Research related to the effects of certain interventions on brain tissue oxygen monitoring was presented in part I: “Research and Usefulness in Critical Care.” In this article, we continue the discussion of brain tissue oxygen monitoring and its effects on patients with severe traumatic brain injury. First, we describe how the interventions of critical care teams are integrated to maintain adequate brain oxygenation as patients progress through the critical phases of hospitalization. Second, we define critical-thinking algorithms that allow the team to implement sequenced interventions to maintain patients with traumatic brain injury in the zone of control. Last, we apply that theoretical and practical information to an actual case study, allowing readers to follow a case and analyze the interventions used to optimize a patient’s recovery.

Putting It All Together

The Guidelines for the Management of Severe Brain Injury describe evidence-based treatment that includes a discussion of appropriate management of intracranial pressure (ICP) and cerebral perfusion pressure (CPP). An algorithm outlines a tiered approach to avoid secondary injury. The literature review in the guidelines and a review of the Traumatic Coma Data Bank clearly indicate that the avoidance of early hypoxia and hypotension and a rapid resuscitation and evacuation of intracranial hematomas are necessary and could improve the chances for a good outcome.
Data presented in the guidelines suggest that a CPP of 70 mm Hg or greater is sufficient for adequate perfusion of neuronal cells in the absence of other brain pathology. The guidelines may enable patients to derive even greater benefit from a targeted approach to therapies that includes monitoring the partial pressure of oxygen in brain tissue (PbtO$_2$) because traumatic brain injury can be such a multifaceted problem.

Meeting the Needs of the Injured Brain

Resuscitating patients with severe brain injury and stabilizing and maintaining their condition require an organized critical care team armed with protocols that define interventions to optimize cerebral oxygenation and reduce ICP. Targeting therapy to maintain ICP at less than 20 mm Hg has been the reference standard for years. With the introduction of methods for monitoring oxygen levels in brain tissue, interventions must take into account this important new variable. The goal of the critical care team is to balance ICP and PbtO$_2$ throughout the critical phases of patients’ hospitalization. In the following section, we present interventions that increase PbtO$_2$ and integrate them into the overall management of patients with severe traumatic brain injury.

Resuscitation Phase: Emergency Department Through Operating Room Procedures

The team’s focus on brain resuscitation begins in the emergency department and continues through diagnostic radiology and operating room procedures. During this time, invasive monitors for evaluating ICP and PbtO$_2$ are not in place. Establishing physiological goals for each phase will assist the team in targeting therapies to each patient’s situation until more definitive treatment can be delivered (Table 1).

Initial interventions in the emergency department include protecting the airway and supporting oxygenation and ventilation. Patients admitted with a score between 3 and 8 on the Glasgow Coma Scale (GCS) require airway protection with endotracheal intubation. Stabilization of the cervical spine during the airway manipulation is essential. Because laryngeal manipulation during intubation has untoward effects on ICP, use of a rapid-sequence intubation regimen allows the team to administer medications and sequence airway maneuvers in such a way as to com-

<table>
<thead>
<tr>
<th>Table 1 Management of resuscitation phase in patients with severe head injury</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Emergency department</strong></td>
</tr>
<tr>
<td>Establish/maintain airway</td>
</tr>
<tr>
<td>Use 100% FIO$_2$ via BVM</td>
</tr>
<tr>
<td>Intubate for GCS score of 3-8 or if patient unable to protect airway</td>
</tr>
<tr>
<td>Use RSI protocol</td>
</tr>
<tr>
<td>Establish 2 IV catheters, obtain blood samples for laboratory studies</td>
</tr>
<tr>
<td>Place nasogastric tube/Foley catheter</td>
</tr>
<tr>
<td>Give mannitol 0.25-1.0 g/kg IV if posturing, unequal or nonreactive pupils</td>
</tr>
<tr>
<td>Goals: Sa$<em>{O2}$ 100%, Pac$</em>{O2}$ 35-40 mm Hg MAP $&gt; 90$ mm Hg</td>
</tr>
<tr>
<td>Drain CSF until ICP $&gt; 20$ mm Hg</td>
</tr>
<tr>
<td>Use propofol as needed for ↑ ICP</td>
</tr>
<tr>
<td>Consider craniectomy for ↑ ICP</td>
</tr>
<tr>
<td>Goals: Sa$<em>{O2}$ 100%, Pac$</em>{O2}$ 35-40 mm Hg, Pbt$<em>{O2}$ $\geq$ 20 mm Hg, Sj$</em>{O2}$ $&gt; 55%$, ICP $&lt; 20$ mm Hg</td>
</tr>
<tr>
<td>Use propofol as needed for ↑ ICP</td>
</tr>
</tbody>
</table>

BVM indicates bag-valve-mask; CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; CVP, central venous pressure; DIC, disseminated intravascular coagulation; FIO$_2$, fraction of inspired oxygen; GCS, Glasgow Coma Scale; Hct, hematocrit; ICP, intracranial pressure; IV, intravenous/intravenously; MAP, mean arterial pressure; NS, isotonic sodium chloride solution; Pbt$_{O2}$, partial pressure of oxygen in brain tissue; PCWP, pulmonary capillary wedge pressure; RBCs, red blood cells; RSI, rapid-sequence intubation; Sa$_{O2}$, arterial oxygen saturation; Sj$_{O2}$, oxygen saturation in jugular vein.

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plete the procedure while minimizing increases in ICP. By combining the sequenced timing of the medications and airway intubation, elevations in ICP can be decreased or minimized. Assessment of the correct placement of the endotracheal tube and the adequacy of oxygenation and ventilation is essential. The fraction of inspired oxygen (FiO2) should be maintained at 100% until respiratory parameters can be assessed definitively via arterial blood gas analysis. The PaCO2 greatly affects the ICP and the size of the cerebral vasculature. A PaCO2 less than 35 mm Hg produces cerebral vasoconstriction, reducing ICP, but may lead to decreases in oxygen delivery to the brain. Therefore, every effort should be made to maintain the PaCO2 between 35 and 45 mm Hg during the early resuscitation period until the team can assess how decreasing PaCO2 will affect Pbto2.

Restoration of adequate blood pressure in the emergency department is important for maintaining perfusion to the brain. Patients who experience episodes of hypotension in the early phases of trauma have more morbidity and mortality than do patients whose blood pressure is normal. Therefore, every effort should be made to provide adequate fluids in the early phases to maintain a mean arterial pressure greater than 90 mm Hg. Multiple interventions (eg, physical examination, initial radiography, and placement of catheters) are done during this phase in an attempt to identify primary and secondary injuries and to provide a means to intervene with and monitor the patient.

Rapidly obtaining radiographic studies, including computed tomography (CT) scans of the brain and radiographs of the cervical spine, chest, and pelvis, assists in detecting injuries. Once severe brain injury has been diagnosed on the basis of findings on clinical examination and radiographic evidence, the patient is taken to the operating room for definitive intervention.

Once again, the goals of the operative phase must be understood by the entire team (Table 1). Placement of an ICP monitor and a brain tissue oxygen monitor as well as central venous monitoring assists the team in meeting the needs of the injured brain during this critical phase. Specific operative interventions, craniotomy for evacuation of acute hemorrhages or craniectomy, help reduce ICP and improve cerebral oxygenation. Careful attention to coagulopathies that develop intraoperatively is important. Prompt attention to correcting the specific deficiency must occur, with administration of blood products, platelets, fresh-frozen plasma, and/or cryoprecipitate.

Because of the possibility of multiple operative procedures and a prolonged period in the operating room, it is helpful to have the intensive care unit (ICU)/trauma nurse present during this phase. This nurse works with the anesthesiologist by helping to intervene when ICP and Pbto2 change rapidly. This nurse also assists with interventions and critical documentation so that an accurate record is kept of all of the interventions that occur during the operating room phase.

The hemodynamic status of many patients is unstable postoperatively. During transfer to the ICU, it is helpful to use a battery-operated ventilator. Doing so ensures that the ventilator mode remains stable. If a manual resuscitation bag is used, the person administering breaths may be inconsistent in delivering the cor-

<table>
<thead>
<tr>
<th>Medication</th>
<th>Category</th>
<th>Goal/rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine 1.0-1.5 mg/kg</td>
<td>Local anesthetic</td>
<td>Assist with blunting increase in intracranial pressure during manipulation</td>
</tr>
<tr>
<td>Etomidate 0.3 mg/kg</td>
<td>Anesthesia inductant</td>
<td>Produce induction of anesthesia without a decrease in blood pressure; decreases intracranial pressure; lasts 8-10 minutes</td>
</tr>
<tr>
<td>Vecuronium 0.1 mg/kg</td>
<td>Paralytic</td>
<td>Relax skeletal muscle to allow manipulation of airway for intubation, reduce elevations in intracranial pressure</td>
</tr>
<tr>
<td>Morphine sulfate 1-2 mg or 0.05-0.1 mg/kg or fentanyl 0.5-1 µg/kg</td>
<td>Narcotic</td>
<td>Decrease cerebral metabolism and provide analgesia, blunt cardiovascular responses to intubation</td>
</tr>
<tr>
<td>Midazolam 1-2 mg or 0.05 mg/kg or lorazepam 1 mg or 0.05-0.1 mg/kg</td>
<td>Sedative</td>
<td>Provide ongoing sedation once etomidate wears off, to reduce anxiety, especially in patients with neuromuscular blockade</td>
</tr>
</tbody>
</table>
rect number of breaths per minute, a situation that can lead to hypocapnia or hypercapnia.

ICU Phase The stabilization period, the first few hours in the ICU, is a critical period during which the patient must be quickly brought into the zone of control. Members of the critical care team must understand the effect that each intervention has on PbtO₂ and ICP. Use of a standardized protocol with team interventions provided via standardized physician orders will help the team prioritize and orchestrate interventions while continuing to stabilize the patient’s condition (Table 3).

PbtO₂ is influenced by multiple factors (Table 4). Critical care nurses must understand how to manipulate each factor and the appropriate sequence in which to initiate changes in order to balance oxygen delivery to the brain and ICP. Maintaining these therapies until brain swelling is reduced and brain oxygenation stays within the normal range is the primary focus of critical care nurses.

When the patient arrives from the operating room, the team rapidly confirms that the ventilatory support is correct, connects all hemodynamic monitors, assesses the patient’s condition, and determines the priorities in care. During the admission, the ICU nurse should ask if a CT scan of the brain was obtained postoperatively to verify location of the LICOX catheter. This question is important because in order to obtain meaningful readings, the catheter must be positioned in viable tissue. If the catheter is in a contusion, a blood clot, or nonviable brain parenchyma, then the reliability of the readings obtained via the catheter are questionable. Once the patient is settled, then the team’s priorities are established. During this initial ICU phase, the ICU nurse also quickly assesses the following parameters: PbtO₂, mean arterial pressure, ICP, CPP, pulmonary capillary wedge pressure,
central venous pressure, cardiac index, oxygen saturation, PaCO₂, and body temperature. The team must bring these values under control. The goals for this phase are to maintain PbtO₂ greater than 20 mm Hg and ICP less than 20 mm Hg.

If the PbtO₂ is less than 15 mm Hg, the team should ensure that the FiO₂ administered via the mechanical ventilator is 100%, even if the oxygen saturation is 100%, because the driving force that moves oxygen into tissue is the gradient between the dissolved oxygen in the blood and brain tissue oxygen levels. Supranormal levels of oxygen may be needed for the first few hours during stabilization in the ICU. The FiO₂ can be slowly decreased once the PbtO₂ is greater than 20 mm Hg and stable. Next, ICP and PaCO₂ are assessed. These 2 variables must be considered together when their effect on PbtO₂ is evaluated. If the ICP is less than 20 mm Hg and PbtO₂ is less than 20 mm Hg, the PaCO₂ is allowed to increase up to 45 mm Hg so long as the ICP remains less than 20 mm Hg. If ICP is greater than 20 mm Hg and PbtO₂ is less than 20 mm Hg, cerebrospinal fluid is drained if a ventriculostomy was done. Increasing the PaCO₂ might worsen the ICP, but it will increase the PbtO₂. In efforts to increase PbtO₂, ICP is considered in relation to mean arterial pressure and CPP. If the CPP is less than 70 mm Hg, efforts should be made to increase the mean arterial pressure. If the pulmonary capillary wedge pressure is less than 8 mm Hg, a bolus of isotonic sodium chloride solution or 250 mL of 5% albumin should be given. If the hematocrit is less than 0.33, packed red blood cells are administered. Then, the patient’s fluid volume status should be reassessed. Fluids should be given until the pulmonary capillary wedge pressure is greater than 8 but less than 12 mm Hg. If the patient is euvoelastic and the CPP is still low, a vasopressor may be given.

Next, the PbtO₂, CPP, and ICP are reevaluated. If PbtO₂ is greater than 20 mm Hg and ICP is less than 20 mm Hg, the CPP should be maintained at that level. The minimal CPP requirement of patients with severe traumatic brain injury will change and must be evaluated frequently. Once CPP is optimized, the ICP and PbtO₂ are reevaluated. If the ICP is still greater than 20 mm Hg, 0.25 to 1.0 g/kg mannitol may be given as an intravenous bolus and/or propofol may be infused intravenously at a rate of 10 to 100 µg/kg per minute. During this time, administration of analgesic and sedative agents should be started and then maintained continuously. Cooling measures should be used to maintain the patient’s body temperature at 36°C to 37°C.

If PbtO₂ is greater than 20 mm Hg on admission to the ICU, assessment of ICP and the other factors will provide information for planning strategies for care. If PbtO₂ and ICP are normal, then the patient’s condition can be stabilized by using an FiO₂ of 30% to 40% and a PaCO₂ of 35 to 40 mm Hg, and efforts can be made to

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### Table 4 Team interventions and PbtO₂

<table>
<thead>
<tr>
<th>Factors that decrease PbtO₂</th>
<th>Ways to increase PbtO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue hypoxia</td>
<td>Increase fraction of inspired oxygen</td>
</tr>
<tr>
<td>Decreasing PaCO₂</td>
<td>Increase PaCO₂ through ventilator changes</td>
</tr>
<tr>
<td>Decreased CPP related to decreased mean arterial pressure</td>
<td>Increase CPP</td>
</tr>
<tr>
<td>Low hemoglobin level/hematocrit</td>
<td>Administer packed red blood cells until hematocrit is 0.33</td>
</tr>
<tr>
<td>Increased ICP</td>
<td>Drain cerebrospinal fluid until ICP &lt; 20 mm Hg</td>
</tr>
<tr>
<td>Increased body temperature</td>
<td>Decrease temperature with cooling measures</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Evaluate and treat: pneumonia, acute respiratory distress syndrome, pulmonary edema, or pleural effusions</td>
</tr>
<tr>
<td>Cardiac/hemodynamic</td>
<td>Evaluate hemodynamic profile and treat</td>
</tr>
<tr>
<td></td>
<td>Check preload (PCWP/central venous pressure)</td>
</tr>
<tr>
<td></td>
<td>Check afterload (systemic vascular resistance)</td>
</tr>
<tr>
<td></td>
<td>Check stroke volume/left ventricular stroke work index for contractility</td>
</tr>
<tr>
<td></td>
<td>Check cardiac enzyme levels, 12-lead electrocardiogram</td>
</tr>
</tbody>
</table>

CPP indicates cerebral perfusion pressure; ICP, intracranial pressure; PbtO₂, partial pressure of oxygen in brain tissue; PCWP, pulmonary capillary wedge pressure.
determine the optimal CPP. A CPP of 55 to 60 mm Hg may be acceptable if the PbtO$_2$ is greater than 20 mm Hg without supplemental increased oxygen support. The fluid volume status should be assessed, and euvolemia should be maintained. Administration of analgesic and sedative agents should begin. The patient’s condition should be evaluated frequently.

If ICP is elevated but PbtO$_2$ is normal, then efforts should be made to reduce ICP. These efforts include draining cerebrospinal fluid, decreasing PaCO$_2$ so long as the PbtO$_2$ is greater than 20 mm Hg, optimizing the mean arterial pressure with fluids and vasopressors, and administering sedatives, analgesic agents, and/or an osmotic diuretic. If the ICP remains greater than 20 mm Hg, consultation with the physician team may lead to an emergent CT scan, orders for propofol and/or barbiturates, and, possibly, a craniectomy. The optimal CPP for a patient with an elevated ICP and normal PbtO$_2$ must be determined and may exceed the “recommended” CPP of 70 mm Hg. In some instances, patients might require a CPP of 90 to 110 mm Hg to decrease the ICP.

Once the patient’s condition is stabilized and the parameters have reached the recommended goals for therapy, then the actions of the critical care team must be organized to keep the situation under control. Rarely does a patient continue without elevations of ICP or decreases in PbtO$_2$. Therefore, the ICU nurse must continually reassess the patient and reprioritize the interventions with other team members. The ICU team usually develops a sense of where the patient’s various parameters should be in order to maintain all parameters within the goal estab-

Table 5  Critical-thinking algorithms for treatment of patients with severe traumatic brain injury

<table>
<thead>
<tr>
<th>PbtO$_2$ &gt; 20-40 mm Hg</th>
<th>PbtO$_2$ &lt; 20 mm Hg</th>
<th>PbtO$_2$ &lt; 20 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICP &gt; 20 mm Hg</td>
<td></td>
<td>ICP &lt; 20 mm Hg</td>
</tr>
</tbody>
</table>

Drain CSF
↓ PaCO$_2$ until ICP < 20; stop ↓ when PbtO$_2$ < 20
Optimize CPP for patient
Use fluids to optimize PCWP, then vasopressors to ↑ CPP
√ analgesia/sedation: ↑ Morphine/oral morphine
↑ Propofol and adjust
Give mannitol 0.25-1.0 g/kg
Call physician
CT scan if ICP > 20 mm Hg

Drain CSF
↑ PaCO$_2$ slowly until ICP ↑ > 5-10 mm Hg
Place patient on 100% FiO$_2$ for 5-15 min
Optimize CPP for patient:
1. Use fluids to optimize CVP/PCWP and
2. Vasopressors
√ analgesia: ↑ morphine if needed
↑ Propofol/adjunct 10-100 µg/kg per minute
Give mannitol 0.25-1.0 g/kg IV
Call physician
CT scan if ICP doesn’t remain < 20 mm Hg

√ PaCO$_2$: if low then ↑ to 40-45 mm Hg as long as ICP stays within range
If still low, place patient on 100% FiO$_2$ for 5-15 min
√ lung sounds/ventilator settings/CVP/PCWP/CI/MAP

1. Hemodynamic evaluation
   √ PCWP/CVP: if ↓ fluids
   √ H/H: transfuse if < 0.33
   √ MAP/SVR/LVSWI
   a) If SVR < 1000, start neo/dopamine
   b) If SVR > 1500, recheck vasopressors
   c) If PVR ↑ and Levophed used, wean off Levophed

2. Pulmonary evaluation
   Consult RCP
   √ vent settings
   √ need for sedation
   √ need for paralytic
   √ need for treatments
   Change vent settings instead of high FiO$_2$
   Evaluate I:E status

3. Fluid overload right pulmonary edema/volume overload
   a) √ I/O balance
   b) √ Chest X-ray
   c) √ PVR/LVSWI
   d) Call physician
   e) Consider Lasix

4. Temperature management: Keep 36-37°C and individualize for each patient

*Refer to decision tree in ICP < 20 mm Hg and PbtO$_2$ < 20 mm Hg for critical analysis of hemodynamic vs pulmonary causes

Cl indicates cardiac index; CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; CT, computed tomography; CVP, central venous pressure; FiO$_2$, fraction of inspired oxygen; H/H, hemoglobin/hematocrit; ICP, intracranial pressure; I:E, inspiration:expiration; IV, intravenous; LVSWI, left ventricular stroke work index; MAP, mean arterial pressure; neo, phenylephrine; PbtO$_2$, partial pressure of oxygen in brain tissue; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RCP, respiratory care practitioner; SV, stroke volume; SVR, systemic vascular resistance; vent, ventilator.

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lished for the patient. Using the interventions listed in Table 3 ensures an organized approach to patients’ management.

Critical-thinking algorithms are extremely helpful in guiding team members in decision making (Table 5). The interventions are prioritized and allow the team to think through the situation in a standardized manner, ensuring that primary interventions are done before more complex interventions. The patient’s status will change. Sometimes the changes are minute to minute in the early phases of care and day to day in the later phases.

Supporting the body’s systems during all phases of the patient’s hospitalization is imperative. Aggressively managing the pulmonary system to prevent complications requires frequent suctioning, position changes, and the use of kinetic therapy. Gastric secretions are drained with a nasogastric or orogastric tube. Early enteral or parenteral nutrition should begin within 48 hours of admission to the ICU. Application of compression stockings and boots reduces the risk of deep vein thrombosis. The patient’s feet should be maintained in an upright position by a rigid device or high-topped tennis shoes to prevent foot drop. Mobility of extremities can be enhanced by using proper positioning of the legs or arms and using passive range of motion exercises. The risk of skin breakdown is reduced by keeping the skin clean and dry and repositioning the patient frequently.

After peak edema has passed, the patient’s intensity of care will diminish. Once the patient has achieved a consistently normal ICP and PbtO2 with little need for interventions, then the team can begin to wean the patient off therapy (Figure 1). The more complex interventions are discontinued first, and the patient is slowly brought out of the coma state.

**Applying Brain Oxygen Monitoring: Case Study**

Applying the theoretical information presented is integral to the transition of research findings from theory to the clinical setting. In the following case study, we strive to integrate theory into practice.

A.P., a 29-year-old woman, was driving home from work at 6:10 PM when she crossed the center line and hit an oncoming car. A.P. was not wearing a seat belt, and her car did not have an air bag. Witnesses called 911. Paramedics arrived at 6:20 PM and described A.P.’s condition as critical. After a prolonged extrication,
A.P.’s clinical examination revealed the following: airway open, breathing shallow; weak pulses/unable to obtain a palpable blood pressure; GCS score 1 for eye opening, 1 for motor response, and 1 for verbal response elicited with painful stimuli; obvious facial, pelvic, and left leg deformities. Initial interventions at the scene were completed. After the base station was contacted, A.P. was designated a critical trauma patient and was sent to the nearest trauma center.

Emergency Department to Radiology Department

At 7:01 PM, A.P. arrived in the emergency department in critical condition. Paramedics were supporting her airway by using a manual resuscitation bag and 100% oxygen. Because of her depressed respirations, inability to maintain a patent airway, and a GCS score of 3, A.P. was emergently intubated by using a rapid-sequence intubation. She was supported with 100% FIO2, and the end-tidal carbon dioxide level (PetCO2) was maintained at 35 mm Hg. Circulation assessment revealed weak pulses of 140/min and a systolic blood pressure of 70 mm Hg. Paramedics had started 1 intravenous catheter. A large-bore intravenous catheter was started by using a rapid-infusion kit and was attached to a rapid-infuser pump to allow intravenous administration of fluids and blood products. A.P.’s neurological status included a GCS score of 3; both pupils were 5 mm in diameter and nonreactive. An oral gastric tube to decompress stomach contents, a Foley catheter for urinary drainage, and an arterial catheter for monitoring blood pressure were inserted. Her hematocrit was 0.25. Diagnostic radiographs revealed fractures of the pelvis, left femur, and left tibia/fibula. By the completion of the 20-minute emergency department phase, she had received 1 L of isotonic sodium chloride solution and 4 units of O-negative packed red blood cells.

A.P. was transported by the trauma team to the radiology department for an emergent CT scan at 7:20 PM. A battery-operated ventilator was used during transport to maintain consistent PetCO2 levels. The unenhanced CT scan of the brain revealed cerebral edema, a small right-sided subdural hematoma, right-to-left shifting of intracranial contents, and multiple facial fractures (Figure 2). Fluid resuscitation continued because of her low blood pressure (80-90 mm Hg systolic). The goal was to achieve a mean arterial pressure of at least 90

![Figure 2 Unenhanced CT scans of A.P.’s brain and face show injuries.](http://ccn.aacnjournals.org/Downloaded from)
mm Hg. A.P. received an additional 5 units of packed red blood cells and 4 units of fresh-frozen plasma. Neurosurgical consultation resulted in a decision to transport her immediately to the operating room.

**Operating Room Phase**

The trauma surgeon quickly inserted a pulmonary artery catheter for continuous measurement of cardiac output and a jugular bulb oxygen saturation catheter. The mean arterial pressure ranged from 80 mm Hg at the beginning of the case to 50 to 60 mm Hg during the first hours. A.P.’s coagulation profile revealed a low fibrinogen level and the presence of fibrin split products. The anesthesiologist and trauma ICU nurses worked diligently, transfusing multiple units of red blood cells, platelets, fresh-frozen plasma, and cryoprecipitate into A.P. Phenylephrine was administered intravenously in an attempt to meet the goal of a mean arterial pressure of 90 mm Hg.

The neurosurgical operative team assisted the neurosurgeon with placement of an ICP catheter and a LICOX brain tissue oxygen catheter. The initial ICP was 35 mm Hg, a considerable elevation from the normal value of less than 20 mm Hg. The PbtO₂ was 3 mm Hg, a critical level (normal PbtO₂ is 20 to 35 mm Hg). During the next 30 minutes, the ICP increased to 50 mm Hg and the PbtO₂ stayed at 3 mm Hg. The team was using multiple interventions during this period in an effort to reduce ICP and improve PbtO₂. Team members administered an FiO₂ of 100%, maintained the PetCO₂ at 35 mm Hg, administered analgesic agents and mannitol, and attempted to increase the mean arterial pressure to greater than 80 mm Hg but were successful only for a short period.

The neurosurgeon elected to perform a craniectomy when the team was unable to control the ICP. By the time the bone was removed, the ICP had reached 60 mm Hg. As the bone was removed and the subdural hematoma evacuated, the ICP decreased to 8 or 10 mm Hg and the PbtO₂ increased dramatically to 20 to 45 mm Hg. Because of the unstable pelvic fracture and A.P.’s critical condition, hoping to stabilize the fractures, the orthopedic surgeon placed a C-ring on the pelvis. Pins were placed in the leg fractures for traction to be applied once the patient reached the ICU. A.P. had a large 7-cm laceration across her forehead, so the plastic surgeon quickly repaired it. The physician team elected not to repair the facial fractures because of A.P.’s critical condition. By the time the operative procedures were completed, the ICP had once again increased to more than 40 mm Hg, and the PbtO₂ had decreased to 2 mm Hg (Figure 3). Despite multiple interventions, the team could not increase the PbtO₂ to more than 2 mm Hg or decrease the ICP to less than 40 mm Hg. A.P.’s condition was highly unstable and grave.

After the operative procedures, A.P. was transported to the trauma/neurological ICU for definitive care. A battery-operated ventilator was used during transport. The presence of multiple intravenous pumps for...
delivering fluids and vasopressors and the orthopedic/neurosurgical equipment complicated the transport. The ICU team received the report from the trauma team. Two ICU nurses were assigned to manage A.P.’s care.

ICU Phase

Initial 24 Hours A.P.’s neurological condition was grave. Her GCS score was 1 (eye opening), 1 (motor response), and 1 (verbal response), and both pupils were 6 mm in diameter and nonreactive. Her ICP was greater than 40 mm Hg, and her PbtO2 was between 2.5 and 3.5 mm Hg. The ventilator was set to maintain an Fio2 of 40% and a pressure setting and rate to maintain the PetCO2 at 35 mm Hg. Intravenous administration of fluids and vasopressors was continued. In addition to phenylephrine, dopamine was added and titrated in an effort to increase the CPP to 70 mm Hg. Because the ICP values were between 40 and 50 mm Hg, the maximum CPP achieved was 40 to 59 mm Hg. The team was unable to bring A.P. into the zone of control or meet the goals that had been established for this phase, which were maintaining an ICP less than 20 mm Hg and a PbtO2 greater than 20 mm Hg.

Within hours of A.P.’s admission to the ICU, the team continued to struggle. The ICU nurse and respiratory care practitioner decided to increase the Fio2 to 70%, a change that resulted in an increase of the PbtO2 to 6.2 mm Hg within 30 minutes. The Fio2 was increased to 100%, and the PbtO2 increased to 23 mm Hg within 25 minutes of that change. Once the target PbtO2 was reached, the team focused on decreasing the ICP (Figure 4). Despite aggressive fluid and vasopressor therapy, the CPP remained in the range of 40 to 50 mm Hg, lower than the goal of 70 mm Hg. Epinephrine was added to the phenylephrine and dopamine in an attempt to increase CPP.

The ICP stayed between 40 and 50 mm Hg. Mannitol was administered intravenously, and the dose of propofol was gradually increased to 100 µg/kg per minute intravenously. Cerebrospinal fluid was drained at least 4 times per hour in an effort to reduce ICP. Despite all these interventions, the ICP did not decrease.

Because the PbtO2 was greater than 20 mm Hg, the team sought to decrease the Fio2 from 100%. Each time the Fio2 was decreased, the PbtO2 decreased precipitously to less than 5 mm Hg. Use of 100% Fio2 was resumed immediately to keep the PbtO2 greater than 20 mm Hg. Full systemic support was instituted.

The interventions in the first 24 hours were focused on optimizing oxygen levels and lowering ICP. Although the PbtO2 goal was achieved, the ICP was dangerously elevated to 40 to 60 mm Hg. The team was encouraged by the ability to maintain PbtO2 but frustrated with the ICP. A.P. was in for a rough hospital course.

Days 2 to 26 The team continued to focus their interventions on decreasing the ICP to less than 20 mm Hg and maintaining the PbtO2 at greater than 20 mm Hg. During day 2, the ICP remained between 40 and 50 mm Hg despite drainage of cerebrospinal fluid, maintenance of PetCO2 at 28 to 30 mm Hg, aggressive CPP therapy, repeated adminis-

Figure 4 Course of events from 12 PM to 7 PM during the first 24 hours of team management for A.P. in the intensive care unit.

CPP indicates cerebral perfusion pressure, which is mean arterial pressure minus ICP; Dopa, dopamine hydrochloride; Epi, epinephrine; ICP, intracranial pressure (mm Hg); Inc, increase; Neo, phenylephrine; PbtO2, partial pressure of oxygen in brain tissue (mm Hg).
tration of mannitol boluses, and use of high doses of propofol intravenously. The CPP was maintained between 50 and 60 mm Hg with fluid therapy and maximum dosages of dopamine, phenylephrine, and epinephrine as intravenous infusions. At one point, the PbtO₂ decreased to 12.4 mm Hg despite the FIO₂ remaining at 100%. A.P.’s hematocrit was 0.24, so the surgeons ordered 2 units of packed red blood cells. The CPP increased by only 5 mm Hg, but the PbtO₂ increased from 12.4 to 22 mm Hg. After the red cells were transfused, PbtO₂ fluctuated when the FIO₂ was decreased to less than 100%. Despite several attempts to decrease the FIO₂ on day 2, it appeared that high-flow FIO₂ was the only intervention that would keep the PbtO₂ greater than 20 mm Hg. No change in A.P.’s neurological status occurred: both pupils remained 7 mm in diameter and fixed. No motor movement or doll’s eyes, blink, or gag reflex was detected.

On day 3, a CT scan of the brain revealed an epidural collection of blood underneath the scalp on the side where the craniectomy had been done. Despite A.P.’s critical state and poor prognosis, the team pressed on with care. The neurosurgeon took A.P. back to the operating room and evacuated the hematoma. The ICP decreased from values between 40 and 50 mm Hg to values between 20 and 30 mm Hg (Figure 5). CPP improved and was maintained at greater than 70 mm Hg with ICP values from 15 to 25 mm Hg. Therapies were continued in an attempt to keep A.P. in the zone. By day 6, small changes in the neurological and pulmonary systems indicated a small ray of hope. A.P.’s right pupil began to react sluggishly to light, and she withdrew her right arm to a central pain stimulus. By day 7, A.P.’s pulmonary status had improved. The inverse inspiratory/expiratory ratio therapy was stopped, and the FIO₂ was at 50%. Her ICP was controllable and was maintained at less than 20 mm Hg with drainage of cerebrospinal fluid. She required less vasopressor support and had been weaned from the
intravenous infusions of metaraminol, norepinephrine, and epinephrine. The most interesting phenomenon was related to the PaCO2 levels needed to maintain PbtO2 greater than 20 mm Hg. Because A.P.’s ICP was low, the team allowed the PaCO2 to increase until the PbtO2 was maintained consistently at greater than 20 mm Hg. The PaCO2 ranged from 45 to 50 mm Hg during this time. Her neurological status continued to improve; both pupils reacted to light and were equal in diameter at 4 mm.

A.P.’s status continued to improve, and the neurosurgeon began weaning her off therapies. Weaning was done in an orderly fashion to minimize relapses or elevations in ICP (Figure 1). During the course of several days, A.P. was weaned off all vasopressors. She went to the operating room on day 11 for repair of the pelvic and leg fractures. On day 12, the LICOX catheter was removed. A.P.’s GCS score was 4 (eye opening), 5 (motor response), and 1 (verbal response). She was moving both upper extremities spontaneously, with 4/5 motor strength on the right side and 2/5 motor strength on the left side. On day 14, A.P. went back to the operating room for a tracheostomy, placement of gastric and jejunostomy tubes, and repair of the facial fractures. Physical, occupational, and speech therapies were started. By day 22, A.P. was weaned from the ventilator, and she was transferred to the surgical unit by day 26.

A.P. continued to improve. She began turning her head and tracking nurses and her family as they moved around the bed. A.P. started to follow commands. She was up in a bedside chair at least 3 times a day. Any time she was out of bed, she used a helmet to protect her head where the bone had been removed. As A.P. continued to improve, she was evaluated for acute rehabilitation. A.P. was transferred to acute rehabilitation on day 50 and was discharged home on day 78.

At 3 months, A.P. underwent a cranioplasty and the bone was replaced. She returned home able to walk, talk, dress, eat, and participate in life’s activities. It has been 18 months since A.P.’s accident, and she is independent in her activities of daily living.

Outcomes Associated With Integrated Care: Oxygen Monitoring/Traumatic Brain Injury Guidelines

The outcomes of patients with severe traumatic brain injury have been greatly improved through the use of ICP/brain oxygen monitoring and the application of the Brain Trauma Foundation/American Association of Neurologic Surgeons’ Guidelines for the Management of Severe Head Injury.2,20 At Mission Hospital, Mission Viejo, Calif, all outcomes for patients with severe traumatic brain injury are tracked for at least 6 months after the patients are discharged from the hospital. In order to maintain consistency, inclusion and exclusion criteria were established. The inclusion criteria included the following: GCS scores of 3 to 8, blunt mechanism of injury, abnormal findings on CT scan and/or clinical examination, and age 8 years or greater. Exclusion criteria were penetrating trauma, pronounced brain dead within 24 hours of ICU admission, age less than 8 years, and normal findings on CT scan and presence of alcohol/drug intoxication, seizure, or a non–central nervous system injury leading to depressed level of consciousness. Since we instituted the guidelines for traumatic brain injury and brain oxygen monitoring in the forms of catheters in the jugular bulb and the brain, the outcomes have improved dramatically (Table 6).

Conclusion

Optimizing care for patients with critical brain injuries requires a well-orchestrated team that understands the physiology behind the injury and agrees to work together to achieve the goals established at

<table>
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<tr>
<th>Outcome</th>
<th>Before guidelines in use</th>
<th>Percentage of patients</th>
<th>After guidelines in use</th>
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<tbody>
<tr>
<td></td>
<td>(No brain tissue oxygen monitoring)</td>
<td></td>
<td>(SjO2 and brain tissue oxygen monitoring)</td>
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<tr>
<td>Good outcome to moderate disability</td>
<td>27</td>
<td>75.8</td>
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<tr>
<td>Severe disability to persistent vegetative state</td>
<td>30</td>
<td>11.6</td>
<td></td>
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<tr>
<td>Death</td>
<td>43</td>
<td>12.6</td>
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each phase of illness. Brain oxygen monitoring adds another variable to consider when choosing interventions to manage patients with severe traumatic brain injury. By integrating brain tissue oxygen levels into the overall management of these patients, team members will be able to enhance the care delivered.

References
