2 INJURIES TO THE CENTRAL NERVOUS SYSTEM

Marike Zwienenberg-Lee, M.D., Kee D. Kim M.D., and J. Paul Muizelaar, M.D., Ph.D.

It is estimated that each year, two million patients present to physicians with a primary or secondary diagnosis of head injury. Of these patients, approximately 400,000 are admitted and 70,000 die, most of traumatic brain injury. Thus, brain injury can be considered epidemic. Neurosurgeons, who number 4,000 in the United States, are probably best trained to manage patients with severe head injuries, but initial resuscitation and stabilization are usually performed by emergency department physicians, general surgeons, and trauma surgeons. These professionals are the ones who can make a difference for patients: current understanding of the pathophysiology of traumatic brain injury indicates that treatment during the first few hours is critical and often determines outcome.

Nonetheless, the importance of care immediately after resuscitation and in the ensuing days is not to be underestimated. Patients with multiple system injuries often receive care in surgical intensive care units under the supervision of a general surgeon. Less than optimal management at an early stage has a greater impact overall because of the larger number of patients involved, but less than optimal management at later stages, even in mildly injured patients, has a much more dramatic impact. Initial recovery, followed by relentless decline attributable to insufficient cerebral perfusion, is not an expected outcome. Although we cannot promote healing of the brain by pharmacologic means, we can prevent secondary injury to the brain by ensuring adequate cerebral circulation and oxygenation.

The reported incidence of spinal cord injury in the United States ranges from 29 to 53 per million.1–3 About 50% of the injuries are related to motor vehicle accidents, 15% to 20% to falls, 15% to 20% to interpersonal violence, and the remaining 15% to 20% to sports and recreational activity. In general, the group at highest risk is between 16 and 30 years of age, not unlike the group at highest risk for head injuries. Most of those injured are males: several studies report that the percentage is approximately 75%.4 Between 45% and 50% of patients with spinal cord injury have other injuries that seriously affect their prognosis.5

The cervical spine is most often involved in spinal cord injury. A major study of trauma outcome, conducted from 1982 to 1989, revealed that the cervical spinal cord was involved in 55% of cases of acute injury, the thoracic spinal cord in 30%, and the lumbar spinal cord in 15%.6 In an analysis of 358 patients with spinal cord injury, complete neurologic injury occurred in 78% of the 71 cases of thoracic injury, 60% of the 202 cases of cervical injury, and 65% of the 85 cases of thoracolumbar injury.7 Average direct costs of spinal cord injury (including hospitalization, rehabilitation, residence modification, and long-term care) are tremendous. In 1992, it was estimated that lifetime costs (in 1989 dollars) were $210,379 for a paraplegic and $571,854 for a quadriplegic.8

Initial resuscitation and evaluation of injured patients are discussed more fully elsewhere [see 7: Life-Threatening Trauma]. In this chapter, we outline approaches to the management of severe head injury and acute spinal cord injury. In addition, we address the pathophysiology of such injuries to provide the reader with the understanding required for making appropriate decisions about diagnosis and treatment of injured patients [see Discussion, below].

Head Injury

EMERGENCY DEPARTMENT MANAGEMENT

Because hypoxia and hypotension interfere with cerebral oxygenation, complete and rapid physiologic resuscitation is the highest priority for patients with head injuries. A large study from the Traumatic Coma Data Bank demonstrated that a single observation of systolic blood pressure below 90 mm Hg in the field or hypoxia (arterial oxygen tension [PaO2] < 60 mm Hg) was a major predictor of poor outcome.9 A multidisciplinary team should provide the patient with an adequate airway and ventilation (intubation, ventilation, and detection of hemothorax or pneumothorax) and restore and maintain hemodynamic stability (with adequate fluid replacement and detection and treatment of any bleeding), all according to the principles developed by the Advanced Trauma Life Support system.10 The ABCs of emergency care (Airway, Breathing, and Circulation) take precedence, irrespective of neurologic injuries. The initial neurologic assessment, which should take no more than a few seconds, consists of rating the patient’s level of consciousness on the Glasgow Coma Scale (GCS) [see Table 1] and assessing the width and reactivity of the pupils. Although the same assessment is made after resuscitation as a guide for prognosis and therapy, it should also be made (and recorded) before resuscitation to permit evaluation of the effect of

<table>
<thead>
<tr>
<th>Test</th>
<th>Response</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye opening (E)</td>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>To verbal command</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Best motor response (arm) (M)</td>
<td>Obedience to verbal command</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Localization of painful stimulus</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Flexion withdrawal response to pain</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Abnormal flexion response to pain (decorticate rigidity)</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Extension response to pain (decerebrate rigidity)</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Best verbal response (V)</td>
<td>Oriented conversation</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Disoriented conversation</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Total (E + M + V)</td>
<td></td>
<td>3–15</td>
</tr>
</tbody>
</table>
resuscitative measures and differentiation between primary and secondary neurologic injury.

Early orotracheal intubation and ventilation are recommended for patients with a GCS score of 8 or lower or a motor score of 4 or lower. Other indications for immediate intubation are loss of protective laryngeal reflexes and ventilatory insufficiency, as manifested by hypoxemia ($P_{O_2} < 60$ mm Hg), hypercarbia (arterial carbon dioxide tension [$P_{CO_2}$] $> 45$ mm Hg), spontaneous hyperventilation (causing $P_{CO_2} < 26$ mm Hg), and respiratory arrhythmia. Indications for intubation before transport are deteriorating consciousness (even if the patient is not in a coma), bilateral fractured mandible, copious bleeding into the mouth (as occurs with fracture of the base of the skull), and seizures. An intubated patient must also be ventilated ($P_{CO_2} = 35$ mm Hg).

Fluid replacement should be performed with isotonic solutions such as normal saline, lactated Ringer solution, or packed red blood cells when appropriate. Glucose-based solutions should be avoided in the acute phase. The patient should be examined rapidly and thoroughly for any concomitant life-threatening injuries.

Patients with spinal cord injury above T5 may have severe hypotension as a result of vasogenic spinal shock. Aggressive treatment is indicated, including volume resuscitation and administration of alpha-adrenergic vasopressors. Intracranial hypertension should be suspected if there is rapid neurologic deterioration. Clinical evidence of intracranial hypertension, manifested by signs of herniation, includes unilateral or bilateral dilatation of the pupils, asymmetrical pupillary reactivity, and motor posturing.

Intracranial hypertension should be treated aggressively. Hyperventilation, which does not interfere with volume resuscitation and results in rapid reduction of intracranial pressure (ICP), should be established immediately in cases of pupillary abnormalities. It has been demonstrated that unilateral or bilateral pupillary abnormalities do not result only from compression of the third cranial nerves, as was previously thought, but also derive from compression of the brain stem, with resulting brain stem ischemia. Therefore, administration of mannitol is effective because it not only decreases ICP but also increases cerebral blood flow (CBF) through modulation of viscosity. Because mannitol is not used to dehydrate the body, all fluid losses through diuresis must be replaced immediately or even preventively, especially in patients suffering shock as a result of blood loss. Arterial hypertension occurring after a severe head injury may reflect intracranial hypertension (Cushing’s phenomenon), especially when accompanied by bradycardia; it should not be treated, because it may be the sole mechanism permitting the brain to maintain perfusion despite increasing ICP.

In the absence of signs of herniation, sedation should be used when required for safe and efficient transport of the patient. Transport time should be kept to a minimum because transport is often accompanied by secondary insults (e.g., hypoxia or hypotension). Pharmacologic paralysis, which interferes with neurologic examination, should be used only if sedation alone is inadequate for safe and effective transport and resuscitation. When pharmacologic paralysis is used, short-acting agents are preferred. Prophylactic hyperventilation, which may exacerbate early ischemia, is not recommended for these patients. Guidelines for the resuscitation and initial treatment of patients with severe head injuries have been established that facilitate management [see Figure 1].

Minimal radiologic evaluation consists of a lateral cervical spine film or a swimmer’s view [see Spinal Cord Injury, below]. After hemodynamic stability is achieved, unenhanced computed tomography of the head should be performed in all patients with persistent impairment of consciousness. In patients with a GCS of 14 or 15 who have experienced transient loss of consciousness or have posttraumatic amnesia, head CT is probably necessary only in the presence of certain specific signs and symptoms [see Table 2].

### Table 2

<table>
<thead>
<tr>
<th>Patient has head injury</th>
<th>Patient does not have surgical lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resuscitate according to ATLS principles.</td>
<td>Admit patient to neurosurgical ICU.</td>
</tr>
<tr>
<td>Rate patient on Glasgow Coma Scale.</td>
<td>Monitor ICP, and perform jugular bulb oximetry.</td>
</tr>
<tr>
<td>Perform emergency diagnostic or therapeutic procedures as indicated.</td>
<td>Treat intracranial hypertension.</td>
</tr>
<tr>
<td>If GCS score $\leq 8$ or motor score $\leq 4$, intubate and ventilate; goals should be $S_{O_2} = 100%$, $P_{CO_2} = 35$ mm Hg, systolic BP $&gt; 90$ mm Hg.</td>
<td>Maintain CPP $&gt; 60$ mm Hg.</td>
</tr>
<tr>
<td>Rate patient again on GCS for assessment of effects of resuscitation.</td>
<td></td>
</tr>
</tbody>
</table>

### Figure 1

Shown is an algorithm for initial management of the patient with a severe head injury.
In many clinical studies indicate that ICP monitoring is useful for early detection of intracranial mass lesions; that it allows calculation, which is the case when the cerebral metabolic rate of oxygen (CMRO2) is low.

**Jugular bulb oximetry** CBF is an important determinant of neurologic outcome, and the arterial–jugular venous oxygen difference (A-VDO2) is an important indicator of the adequacy of CBF. Monitoring of therapy by measuring CBF and A-VDO2 would be ideal, but there is no practical way of doing this directly and continuously.

An estimate of global A-VDO2 can be obtained from simultaneous monitoring of arterial oxygen saturation (Sao2) and Sjvo2. Jugular venous oxygen saturation is monitored by percutaneous retrograde insertion of a fiberoptic catheter in the internal jugular vein, with the tip of the catheter located in the jugular bulb. The catheter is usually inserted into the jugular vein with the dominant cerebral venous drainage (the right jugular vein in 80% to 90% of the population), but some prefer to insert the catheter at the site of the most significant brain damage. A-VDO2 is calculated according to the following formula:

\[
A-VDO2 = (Sao2 - Sjvo2) \times 1.34 \times Hb + \frac{[Pao2 - Pjvo2] \times 0.0031}{Hb + \frac{Sao2 - Sjvo2}{1.34}}
\]

The contribution of the variables within the brackets, which is small, is usually ignored for practical purposes. Because calculation of A-VDO2 requires the drawing of blood samples, it can be done only intermittently.

For continuous monitoring, Sjvo2, the result of arterial oxygen input and cerebral extraction, is used. In normal individuals, Sjvo2 ranges from 50% to 70%. If Sjvo2 values below 50% last for more than 15 minutes, they are considered desaturations, resulting from insufficient arterial oxygenation (Sao2), inadequate oxygen-carrying capacity (Hb concentration), or, when arterial saturation and oxygen-carrying capacity are normal, from inadequate CBF. A 1994 study described a relation between the occurrence of desaturations and neurologic outcome in patients with severe head injuries [see Table 3]. Without desaturation, mortality was 16%; with one documented desaturation, 42%; and with multiple desaturations, 70%. High Sjvo2 values indicate low oxygen extraction, which is the case when the cerebral metabolic rate of oxygen (CMRO2) is low.

The limitations of jugular bulb oximetry should be kept in mind when Sjvo2 values are interpreted. Because Sjvo2 represents global oxygenation, regional ischemia may go undetected if the ischemic region is too small to be represented in the total hemispheric Sjvo2 value. Ischemia may also occur in a part of the brain being drained by the opposite jugular vein. In addition, extracerebral veins drain into the internal jugular vein approximately 2 cm below the jugular bulb. With low flow values, significant extracere-

### Table 2 Constitutional Signs and Symptoms Necessitating Follow-up Nonenhanced Head CT Scan in Patients with Loss of Conscience and GCS Score of 14 or 15

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>45</td>
</tr>
<tr>
<td>Somnolence</td>
<td>39</td>
</tr>
<tr>
<td>Mental-status changes or confusion</td>
<td>32</td>
</tr>
<tr>
<td>Nausea or vomiting</td>
<td>20</td>
</tr>
<tr>
<td>Seizure</td>
<td>10</td>
</tr>
</tbody>
</table>

*Defined as jugular venous oxygen saturation < 50% for longer than 15 min.

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**Jugular Desaturation* and Outcome in 116 Patients with Severe Head Injuries**

<table>
<thead>
<tr>
<th>Jugular Desaturations (No.)</th>
<th>Good Recovery/ Moderate Disability (%)</th>
<th>Severe Disability/ Vegetative (%)</th>
<th>Deceased (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>45</td>
<td>39</td>
<td>16</td>
</tr>
<tr>
<td>1</td>
<td>26</td>
<td>32</td>
<td>42</td>
</tr>
<tr>
<td>&gt;1</td>
<td>10</td>
<td>20</td>
<td>70</td>
</tr>
</tbody>
</table>

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**ACS Surgery: Principles and Practice**

2 injuries to the CNS — 3

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brial contamination may occur, resulting in deceptively high \( S_O_2 \) values. Finally, artifactual readings are often encountered as a result of reduced light intensity when the catheter lodges against the vessel wall. Technical improvements in catheters, however, have markedly reduced the number of artifacts observed.

**Tissue oxygen monitoring**  Tissue oxygen monitoring has been employed for years in Europe to treat patients with brain injuries but was not approved by the Food and Drug Administration for use in the United States until relatively recently. Tissue oxygen tension (\( P_{O_2} \)) in the brain is measured via a fiberoptic monitor locally inserted through a separate bur hole.\(^{23}\) The oxygen pressure recorded thus reflects a combination of oxygen supply and cerebral oxygen extraction, which makes interpretation of the values less than straightforward. Nevertheless, several investigators have demonstrated a relation between low local tissue \( O_2 \) values and poor outcome and have documented improved cerebral oxygenation after introduction of resuscitative measures (e.g., optimization of CPP and blood oxygenation).\(^{25}\) In normal persons, \( P_{O_2} \) ranges from 25 to 30 mm Hg. Critical thresholds for \( P_{O_2} \) values include prolonged and repeated episodes below 10 to 15 mm Hg and any episode below 5 mm Hg.\(^{26}\)

**Management of Cerebral Perfusion Pressure**

The rationale behind CPP therapy is expressed in Poiseuille’s law [see Discussion, Pathophysiology, below]. Although the effect of CPP therapy has not been investigated in a randomized, controlled clinical trial, several studies suggest that a CPP of 70 to 80 mm Hg may be the clinical threshold below which mortality and morbidity increase.\(^{27-29}\) In addition, there are now class II data (not yet published as of December 2003) indicating that maintenance of a CPP higher than 60 mm Hg is sufficient to ensure optimal cerebral perfusion and oxygenation (personal communication, American Brain Injury Consortium).

CPP therapy involves manipulation of both arterial BP and ICP; but its objective is the reduction of ICP. If ICP reduction does not achieve a CPP of 60 mm Hg, arterial hypertension is instituted. Mean arterial BP should be raised first by optimizing volume status: ample fluids, including albumin (25 to 30 ml/hr), are administered to maintain central venous pressure at 5 to 10 mm Hg. A pulmonary arterial catheter is suggested for patients older than 50 years and for individuals with known cardiac disease, multiple trauma (particularly chest or abdominal injuries), or a need for vasopressors or high-dose barbiturates. Pulmonary arterial wedge pressure (PAWP) should be maintained between 10 and 14 mm Hg. If necessary, an alpha-adrenergic drug (e.g., phenylephrine, 80 mg in 250 or 500 ml of normal saline) can be combined with the fluids.

**Management of Intracranial Pressure**

Because ICP is a determinant of CPP, treatment of ICP inevitably affects CPP. Given that the goal is maintenance or improvement of CBF, measures for treating ICP should be evaluated in the light of their effect on CBF. It is not possible to establish an arbitrary threshold for treatment of elevated ICP that would be applicable in all situations. Any interpretation of ICP must be combined with assessment of clinical features and evaluation of CT scan findings. It is possible, for example, to have transtentorial herniation with an ICP of 15 mm Hg in the presence of a mass lesion. Conversely, with diffuse brain swelling, adequate CPP can be maintained despite an ICP as high as 30 mm Hg. As a general rule, ICP values between 20 and 25 mm Hg indicate that therapy should be initiated.

The recommended regimen for treatment of ICP starts with drainage of CSF through a ventriculostomy-ICP catheter and continues as necessary in a stepwise fashion with sedation, paralysis, osmotic therapy, hyperventilation, induction of a metabolic coma, and decompressive surgery [see Figure 2].

Although drainage of CSF has no documented deleterious side effects, it does have the potential to aggravate brain shift. Therefore, only a minimal amount should be drained, sufficient to bring the ICP below 20 mm Hg.

Sedation with morphine sulfate, 2 to 5 mg/hr I.V., is standard treatment; fentanyl, lorazepam, and midazolam are commonly used alternatives. In some centers, propofol is now used for routine sedation. Propofol has a short half-life, which is advantageous for the purposes of neurologic evaluation, but it is expensive and can have deleterious side effects after prolonged use (i.e., > 48 to 72 hours).\(^{30}\)

Muscular paralysis is employed by many clinicians as the next step in therapy. Its major downside is that it renders neurologic examination pointless, except for assessment of the pupillary response. In addition, the risk of respiratory complications is increased with neuromuscular blockade.

Mannitol is usually administered in I.V. boluses of 0.25 to 1 g/kg over 10 to 15 minutes until either ICP is controlled or serum osmolarity reaches 320 mOsm/L. It now appears, however, that higher doses (e.g., 1.4 g/kg) may be more effective.\(^{16,17}\) Because volume depletion is an important side effect of mannitol therapy, urine losses should be replaced. Hypertonic saline (3% to 10%) may be used in place of mannitol: it appears to be just as effective as or even more effective than mannitol for ICP control, especially in higher concentrations (e.g., 7.5%).\(^{31}\) In addition, hypertonic saline is not associated with volume depletion but actually increases intravascular volume. The effect probably is not purely osmotic but is partly related to viscosity, as is the case with mannitol.\(^{32}\)

As noted, hyperventilation reduces ICP (by vasoconstriction) and CBF, which may be at ischemic levels in certain parts of the brain. Therefore, hyperventilation (\( P_{CO_2} < 30 \text{ mm Hg} \)) should not be instituted prophylactically but should be reserved for acute decompensation and employed as a short-term temporizing measure until more definitive therapy can be instituted. If \( P_{CO_2} \) must be reduced to extremely low levels, hyperventilation can be combined with mannitol, thereby improving CBF by reducing blood viscosity. Jugular bulb oximetry is recommended in these situations because it will determine how much the cerebral vessels can be constricted.

**Hemoglobin, Hematocrit, and Blood Viscosity**

The hematocrit and viscosity are inversely related, and a balance must be established to optimize oxygenation. If the hematocrit is too high, viscosity increases; if the hematocrit is too low, the oxygen-carrying capacity of blood decreases. Maintaining the hematocrit between 0.30 and 0.35 is recommended: below 0.30, oxygen-carrying capacity falls without a significant change in viscosity, and above 0.35, viscosity increases out of proportion to oxygen-carrying capacity.\(^{33}\) Preferably, blood that has been banked for less than 2 weeks should be used for transfusion. There are some data indicating that the effect of transfusion on cerebral oxygenation is linearly related to the duration of storage of packed red cells.\(^{34}\)

**Brain Protection**

When oxygen delivery cannot be sufficiently improved, the brain can be protected by decreasing CMRO\(_2\). Barbiturates appear to protect the brain and lower ICP through several mechanisms,
including alteration of vascular tone, suppression of metabolism, and inhibition of free radical lipid peroxidation. The most important effect may involve coupling of CBF to regional metabolic demands, so that the lower the metabolic requirements, the lower the CBF and the related cerebral blood volume (CBV), with subsequent beneficial effects on ICP and global cerebral perfusion. Barbiturate therapy (usually pentobarbital to a blood level of 4 mg/L) is instituted when other measures to control ICP fail. In one series of 25 patients with an ICP higher than 40 mm Hg, barbiturates not only controlled ICP but also improved outcome. Of the patients whose ICP was controlled by barbiturates, 50% had a good recovery; of the patients whose ICP was not controlled, 83% died. In another trial, prophylactic barbiturate therapy failed to improve neurologic outcome. Etomidate, a rapidly acting agent with hypnotic properties similar to those of barbiturates, has fewer adverse effects on systemic BP or ICP. However, it suppresses adrenocortical function, and its solvent, propylene glycol, can cause renal insufficiency. Propofol is a sedative hypnotic with a rapid onset and a short duration of action. It depresses CMRO₂, but not as effectively as barbiturates and etomidate do. Studies of patients with head injuries have demonstrated that ICP decreases with administration of propofol, but systemic BP usually decreases as well, resulting in a net decrease in CPP. Blood lactate levels do not increase when propofol is administered, indicating that cerebral oxygenation is adequate. If propofol is used, correction of hypovolemia is recommended to prevent hypotension associated with bolus injection. Finally, because of its preservative-free, lipid-base vehicle, there is an increased risk of bacterial or fungal infection, and the high caloric content (1 kcal/ml) may be problematic during a prolonged infusion.

Hypothermia produces a balanced reduction in energy production and utilization, decreasing CMRO₂ and CBF proportionally. Protocols for hypothermia include cooling to 32º to 33º C (89.6º to 91.4º F) within 6 hours of injury and maintenance of this temperature for 24 to 48 hours. Hypothermia to 33º C has been shown to be effective for the control of refractory high ICP. Two pilot clinical trials reported improved neurologic outcome, but a multicenter randomized clinical trial failed to demonstrate any overall improvement. Side effects of therapy, which in this case mainly included medical complications in the elderly patients, resulted in the absence of a treatment effect. In addition, rewarming of patients who were hypothermic on admission appeared to be detrimental. However, a subgroup of patients who were already hypothermic on admission did benefit from continued treatment with hypothermia, and these patients were subsequently enrolled in a second phase III trial (NABISH-II).

The main side effects of hypothermia are cardiac arrhythmias and coagulation disorders, reported after cooling to 32º to 33º C. Other drawbacks of hypothermia include the difficulty of detect-
Spinal Cord Injury

DIAGNOSIS AND INITIAL MANAGEMENT

In the field, all patients with significant trauma, any trauma patients who lose consciousness, and any patients suffering minor trauma who have complaints referable to the spine or the spinal cord should be treated as if they had a spinal cord injury until proven otherwise [see Figure 3]. If cardiopulmonary resuscitation is necessary, it takes precedence [see 7.1 Life-Threatening Trauma]. The objectives are to maintain adequate oxygenation and maintain BP by administering fluids and vasopressors. The main concerns of management in the field are immobilization before and during extrication from a vehicle (or removal from the scene of another type of accident) and immobilization during transport to prevent active or passive movement of the spine. Subsequently, the patient may require a rigid Philadelphia collar, support from sandbags and straps, a spine board, or a log-roll for turning. A brief motor examination may detect possible deficits.

When the patient arrives at the hospital, care should be taken to provide adequate oxygenation, prevent hypotension, and maintain immobilization. Patients with an injury above C4, who may have respiratory paralysis, may need ventilatory assistance. Lesions above T5 may be accompanied by loss of sympathetic tone and consequently by significant venous pooling and arterial hypotension. Because paralytic ileus is common, usually lasting several days, a nasogastric tube should be placed to prevent vomiting and aspiration. Urinary retention is also a common occurrence, and a Foley catheter should therefore be inserted. Vasomotor paralysis may cause poikilothermia (uncontrolled temperature regulation), and normothermia should therefore be maintained.

A detailed neurologic examination is required to determine whether the injury is complete or incomplete and at what level of the spinal cord the injury occurred. If possible, a history should determine the mechanism of injury (e.g., hyperflexion, extension, axial loading, or rotation). The American Spinal Injury Association (ASIA) (www.asia-spinalinjury.org) has developed a protocol for sensory and motor examination of patients with spinal cord injuries that is precise and relatively easy to follow [see Figure 4].

Spinal shock consists of loss of all or most motor, sensory, and autonomic function below the level of the lesion. It usually develops in the setting of a severe spinal cord injury that occurs over a brief period, and it is most commonly witnessed immediately after the injury (though it may also appear hours later in cases of progressive injury). As originally defined, the term spinal shock referred to arterial hypotension resulting from loss of sympathetic tone with normal alignment, (2) deterioration occurs, (3) fracture level deficit level, or (4) a bony injury cannot be identified.

Patient has suspected cervical spinal injury

[See Figure 5.]

Patient has suspected thoracolumbar spine injury

[See Figure 6.]

Figure 3 Shown is an algorithm for management of the patient with an acute spinal cord injury.

Spinal shock should be differentiated from spinal concussion. Spinal concussion is a poorly understood phenomenon. It is defined as partial spinal cord sensory or motor deficits that resolve completely within 24 to 72 hours and that are never associated with permanent spinal cord injury. Spinal concussion is rare and has never been described in conjunction with spinal shock.

All patients with possible spine injuries should be examined radiologically. Roentgenography of the cervical spine with the patient in a rigid collar includes a lateral view showing both the craniovertebral and the cervicothoracic (C7-T1) junction. If the lateral view is normal and the patient is coherent and has no neck tenderness or neurologic deficit, the collar can be removed for anteroposterior and odontoid views. If the lower cervical spine or the cervicothoracic junction is not well visualized, a lateral view with caudal traction on the arms, or a swimmer’s view, is required. If areas of the spine are still not visualized or if there is a neurologic deficit, a CT scan with sagittal reconstruction should be obtained through the poorly visualized levels.

The use of flexion-extension films (i.e., testing of the active range of motion of the cervical spine from maximal anterior-posterior flexion to extension) is typically limited to patients who are
awake and able to cooperate. Although spinal instability is probably best demonstrated with this type of imaging, it should be noted that in patients with neck pain, muscle spasm can limit range of motion and thereby mask a subluxation. Accordingly, it is recommended that patients with posttraumatic neck pain who are neurologically intact, whose plain radiographs are normal, and who are capable of limited flexion and extension effort (i.e., < 30° of motion) be placed in a rigid cervical collar and reevaluated 1 to 2 weeks later. In comatose patients, dynamic films are sometimes obtained under direct fluoroscopic guidance, though magnetic resonance imaging appears to render such studies unnecessary.

CT is particularly helpful in the further evaluation of fractures diagnosed on plain films: it shows bone in greater detail and at higher resolution than plain films do, it achieves better visualization of fixed subluxations, and it allows more accurate assessment of the central bony canal and the neuronal foramina. CT is highly accurate in visualizing body fractures, Jefferson (C1) and hangman (C2) fractures, and bilateral locked facets. It appears to be less accurate, however, in visualizing transverse C2, posterior element, and nondisplaced spinous process fractures. Moreover, it sometimes misses superior articular process fractures. MRI of the cervical spine is the study of choice for evaluating injury to the soft tissues and ligaments. Intrinsic cord damage (e.g., edema, hematoma, or contusion) and injury to the surrounding ligaments, disks, and paravertebral soft tissues are well visualized. In addition, MRI is helpful in the assessment of brachial plexus injury, though a series of special coronal images is usually required. A fatsuppression image usually identifies ligamentous injury to the posterior elements quite well, and fine-cut gradient echo (GRE) imaging of the transverse ligament of C2 is extremely sensitive in detecting disruption. The major drawbacks of MRI are the length of the imaging time, which may be a problem in the critically ill trauma patient, the susceptibility to movement artifacts, and the need for MRI-compatible monitoring devices and traction equipment. Moreover, in this setting, it is often difficult to obtain an adequate medical history with regard to implanted medical devices or old bullet fragments, both of which preclude MRI. Radiologic evaluation and clearance of the cervical spine can be facilitated by the use of an established protocol.

STANDARD NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY

**MOTOR**

<table>
<thead>
<tr>
<th>C2</th>
<th>R</th>
<th>C2</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Elbow flexors</strong></td>
<td>R</td>
<td><strong>Elbow extensors</strong></td>
<td>L</td>
</tr>
<tr>
<td><strong>Wrist extensors</strong></td>
<td>R</td>
<td><strong>Finger flexors (distal phalanx of middle finger)</strong></td>
<td>L</td>
</tr>
<tr>
<td><strong>Finger abductors (little finger)</strong></td>
<td>R</td>
<td><strong>T1</strong></td>
<td>L</td>
</tr>
<tr>
<td><strong>T2</strong></td>
<td>R</td>
<td><strong>T2</strong></td>
<td>L</td>
</tr>
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**SENSORY**

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**VERSION OF THE AMERICAN SPINAL INJURY ASSOCIATION**

**Neurological Motor**

**Sensory**

**Complete or Incomplete?**

**Zone of Partial Preservation**

This form may be copied freely but should not be altered without permission from the American Spinal Injury Association.

**Figure 4** Shown is a form developed by the American Spinal Injury Association to record the principal information about motor, sensory, and sphincter function required for accurate neurologic classification of spinal cord injury. For the motor examination, 10 key muscles are tested (left). For the sensory examination, 28 key dermatomes are tested (right).
Anteroposterior and lateral views of thoracic and lumbosacral vertebrae should be obtained for all trauma patients who were thrown from a vehicle or fell more than 2 m to the ground, complain of back pain, are unconscious, cannot reliably describe back pain or have altered mental status preventing adequate examination, or have an unknown mechanism of injury or other injuries that suggest the possibility of spinal injury. If a fracture or subluxation is found on the plain films, a CT with sagittal reconstruction extending from one level above the fracture/subluxation to one level below it is recommended.

Indications for urgent MRI include the following: an incomplete lesion with normal alignment (to rule out the possibility of compression); deterioration (worsening deficit or rising level); a fracture level different from the level of deficit; and inability to identify a bony injury (to rule out the possibilities of soft tissue compression, disk herniation, or hematoma that would necessitate surgery). Radiologic evaluation of patients with suspected thoracolumbar spine injuries is also facilitated by use of a protocol [see Figure 6].

In most patients, the spine can be cleared on the basis of plain x-rays and neurologic examination. Obtunded or comatose patients whose plain x-rays are normal but who are at high risk for spine injury (e.g., those injured in high-speed motor vehicle accidents or by falls from great heights) should be evaluated by a spine specialist before spine clearance.

**TREATMENT**

**Traction**

The objectives of craniocervical traction are to reduce fracture-dislocations, to maintain normal alignment or immobility of the cervical spine, to prevent further injury, to decompress the spinal cord and roots, and to facilitate bone healing. A common technique is placement of Gardner-Wells tongs, a U-shaped device with pins that are anchored to the skull just above the pinna. Traction is applied with the patient in supine position by adding weights to the traction ring. Alternatively, traction may be applied with the patient in a halo ring. A special traction triangle is then added to the ring. The advantage of this approach is that the patient can be stabilized in a halo vest as soon as reduction is obtained. Furthermore, the halo vest is helpful in dealing with a highly unstable spine that requires instrumentation because the patient can be easily turned onto the operating table without the risk of significant movement of the spine.

Traction should always be applied under strict neurologic monitoring. If the patient’s condition deteriorates when the weight is increased, the additional weight should be removed and the patient should immediately undergo imaging (e.g., with plain films, MRI, or both). In the case of a highly unstable fracture, traction should be guided by fluoroscopy rather than serial x-rays.

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**Figure 5** Shown is an algorithm depicting the protocol for radiologic evaluation and clearance of cervical spinal cord injury.
Pharmacologic Treatment

A number of drugs are known to interfere with the processes of secondary injury. The challenge is to identify the most effective treatment or combination of treatments with the fewest severe side effects—a task requiring many experiments for each treatment tested. Methylprednisolone (MP), thought to act by scavenging free radicals, has been reported to be neuroprotective in patients with spinal cord injuries.\textsuperscript{48-50} Considerable controversy remains, however, regarding the clinical benefit of MP administration after acute spinal cord injury.

Three multicenter, randomized, controlled clinical trials carried out in the United States evaluated MP in this setting: National Spinal Cord Injury Study (NASCIS) I, NASCIS II, and NASCIS III.\textsuperscript{48-50} In NASCIS I, reported in 1984, the administration of a 100 mg MP bolus followed by 100 mg/day for 10 days was compared with administration of a 1,000 mg MP bolus followed by 1,000 mg/day for 10 days. There was no placebo group. No difference was noted between the two MP groups with respect to motor or sensory outcome at 6 weeks, 6 months, and 1 year.

Because data from animal studies suggested that the MP doses used in NASCIS I were too low to yield a significant difference in outcome, NASCIS II was initiated in 1985. In this trial, administration of high-dose MP (i.e., a 30 mg/kg bolus followed by 5.4 mg/kg/hour for 23 hours) was compared with administration of naloxone (a 5.4 mg/kg bolus followed by 4 mg/kg/hour for 23 hours) and with placebo. Neurologic outcome was graded by evaluating sensory and motor function at 6 weeks and 6 months after injury. A total of 14 muscle groups were examined and graded on a scale of 0 to 5. The scores were added, with a total score of 0 indicating no motor activity below the level of the lesion and a total score of 70 representing normal motor function. For sensory assessment, 29 spinal cord segments were similarly evaluated and graded on a scale of 1 to 3. A total score of 29 indicated no response in any segment, and a total score of 87 indicated that all sensory segments were normal. The authors reported a statistically significant mean motor and sensory improvement after MP administration. In the MP group, there was a mean improvement of 16.0 in the motor score, compared with a mean improvement of 11.2 in the placebo group. The mean changes in sensory scores were 11.4 (MP) and 6.6 (placebo) for sensation to pinprick and 8.9 (MP) and 4.3 (placebo) for sensation.

In NASCIS III, patients who received a 24-hour regimen of MP (a 30 mg/kg bolus followed by 5.4 mg/kg/hour), a similar 48-hour regimen, or a 48-hour regimen of tyrilazad mesylate (2.5 mg/kg every 6 hours) were evaluated. There was no placebo group. No differences in sensory or motor recovery were observed between groups. In a subgroup analysis, it was noted that in patients whose treatment was initiated more than 3 hours after injury, motor function improved more with the 48-hour MP regimen than with the 24-hour regimen. The Functional Independence Measurement (FIM) did not show any statistically significant differences between groups.

NASCIS II and III have been criticized for flaws in research design and data analysis. For example, patients with a normal motor examination and patients with a combined conus-cauda injury were included. Furthermore, only motor and sensory scores from the right side of the body were reported. Moreover, in the statistical analysis, the only statistically significant results were the result of a post hoc analysis.

A 2000 study presented a statistical reanalysis of the NASCIS II and III data.\textsuperscript{51} In this reanalysis, no difference in neurologic recovery between the placebo, 24-hour MP, and 48-hour MP groups was found, but an increased mortality was documented in the 48-hour MP group—a finding that was not reported in the NASCIS studies. Current guidelines take the position that the available medical evidence does not conclusively establish the existence of a significant clinical benefit from administration of MP for either 24 or 48 hours and that the harmful side effects may actually outweigh any clinical benefits.\textsuperscript{52} MP administration is therefore considered a treatment option to be used at the discretion of the treating physician.

The calcium channel blocker nimodipine causes significant increases in blood flow in the spinal cord,\textsuperscript{53,54} but, paradoxically, the dosage necessary to exert this effect is accompanied by significant systemic hypotension. Administration of GM\textsubscript{1} ganglioside, a compound that occurs naturally in the membranes of mammals and is particularly abundant in the CNS, has been shown to have short-term neuroprotective effects and long-term regenerative effects in animal models. In a prospective, placebo-controlled, double-blind study, some improvement in motor, sensory, and bladder function was seen in patients treated with GM\textsubscript{1} ganglioside, but no overall improvement in neurologic outcome was noted.\textsuperscript{55} This agent is currently an optional treatment for patients with spinal cord injuries. Treatment with GM\textsubscript{1} ganglioside is started after the completion of MP therapy and continued for 56 days.

**Figure 6** Shown is an algorithm depicting the protocol for radiologic evaluation and clearance of thoracolumbar spinal cord injury.
primary goals of treatment are to decompress and protect underlying neural structures, to restore spinal stability and alignment, to facilitate early mobilization and rehabilitation, and to maximize neurologic recovery.

There is general agreement among physicians that immobilization of the patient to prevent further injury and early stabilization of fractures and dislocations of the spine are necessary. The single widely accepted indication for early urgent surgical treatment is ongoing neurologic deterioration in the presence of spinal canal compromise from bone and disk fragments, hematoma, or unreduced subluxation. Surgical indications still under debate include incomplete spinal cord injury (with persistent spinal cord compression) and complete spinal cord injury with the possibility of some neurologic recovery.

In some studies, early surgical intervention has been associated with an increased risk of systemic complications (especially pulmonary complications) and neurologic deterioration. One group of investigators found that one third of all cases of neurologic deterioration could be attributed directly to surgical intervention; four of 26 patients who underwent spinal surgery within 5 days experienced deterioration, whereas none of the patients treated after 5 days had any neurologic sequelae. Other studies, however, have not found an increased risk of deterioration with early intervention. One study evaluated 110 patients with cervical spinal cord injury, of whom 88 underwent surgery for spinal stabilization; in the 39 patients treated within 24 hours, the incidence of systemic complications was reduced by 50% in comparison with the incidence in the 49 patients treated 24 hours to 3 weeks after injury. In addition, the incidence of neurologic deterioration was 0% in the early-stabilization group compared with 2.5% in the late-stabilization group. Data from NASCIS II showed improved outcome in patients undergoing surgery within 24 hours of injury compared with patients treated after 200 hours, but the difference was not statistically significant.

We adhere to the following protocol in treating patients with cervical spine fracture-dislocations. After systemic stabilization, patients are placed in a halo ring and traction as soon as possible. We prefer to set up traction in the neurologic ICU, but this can be done in the emergency department when necessary. Closed reduction is attempted under the guidance of fluoroscopy or serial x-rays, depending on the degree of spinal cord compromise or expected instability. Patients are then secured in their halo vests and undergo MRI evaluation, regardless of whether reduction attempts succeeded or failed. Patients with reduced and realigned injuries and no cord compromise are kept in their halo vests and offered nonurgent surgical stabilization, depending on the nature of the injury. Patients with irreducible fractures or continued compromise of the neural elements are taken to the OR promptly for urgent surgical decompression and fixation, regardless of whether the neurologic injury is complete or incomplete.

The efficacy of early surgical decompression in patients with thoracolumbar fractures is not established, except in cases where the neurologic examination reveals deterioration. Most surgeons, however, advocate urgent decompression in patients who have canal compromise and an incomplete injury; patients who do not fall into this category can be kept on strict spinal precautions, with definitive therapy instituted on a nonurgent basis.

Diagnosis and Treatment of Specific Fractures and Dislocations

Cervical spine Injuries to the cervical spine include atlas fractures, axis fractures, fractures of the lower cervical spine, and atlanto-occipital and atlantoaxial dislocations. Atlanto-occipital dislocation (AOD) is rare, occurring in approximately 1% of patients with cervical spine injuries. Many of these patients die immediately after trauma as a result of brain stem injury and respiratory arrest. Since the 1980s, advances in emergency patient management in the field, reduced transport time, and better recognition of the condition have improved the survival rate after AOD. Nevertheless, since 1966, fewer than 100 survivors of AOD have been reported in the literature. Patients who survive AOD often have neurologic deficits, such as lower cranial neuropathies, unilateral or bilateral weakness, and quadriplegia. Some 20% of patients, however, exhibit no abnormalities on neurologic examination at presentation. AOD is difficult to diagnose and is often missed on the initial cervical radiograph. Accordingly, a high index of suspicion for this condition should be maintained, particularly when signs such as prevertebral soft tissue swelling on the plain cervical radiograph or subarachnoid hemorrhage at the craniovertebral junction on CT are present. Additional investigation with MRI may be necessary.

Because AOD mostly involves ligamentous injury, treatment generally consists of operative fusion. Traction is rarely employed, because even a small weight may cause distraction injury with these highly unstable dislocations.

Atlantoaxial dislocations are often fatal as well. Like AODs, they are highly unstable lesions associated with severe ligamentous injury. Axis (Jefferson, or C1) fractures, which represent 5% to 10% of all cervical spine fractures, result from axial loading. Because of the large diameter of the spinal canal and the tendency of fragments to move outward, these fractures usually are not accompanied by significant neurologic injury. However, 40% of patients with an atlas fracture have another cervical fracture as well (usually involving C2). The integrity of the transverse ligament largely determines whether the fracture is stable. Injuries that involve the midportion of the transverse ligament or the periosteal insertion (e.g., types Ia and Ib) will not heal spontaneously and must be treated with surgical fixation of the C1-C2 complex. In contrast, type II injuries (e.g., avulsion injuries or comminuted lateral mass fractures) will usually heal in a rigid external orthosis (e.g., a halo vest). Most other atlas fractures can be managed in a rigid cervical collar, except for widely displaced or comminuted fractures, which also require that the patient be immobilized in a halo vest.

Axis (C2) fractures account for 10% to 20% of all cervical spine fractures in adults and 70% of cervical fractures in children. The odontoid process is the part of C2 that is most commonly fractured (accounting for 60% of axis fractures). The management of odontoid fractures remains controversial: only class III data are available, which are insufficient to establish practice guidelines.

Three different types of odontoid fractures are recognized. Fractures of the tip of the odontoid process (avulsion fracture, type I) are uncommon but are thought to be stable in most cases. They can be treated by using a hard cervical collar with or without preceding cervical traction or by immobilizing the patient in a halo vest.

Fractures of the neck (type II) and fractures at the junction of the odontoid process and the axis body (type III) are more common (accounting for 65% to 80% and 20% to 35% of odontoid fractures, respectively). Management of type II fractures depends on the degree to which the dens is displaced: fractures with more than 5 mm of displacement are typically managed surgically, whereas fractures with less than 5 mm of displacement can be treated nonsurgically with a halo vest or a semirigid orthosis (e.g., a sterno-occipito-mandibular immobilizer [SOMI] brace). The management of type II fractures also depends on the age and general medical condition of the pa-
tient. Halo-vest immobilization is associated with a significantly increased risk of pulmonary complications and death in elderly patients and thus should be used with caution in this population. Surgical stabilization and fusion is recommended for type II fractures in patients older than 50 years.60

The type IIa subcategory of odontoid fracture warrants special mention. These fractures have either anterior or posterior chip-fracture fragments at the base of the dens and are considered unstable. They are often widely displaced and have a nonunion rate of 75% to 85% with halo-vest immobilization. Accordingly, surgical management is recommended. The integrity of the transverse ligament should be evaluated in patients with type II injuries; fixation with an odontoid screw is contraindicated, and C1-C2 fixation should be performed instead.

Most type III odontoid fractures will fuse with rigid external mobilization (i.e., either cervical traction followed by placement in a rigid cervical collar or placement in a halo vest). Again, more than 5 mm of displacement is an indication for surgical stabilization.60

Traumatic spondylolisthesis, or hangman’s fracture, accounts for approximately 20% of C2 fractures. Injury usually results from axial compression in combination with hyperextension of the occipito-atlanto-axial complex on the lower spine, resulting in bilateral fracture of the pars interarticularis. Fractures affecting the ring of the axis without C2-C3 angulation are stable and can be treated with immobilization in a Philadelphia collar or a SOMI brace. Halo-vest immobilization is recommended in unreliable patients or patients with both C1 and C2 fractures. The average healing time is 12 weeks. Fractures with angulation, subluxation, or C2-C3 locked facets are treated with halo-vest immobilization if they are adequately reducible and with surgical intervention if they are nonreducible, are associated with disruption of the C2-C3 disk space, or are subject to recurrent subluxation.47,60

Approximately 80% of all fractures of the lower cervical spine are produced by indirect forces. The vertebra most commonly involved is C5, and dislocations are most frequent at the C5-C6 level. The following injury mechanisms are observed: flexion and distraction (approximately 40% of cases), flexion and compression (22%), vertical compression (8%), extension and compression (24%), extension and distraction (6%), and lateral flexion (3%). Flexion and distraction injuries usually result from a blow to the occiput from below. The initial disruption is within the posterior ligamentous complex, leading to facet dislocation and an abnormally large divergence of the spinous processes. Unilateral facet dislocation and facet interlocking result when a rotary component is involved. Bilateral facet dislocation with anterior translation of the superior vertebra results from severe hyperflexion; the translation is usually at least 50% in such cases. Cord and root involvement vary with the degree of subluxation and translation: 50% of patients with unilateral facet dislocation present with moderate cord and root injury, and 90% of patients with bilateral facet dislocation and a full translation of the vertebral body have a neurologic deficit (most often a complete cord lesion). Teardrop fractures (characterized by a bone chip just beyond the anterior inferior edge of the vertebral body) result from severe hyperflexion injury, and the fractured vertebra is usually displaced posteriorly on the vertebra below; these patients are often quadriplegic.

Flexion and compression injuries, usually observed at the C4-C5 and C5-C6 levels, typically result from a blow to the back of the head. The effect on the anterior vertebral body varies from a moderate rounding or loss of anterior height to a wedge shape with an oblique fracture from the anterior surface to the inferior subchondral plate. Approximately 50% of patients with the latter type of injury have a neurologic deficit. More severe injuries are accompanied by translation of the inferior posterior margin of the vertebral body into the neural canal. About 75% of patients have neurologic involvement. Translations of more than 3 mm result in a complete spinal cord lesion in most cases.

Extension and compression injuries are usually caused by a blow to the forehead and result in fractures of the posterior complex. About 40% of patients with unilateral vertebral arch fractures (articular process, pedicle, or lamina) have a neurologic deficit (most often a radiculopathy). Bilaminar fractures are accompanied by a complete cord lesion in 40% of cases. Bilateral vertebral arch fractures with complete anterior translation of the vertebral body present with radiculopathy (30% of cases), central cord syndrome (30%), or an incomplete cord lesion (30%). In one series, no complete cord lesions were observed with this type of injury.61

Treatment of injuries to the lower cervical spine has not been standardized. As a general rule, severe ligamentous involvement and severe vertical compression call for surgical intervention. Severely comminuted vertebral body fractures may also necessitate surgery because of the high risk of progressive kyphosis. Isolated spinous process fractures and unilateral lamina and pedicle fractures are usually managed conservatively with placement in a rigid cervical collar. If there is a fracture through the transverse foramen above C6, the vertebral artery should be evaluated for dissection by means of magnetic resonance angiography (MRA), CT angiography, or catheter angiography.45 Surgical intervention should be carefully considered, especially in young trauma victims. In a series from 1988, 87% of patients with destructive flexion injury and 88% of those with compressive flexion injury healed with halo-vest immobilization.62

Thoracolumbar spine Approximately 64% of fractures of the spine occur at the T12-L1 junction, and 70% of these fractures are unaccompanied by immediate neurologic injury. Evaluation according to Denis’s three-column principle [see Figure 7] is useful for determining whether a fracture is stable, though the precise definition of stability remains controversial.63 Fractures of the thoracic spine are more stable because of support from the surrounding rib cage and the strong costovertebral ligaments. When two of the three columns are affected, the fracture is considered unstable, and surgical intervention is generally required.

The four major types of thoracolumbar spine injuries are compression fractures, burst fractures, seat-belt fractures (Chance fractures), and fracture-dislocations. These four types of fracture involve the anterior, middle, and posterior columns of the spine in different ways [see Table 4]. Transverse process fractures are rarely unstable and are typically managed conservatively with analgesics or muscle relaxants.

Minimal to moderate compression fractures (< 50% loss of height or < 30° of angulation) with an intact posterior column can be treated with analgesics and bed rest. Ambulation should be started early, and depending on the degree of kyphosis, external immobilization (with a thoracolumbar orthosis or a Boston brace) may or may not be indicated. Severe compression injuries should be treated with external immobilization in extension. If the loss of anterior height of the vertebral body exceeds 50%, there is an increased risk of progressive kyphosis; evaluation with follow-up radiographs is indicated. Occasionally, surgical intervention is required. An anterior injury is considered unstable if it involves more than three adjacent elements or if height loss in a single element exceeds 50% with more than 30° of angulation.64

Burst fractures are considered unstable even if there is no initial neurologic deficit. Early ambulation should be avoided because
the axial loading may result in progressive collapse or angulation, with concomitant neurologic damage. Indications for the surgical treatment of burst fractures are as follows:

1. Loss of more than 50% of body height.
2. Retropulsed bony fragments narrowing the canal by more than 50%.
3. Kyphotic angulation of 25° or more.

A Chance fracture is a horizontal fracture through all three columns. It occurs most commonly in the lower lumbar spine and is a highly unstable injury. Chance fractures are now less frequent than they once were because of the widespread use of shoulder belts in addition to lap belts, which prevents upper torso flexion during deceleration. Chance fractures are treated with surgical stabilization, and decompression and correction of spinal alignment may be required as well.

Fracture-dislocations, also known as fracture subluxations, are three-column injuries that usually involve disruption of the ligamentous structures or the disk space. Dural lacerations and neurologic injury are common with such injuries. Fracture-dislocations are considered unstable and are treated with surgical decompression and stabilization.

### Discussion

#### Pathophysiology

##### HEAD INJURY

**Cerebral Metabolism**

At 1,200 to 1,400 g, the brain accounts for only 2% to 3% of total body weight and does not do any mechanical work; yet it receives 15% to 20% of all cardiac output to meet its high metabolic demands. Of the total energy generated, 50% is used for interneuronal communication and the generation, release, and reuptake of neurotransmitters (synaptic activity); 25% is used for maintenance and restoration of ion gradients across the cell membrane; and the remaining 25% is used for molecular transport, biosynthesis, and other, as yet unidentified, processes.

Cell metabolism involves the consumption of adenosine triphosphate (ATP) during work and the ensuing consumption of metabolic substrates to resynthesize ATP from adenosine diphosphate (ADP). ATP is generated both in the cytosol (via glycolysis) and in the mitochondria (via oxidative phosphorylation). Glucose is the sole energy substrate, unless there is ketosis, and 95% of the energy requirement of the normal brain comes from aerobic conversion of glucose to water and CO₂. ATP generation is highly efficient. Glycolysis and subsequent oxidative phosphorylation result in the generation of 38 molecules of ATP for each molecule of glucose:

\[ 1 \text{ glucose} + 6 \text{ O}_2 + 38 \text{ ADP} + 38 \text{ P}_i \rightarrow 6 \text{ CO}_2 + 44 \text{ H}_2\text{O} + 38 \text{ ATP} \]

In the absence of oxygen, anaerobic glycolysis can proceed, but energy production is much less efficient. Two molecules of ATP and two molecules of lactate are generated for each molecule of glucose:

\[ 1 \text{ glucose} + 2 \text{ ADP} + 2 \text{ P}_i \rightarrow 2 \text{ lactate} + 2 \text{ ATP} \]

**Regulation of Blood Flow**

Because the reserves of glucose and glycogen within the astrocytes of the brain are limited and there is no significant storage capacity for oxygen, the brain depends on blood to supply the oxygen and glucose it requires. More specifically, substrate availability is determined by its concentration in blood, flow volume, and the rate of passage across the blood-brain barrier.

Under normal circumstances and with certain physiologic alterations, an adequate supply of substrates can be maintained by regulation of CBF. CBF increases with vasodilatation and decreases with vasoconstriction. Caliber changes take place mainly in cerebral resistance vessels (i.e., arterioles with a diameter of 300 µm down to 15 µm). Control of CBF by influencing vessel caliber is commonly referred to as autoregulation of blood flow.

**Metabolic autoregulation** CBF is functionally coupled to cerebral metabolism, changing proportionally with increasing or decreasing regional or global metabolic demand. Thus, the brain precisely matches local CBF to local metabolic needs. Because 95% of the energy in the normal brain is generated by oxidative metabolism of glucose, CMRO₂ is considered to be a sensitive measure of cerebral metabolism. The relation between CBF and metabolism is expressed in the Fick equation:

\[ \text{CMRO}_2 = \text{CBF} \times \Delta \text{VDO}_2 \]
CMRO₂, expressed in milliliters per 100 g of brain tissue, is normally about 3.2 ml/100 g/min in awake adults. The average CBF value for mixed cortical flow is 53 ml/100 g/min in a healthy adult. A-VDO₂, a measure of cerebral oxygen extraction, can be calculated by subtracting the oxygen content of jugular venous blood (6.7 ml/dl) from that of arterial blood (13 ml/dl), resulting in a value of 6.3 ml/dl; this value can then be corrected for hemoglobin content according to the formula discussed earlier [see Head Injury, ICU Management, above]. Under conditions of increasing metabolic demand (increased CMRO₂), such as seizures or fever, CBF increases proportionally, thus keeping A-VDO₂ constant. With decreasing metabolism (anesthesia, deep coma), CBF decreases.

**Pressure autoregulation** Another important physiologic property of the cerebral circulation is maintenance of a constant supply of substrates at the level set by metabolism. According to Poiseuille’s equation,

\[
\text{CBF} = k \frac{\text{CPP} \times d^4}{(8 \times 1 \times v)}
\]

in which \(k\) is a constant of proportionality, \(d\) is vessel diameter, \(l\) is vessel length, and \(v\) is blood viscosity, changes in CPP (e.g., arterial hypotension or increases in ICP) are followed by changes in CBF, unless diameter regulation (pressure autoregulation) takes place. In humans, the limits of pressure autoregulation range from 40 to 150 mm Hg of CPP.

**Viscosity autoregulation** In accordance with Poiseuille’s equation, CBF can vary with changes in the viscosity of blood. Blood viscosity changes with variations in hematocrit, \(\gamma\)-globulin, and fibrinogen components of plasma protein. Increased viscosity would increase cerebrovascular resistance \((8 \times 1 \times v / d^4)\). By means of diameter adjustment (viscosity autoregulation), cerebrovascular resistance is decreased and CBF can be kept constant.

**CO₂ reactivity** Vascular caliber and cerebral blood flow are also responsive to changes in \(P_{\text{CO}_2}\). Cerebral blood flow changes 2% to 3% for each mm Hg change in \(P_{\text{CO}_2}\) within the range of 20 to 60 mm Hg. Hypercarbia (hypoventilation) results in vasodilatation and higher CBF, and hypocarbia (hyperventilation) results in vasoconstriction and lower CBF. Autoregulation is a compensatory or adaptive response adjusting CBF to metabolism; with CO₂ variation, vessel caliber changes and CBF follow passively. The vessels respond not to changes in \(P_{\text{CO}_2}\) but to the pH in the perivascular space. CO₂ can cross the blood-brain barrier freely, thus changing the pH, but over 20 to 24 hours, with a constant new level of \(P_{\text{CO}_2}\), the pH in blood and in the perivascular space returns to baseline, and the diameter of the cerebral blood vessels also returns to baseline. With CO₂ reactivity, changes in CBF are compensated for by changes in A-VDO₂, so that a constant supply of substrates is maintained at the level set by metabolism (CMRO₂). A constant A-VDO₂ is a common feature of metabolic, pressure, and viscosity autoregulation; because CBF is tuned to metabolism, A-VDO₂ can be kept constant.

Cerebral Circulation and Metabolism after Severe Head Injury

**Arterial hypoxia and hypotension** It is known from eyewitness reports of head injury and experimental studies immediately after the impact that arterial hypotension and interruption of normal respiration, sometimes with a period of prolonged apnea, are common findings. In the days after a head injury, there are many occasions and opportunities for hypoxic and hypotensive insults. Studies have identified hypotension (systolic BP < 90 mm Hg) and hypoxia (\(P_{\text{O}_2}<60\) mm Hg) as major determinants of poor outcome.

The effect of hypotension on the brain depends on the status of autoregulation. If autoregulation is defective, decreased BP leads directly and linearly to a reduction in CBF. If autoregulation is intact, arterial hypotension can lead to a considerable increase in ICP, which interferes with CBF by decreasing perfusion pressure.

**Elevated ICP** According to the Monro-Kellie doctrine, ICP is governed by three factors within the confines of the skull: brain parenchyma plus cytotoxic edema; CSF plus vasogenic edema; and CBV. When the volume in one compartment increases, ICP increases unless there is a compensatory decrease in volume in the other compartments. The relationship between intracranial volume and ICP is expressed in the pressure-volume index (PVI). PVI is defined by the volume that must be added to or withdrawn from the craniospinal axis to raise or decrease ICP 10-fold:

\[
PVI = \frac{\Delta V}{\log ICP_i / ICP_o}
\]

where \(\Delta V\) is the change in volume, ICP₀ is ICP before the volume change, and ICP is ICP after the volume change. PVI is thus a measure for the compliance (\(\Delta V/\Delta P\)) or tightness of the brain. Under normal circumstances, PVI is 26 ± 4 ml; 26 ml of volume will raise ICP from 1 to 10 mm Hg, but the same volume will also raise ICP from 10 to 100 mm Hg. Conversely, a change in volume of only 6.4 ml is necessary to increase ICP from 10 mm Hg (normal) to the treatment threshold of 20 mm Hg. Thus, small changes in volume have a relatively large effect on ICP. PVI values as low as 5 ml have been reported in patients with head injuries.
Apart from mass lesions, ICP typically increases after severe head injury because of cerebral edema. Initial compensation is by displacement of CSF from the cranium, which is visualized in a CT scan of the head as small ventricles and basal cisterns. Subsequent compensation would be by a decrease in CBV, which can be accomplished by means of vasoconstriction.

**Relation between vessel diameter, CBV, and ICP** The total diameter of the cerebrovascular bed determines CBV. Cerebral veins contain most of the total blood volume, but their diameter and thus their volume are relatively constant. Approximately 20 ml of blood (i.e., one third of total CBV) is located in the cerebral resistance vessels (which range in diameter from 300 μm down to 15 μm).^69^ Because most autoregulatory and CO₂-dependent variations in diameter take place in these vessels, CBV is determined mainly by their diameter. Typically, the diameter ranges from 80% to 160% of baseline, resulting in volume changes between 64% and 256% of baseline. With a baseline value of 20 ml in the resistance vessels, CBV will range from 13 ml (maximal vasodilation) to 51 ml (maximal vasodilatation). Given a PVI of 26, change from maximal vasoconstriction to maximal vasodilatation will be accompanied by an almost 29-fold change in ICP.

**CBV, ICP, and CBF** CBF and CBV are governed by vascular diameter. Thus, depending on other parameters influencing CBF (such as mean arterial BP, ICP, and blood viscosity), changes in vascular caliber also affect CBF.

Hypocarbia reduces ICP by means of vasoconstriction, consequently improving CPP. However, net CBF is decreased because in Poiseuille’s equation, vessel diameter is carried to the fourth power. A randomized clinical trial has shown that preventive hyperventilation retards clinical improvement after severe head injury, perhaps through reduction of CBF to ischemic levels.^74^ However, its rapid effect on ICP is a great advantage in cases of acute neurologic deterioration (e.g., in the presence of an expanding mass lesion before evacuation can take place) and should be reserved for these situations.

There are two methods of reducing ICP by means of vasoconstriction without affecting CBF: The first is to reduce blood viscosity. As can be deduced from Poiseuille’s equation, decreasing the blood viscosity will, by itself, lead to vasoconstriction, provided that viscosity autoregulation is intact. With impaired autoregulation, decreased viscosity will result in an increase in CBF but no decrease in ICP. However, this effect can be used to maintain CBF under vasoconstriction with hypocarbia. The effect of mannitol on ICP is thought to be mediated in part by lowering blood viscosity.^75,76^

The second method of reducing ICP without affecting CBF is to increase CPP, which can be done by raising blood pressure. Again, with intact autoregulation, an increase in CPP will lead to vasoconstriction, with net CBF remaining constant. With impaired autoregulation, CBF will follow CPP passively, and maintenance of normal BP may be indicated in these cases. More important, however, is the avoidance of hypotension under these circumstances; the effect of CPP therapy may be attributable in part simply to prevention of hypotension.26,77

**Cerebral ischemia** Cerebral ischemia, defined as CBF that is inadequate to meet the metabolic demands of the brain, is an important mechanism of secondary injury in patients with severe head injury, and the adequacy of CBF has been associated with neurologic outcome. In autopsy findings from patients dying after severe head injury, histologic damage indicative of cerebral ischemia was seen in 80% of cases.78 One group of investigators found that ischemia (CBF < 18 ml/dl with abnormally high A-VDO₂ values) occurred in 20% to 33% of patients with severe head injuries within 4 to 12 hours of injury and that the ischemia was associated with a poor prognosis.79 Of the intracranial lesions, acute subdural hematoma and diffuse cerebral swelling were most often associated with ischemia.

The relation between cerebral metabolism and CBF is expressed in the Fick equation [see Metabolic Autoregulation, above]. The normal brain tends to keep A-VDO₂ constant and to react to changes in metabolism by adjusting blood flow. When CBF decreases in response to metabolism (as with hyperventilation or decreasing CPP with impaired autoregulation), oxygen supply is maintained by increasing oxygen extraction (i.e., A-VDO₂ increases). A rising A-VDO₂ is thus a sensitive marker of inadequate cerebral perfusion. However, the extent to which oxygen extraction can be increased is limited, and this limit is reached when A-VDO₂ is doubled (13.2 ml/dl). Consequently, any further reduction in CBF results in neuronal dysfunction (i.e., CMRO₂ decreases). Because 50% of the energy is used for synaptic activity, a reversible and functional loss is usually observed first. Further decline, however, will result in ion pump failure, loss of membrane integrity, consequent cell swelling (cytotoxic edema), and cell death (irreversible infarction). The occurrence of irreversible infarction depends on both the level and the duration of ischemia. When CBF falls to approximately 18 ml/100 g/min for more than 4 hours, it reaches the threshold for irreversible infarction.80

Maintenance or improvement of CPP is thus essential to the treatment of severe head injury; and A-VDO₂ is a sensitive marker of the adequacy of therapy. When therapeutic measures fail to sustain CPP, CMRO₂ can be decreased to reinstate the match between CBF and metabolism. CNS suppression can be achieved by administering hypnotic agents (e.g., barbiturates or propofol) or inducing hypothermia. Decreasing cell metabolism will result in reduced production of CO₂, lactic acid, or both and (with blood vessels almost always remaining responsive to pialessicular pH changes) in vasoconstriction accompanied by reductions in both CPP and ICP. The relations between CMRO₂, CBF, CBV, CPP, and A-VDO₂ are complicated. An overview is available elsewhere [see Table 5].81

**Table 5** Changes in CBF, CBV, ICP, and A-VDO₂ Associated with Primary Reduction of Selected Variables

<table>
<thead>
<tr>
<th>Variable Reduced</th>
<th>CBF</th>
<th>CBV (ICP)</th>
<th>A-VDO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>CMRO₂</td>
<td>↓</td>
<td>↓</td>
<td>—</td>
</tr>
<tr>
<td>CPP (autoregulation intact)</td>
<td>—</td>
<td>↑</td>
<td>—</td>
</tr>
<tr>
<td>CPP (autoregulation defective)</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Blood viscosity (autoregulation intact)</td>
<td>—</td>
<td>↓</td>
<td>—</td>
</tr>
<tr>
<td>Blood viscosity (autoregulation defective)</td>
<td>↑</td>
<td>—</td>
<td>↓</td>
</tr>
<tr>
<td>P&lt;sub&gt;C&lt;/sub&gt;O₂</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Conductance vessel diameter (vasospasm above ischemia threshold)</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

A-VDO₂—arteriovenous oxygen content difference CBF—cerebral blood flow CBV—cerebral blood volume CMRO₂—cerebral metabolic rate of oxygen CPP—cerebral perfusion pressure ICP—intracranial pressure P<sub>C</sub>O₂—arterial carbon dioxide tension
poikia/ischemia. Increased lactate production, hyperglycolysis, and low tissue glucose levels have been observed after severe head injury, suggesting an increased turnover of glucose by the anaerobic glycolytic pathway. Increased lactate levels have also been found in the presence of preserved CBF, suggesting impairment not only of oxygen delivery but also of oxidative metabolism (i.e., of mitochondrial function). Data from animal and human models indicate that mitochondrial function is impaired after severe head injury, which may explain the poor outcomes despite adequate CBF levels; ATP generation by anaerobic glycolysis is usually insufficient to maintain the metabolic activity of the brain. In part, however, such poor outcomes may be attributable to the effects of lactate production (acidosis), because high lactate and hydrogen ion levels interfere with the functional recovery of tissue.

**SPINAL CORD INJURY**

Spinal cord injury is often viewed as an all-or-nothing event that is irreversible from the moment of injury. By this view, spinal cord injury is classified as either incomplete or complete. This dichotomy is not absolute, however, because some functional recovery occurs even after severe spinal cord injury. NASCIS II revealed that patients with so-called complete loss of neurologic function recovered, on average, 8% of the function they had lost, and patients with an incomplete injury recovered 59%. An injury classified as complete does not necessarily involve loss of all connections. Several studies have demonstrated that many patients with a clinically complete lesion show evidence of residual connections. A certain number of intact connections is probably necessary for functional recovery. The determinants of functional outcome are complex, however, and probably include not only the extent of axonal loss but also the level of dysfunction of the surviving axons and the plasticity of the spinal cord.

Animal studies have shown that a small number of axons may be sufficient to support functional recovery. Animals recover evoked potentials and the ability to walk with as few as 10% of their spinal axons. Nerve sprouting, one of the mechanisms of plasticity, allows a few nerves to carry out the function of many. Finally, animal studies have also shown that many of the axons surviving traumatic injury are dysfunctional and that many of these axons have lost part or all of their myelin sheath, which is the structural component that improves the reliability and speed of conduction. 4-Aminopyridine, an axon-excitatory drug used for the treatment of multiple sclerosis, has significantly improved conduction in animals and humans with spinal cord injury. Unfortunately, the drug must be given continuously to support axon function, and this is not feasible in humans because of its side effects (seizures, tachycardia, and hyperthermia).

Injury initiates complex responses in the body and the spinal cord. Ischemia is a prominent feature of events occurring after spinal cord injury. Within 2 hours of a spinal cord injury, there is a significant reduction in spinal cord blood flow. It is unclear whether this reduction is mechanically or biochemically induced. Like the brain, the spinal cord possesses autoregulatory capacity (pressure autoregulation). When this autoregulation is impaired, blood flow becomes dependent on systemic blood pressure. In a patient with multiple injuries or vasogenic spinal shock (a lesion above T5) complicating the spinal cord injury, severe systemic hypotension may exacerbate the effects of spinal cord injury.

Edema, another prominent feature of spinal cord injury, tends to develop first at the injured site, subsequently spreading to adjacent and sometimes distant segments. The relation between spreading edema and potential worsening of neurologic function is poorly understood. The inflammatory response to injury is mounted in part to scavenge cellular debris and repair tissue. This response is accompanied, however, by the release of toxic substances, which cause further tissue damage, or secondary injury. Processes resulting in secondary injury involve generation of free radicals, excessive calcium influx and excitotoxicity, the release of eicosanoids and cytokines, and programmed cell death.

Some evidence from experimental studies of spinal cord injury suggests that macrophages may play a key role in CNS repair. Administration of stimulated macrophages to the CNS, where the number of macrophages is limited and their activity restricted in comparison with other tissues, has led to partial motor recovery in a completely transected spinal cord in adult rats. Clinical trials have been initiated to evaluate this approach further.

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Acknowledgments

Figure 2 Seward Hung.
Figure 4 Reprinted courtesy of the American Spinal Injury Association, Chicago, Illinois.