of the middle cerebral artery, Dings et al$^5$ found a positive correlation between CPP and PbtO$_2$ changes in the 7 days after evacuation of a hematoma.

Kiening et al$^6$ also reviewed the correlation of measurements of ICP, CPP, pulse oximetry, SjvO$_2$, and end tidal carbon dioxide with PbtO$_2$ in 15 patients with traumatic brain injury. The “time of good data quality” was 95% for PbtO$_2$ compared with only 43% for SjvO$_2$, supporting the use of PbtO$_2$ monitoring as an adjunct to ICP and CPP monitoring. Both SjvO$_2$ and PbtO$_2$ correlated with CPP during decreases in CPP, and the correlation was best when the CPP decreased to a level less than a breakpoint of 60 mm Hg, suggesting intact autoregulation. They concluded that tissue oxygen monitoring is safe, reliable, and suitable for long-term monitoring.

In a study of 35 patients with severe head injury, Bardt et al$^7$ found that during elevation of ICP and decrease in CPP, the patients experienced cerebral hypoxia and that the degree and prevalence of these episodes affected the patients’ outcome. Low PbtO$_2$ readings occurred in 11.5% of the patients with an ICP of 20 mm Hg or greater. Critchley et al$^8$ measured ICP and CPP during craniotomy for aneurysm clipping and found that PbtO$_2$ was improved when ICP was reduced and that the effect was unrelated to CPP. They also found that PbtO$_2$ was an indicator of cerebral ischemia in these patients.

Mannitol (an osmotic diuretic) has been routinely used to decrease ICP and improve CPP, but in a sample of 11 patients with severe head injury, Hartl et al$^9$ found that although ICP and CPP improved, PbtO$_2$ did not. The investigators concluded that ICP was not a surrogate measure of ischemic episodes.

Barbiturates have also been used to decrease elevated ICP. McKinley et al$^{10}$ reported the impact of barbiturate therapy on PbtO$_2$ in a study of 10 patients with severe head injury. In 3 of the patients, pentobarbital coma was initiated, resulting in an increase in PbtO$_2$.

In most patients with severe head injury, strategies to increase CPP and decrease ICP improve PbtO$_2$. The studies underscore the need for individualization of care based on each patient’s response to therapy.

### Influence of Temperature

Decreasing body temperature to less than 37°C, that is, inducing hypothermia, can decrease oxygen utilization in the brain.$^{11}$ In contrast, in a multicenter study of the effects of hypothermia in severe traumatic brain injury, outcome after 6 months did not differ significantly between patients who had hypothermia induced and those who did not.$^{12}$

As discussed earlier, several authors$^{13,14}$ reported differences between brain and body temperatures. Anecdotally, many clinicians have found that increases in temperature are accompanied by a decrease in PbtO$_2$.

### Conclusion

Interventions that alter FIO$_2$, PacO$_2$, CPP, hemoglobin level, ICP, and temperature and use of medications such as barbiturates can affect oxygen delivery and/or consumption in brain tissue. The key is striking a balance between all of the parameters to ensure an adequate PbtO$_2$.

PbtO$_2$ monitoring adds a dimension to care of patients with severe traumatic brain injury. Research has provided a basis for interventions that can affect critical oxygen levels in the brain. By understanding the causes of hypoxia and low oxygen states in the brain and planning interventions to adjust oxygen delivery, the critical care team can maximize patients’ recovery from injury.


Brain Tissue Oxygen Monitoring in Severe Brain Injury, I: Research and Usefulness in Critical Care

Objectives:
1. Discuss the dynamics of brain injury related to oxygen
2. Identify the influence of interventions on brain tissue oxygen
3. Describe the use of brain tissue oxygen monitoring in severe brain injury

Mark your answers clearly in the appropriate box. There is only 1 correct answer. You may photocopy this form.

1. [ ] Agree [ ] Neutral [ ] Disagree
2. [ ] Agree [ ] Neutral [ ] Disagree
3. [ ] Agree [ ] Neutral [ ] Disagree
4. [ ] Agree [ ] Neutral [ ] Disagree
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1. How many cases of traumatic brain injury are reported each year in the United States?  
   a. 80,000  
   b. 500,000  
   c. 100,000,000  
   d. 1,500,000

2. Which one of the following is not associated with secondary brain injury?  
   a. Cerebral edema  
   b. Cerebral ischemia  
   c. Chemical changes associated with direct trauma  
   d. Disruption of axons with primary injury

3. What is the correct percentages of brain-to-fluid ratio within the brain?  
   a. 60% brain tissue, 20% blood, 20% cerebrospinal fluid  
   b. 70% brain tissue, 20% blood, 10% cerebrospinal fluid  
   c. 80% brain tissue, 10% blood, 10% cerebrospinal fluid  
   d. 90% brain tissue, 5% blood, 5% cerebrospinal fluid

4. What is the location of probe placement for brain tissue oxygen monitoring?  
   a. Grey matter  
   b. Posterior ventricle  
   c. Deep white matter  
   d. Central culcus

5. After brain injury, what is the relationship of brain temperature to core body temperature?  
   a. Brain temperature is higher than core body temperature  
   b. Core body temperature is higher than brain temperature  
   c. Temperatures are similar  
   d. Temperatures are identical

6. What is the response in the partial pressure of brain tissue oxygen (PbtO2) to a fever?  
   a. Pressure increases  
   b. Pressure decreases  
   c. No change  
   d. Pressure initially increases, then decreases

7. What is the level of PbtO2 in an uninjured brain?  
   a. 20 mm Hg to 35 mm Hg  
   b. 15 mm Hg to 20 mm Hg  
   c. 10 mm Hg to 20 mm Hg  
   d. 5 mm Hg to 10 mm Hg

8. What level of PbtO2 is associated with an increased risk of death?  
   a. Greater than 15 mm Hg  
   b. Less than 15 mm Hg  
   c. There is no association  
   d. None of the above

9. Which one of the following is not a strategy to increase PbtO2?  
   a. Increase cerebral perfusion pressure (CPP)  
   b. Decrease intracranial pressure  
   c. Increase mean arterial blood pressure  
   d. Decrease body temperature to less than 37°C

10. What level of CPP causes a decrease in PbtO2?  
    a. Greater than 60 mm Hg  
    b. Less than 60 mm Hg  
    c. CPP has no effect on PbtO2  
    d. None of the above

11. Which one of the following measures does not result in a decrease in intracranial pressure or an increase in CPP?  
    a. Drainage of cerebrospinal fluid  
    b. Administration of mannitol  
    c. Sedation  
    d. Decreasing mean arterial blood pressure

12. With brain injury, placement of a brain oxygen monitor in which cerebral hemisphere allows for the detection of global oxygen status?  
    a. Contralateral  
    b. Superior  
    c. Inferior  
    d. Ipsilateral