

Chapter 16

NEUROLOGICAL INJURIES

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INTRODUCTION

Traumatic brain injury is classified as *open* or *closed*. Closed head trauma is further subdivided into *severe* (characterized by prolonged coma; ie, inability to obey simple verbal commands), *moderate* (either requiring craniotomy or producing coma of short duration), and *minor* (neither requiring surgery nor resulting in coma). Because of the enormous economic and social toll exacted by vehicular trauma, closed head injury, which is a common cause of death after civilian blunt trauma, has been extensively studied in patients and in experimental models. Open head injury, which is more relevant to military trauma, has been studied much less well. Therefore, many of the principles of nonoperative management of penetrating brain injury are based on the assumption that knowledge gained from clinical or experimental closed brain injury can be extrapolated to injuries caused by penetrating missiles. One central assumption is that secondary brain injury—an often-preventable consequence of hypotension, hypercarbia, hypoxemia, or intracranial hypertension—contributes heavily to death or disability caused by the original injury.^{1,2}

Penetrating injuries to the spinal cord do not play a large role in military medicine: most casualties whose spinal cords are transected are killed when the missile (bullet or fragment) continues on its

path and injures nearby organs (eg, the heart and great vessels, liver, spleen). In addition, data gathered during the Vietnam War by the Wound Data and Munitions Effectiveness Team (WDMET) indicate that 98% of soldiers with gunshot wounds of the cervical spine that included the cord were fatally injured.³ Closed injuries to a soldier's spinal cord are not different from those that occur to civilians, and occur for the same reasons (eg, blunt trauma due to vehicular accidents and falls). There are far more casualties with brain than spinal cord injuries; furthermore, the chances that therapeutic intervention will effect improved results are greater with brain than with spinal cord injuries. For these reasons, most of this chapter pertains to traumatic brain injury; spinal cord injury is treated much less extensively.

This chapter discusses the perioperative care of the neurologically injured patient as it is practiced today in medical centers. Because the equipment and facilities found in the Department of Defense's Deployable Medical Systems (DEPMEDS) hospitals replicate to some extent the sophistication of civilian trauma centers, we have included descriptions of state-of-the-art systems and management techniques. However, we also provide practical suggestions for use when advanced support systems are not available.

THE NATURE OF HEAD INJURIES SUSTAINED IN COMBAT

Combat casualties with head trauma will present with either open or closed injuries. In open injuries, the scalp is lacerated and the bone is fractured, usually with significant depression, thereby exposing the dura; this in turn is usually lacerated, exposing the cortex, which is almost invariably damaged as well. Closed head injuries may or may not involve laceration of the scalp, but if a laceration is present, the calvarium either will be uninjured or will have no more than a simple, nondepressed, linear fracture. In most cases of closed head injury, there is no scalp laceration or skull fracture at all.

Combat casualties with head trauma can also be classified as having either *blunt* or *penetrating* injuries, depending on the mechanism of injury. Blunt trauma usually results in closed injuries, while penetrating wounds are almost always open. In the combat zone, the overwhelming majority of battle casualties with head trauma have penetrating missile wounds made either by fragments from explo-

sive munitions or bullets fired by small arms. Most of the latter wounds are open in the sense that the dura has been lacerated and the cranial contents are exposed. When closed injuries due to blunt trauma occur in the combat zone, they are usually due to accidents not related to hostile action. Such casualties are classified as having *nonbattle* injuries.

Penetrating missile wounds of the head are extremely lethal. WDMET data from the Vietnam War indicate that the probability of being fatally wounded by a missile—either fragment or bullet—that hit the skull was close to 4 out of 5 (Chapter 1, Combat Trauma Overview, discusses the WDMET data more fully). Bullet wounds to the head made by assault rifles were fatal in about 95% of cases.³ Combat casualties with penetrating head trauma who survive to reach a deployable hospital fall into two categories: (1) most commonly, a small fragment wound, with injuries to one cerebral lobe; or (2) less commonly, a tangential gunshot wound, in

which the bullet causes a depressed skull fracture, which, in turn, actually lacerates the dura and the brain. Depending on how deeply the bullet has penetrated into the intracranial compartment, it may or may not, in and of itself, directly injure the brain parenchyma. Neurosurgical practice in the combat zone consists of managing both these categories of casualties and those with multiple lobe injuries, such as those caused by bullets. These casualties are in addition to those who demonstrate the spectrum of closed injury, which can range from transient loss of consciousness to deep coma.



Fig. 16-1. The protection provided by Kevlar helmets is impressive: this helmet, seen close up, was struck by an 0.45 ACP (automatic Colt pistol) at a range of 3 m. The defeated bullet is shown on the left; an unfired bullet is shown on the right. By way of contrast, the 9 x 19-mm Parabellum round will usually perforate a Kevlar helmet. Photograph: Wound Ballistics Laboratory, Letterman Army Institute of Research, Presidio of San Francisco, Calif.

Given the dismal outcome of penetrating missile wounds to the head, preventive measures such as protective helmets have long been recognized to be of great importance. The presently fielded military helmet will stop most fragments produced by improved fragmentation munitions as well as certain types of bullets fired from pistols (Figure 16-1). Fielded helmets will rarely stop a bullet fired from a rifle or machine gun unless the bullet's performance has been severely degraded by passing through a solid object (such as another casualty), has ricocheted off an object and is traveling sideways, or is at the end of its trajectory (1,000 m or so) (Figure 16-2).

During the Persian Gulf War, the Kevlar helmet was found to be extremely beneficial and was associated with a significant diminution of open head injuries, compared to what would have been expected and considering the number of injuries seen to other parts of the body, particularly when compared to the number of injuries to the face and eyes.⁴ For this reason, we might well expect that in future conflicts (in which explosive munitions are the predominant source of penetrating missiles, as they were during the



Fig. 16-2. Both sides of this Kevlar helmet have been perforated by a 5.45-mm bullet (fired by an AK74 at range of 3 m), which entered from the viewer's left. Notice the mushroomlike mass of disrupted Kevlar at the site of the bullet's exit. Kevlar fibers have been found lining tissue wound tracts in experimental ballistic studies. Photograph: Wound Ballistics Laboratory, Letterman Army Institute of Research, Presidio of San Francisco, Calif.

Persian Gulf War), a greater percentage of hospitalized casualties will have closed rather than open head injuries.

Casualties who have sustained closed head injuries and survive to reach the hospital level often suffer more significant brain injury than do those who have sustained open injuries. This is due, in part, to the fact that open injuries generally allow some decompression of the immediately surrounding cerebral substance through the break in the skull. In closed injuries, in contrast, the additional volume of blood clot and cerebral swelling must be accommodated within the space of the bony intracranial compartment, which does not change in volume. Therefore, the brain tissue itself is subjected to the total obligatory compression (aside from the small amount that is accommodated by the vessels and the ventricles).

In addition, the physical factors that determine whether an injury will actually break the calvarium are generally associated with a more focal and less diffuse distribution of kinetic energy. The injury is, therefore, usually greater at a small, focal area. The casualty with a significant closed head injury will usually present in coma, with or without lateralizing signs such as extraocular motility and pupillary pareses or asymmetrical posturing. Closed or open facial injuries may well be present also, and airway obstruction must always be the first consideration. Injuries to other parts of the body must be suspected and searched for. Although most casualties with a significant, life-threatening head injury will be comatose when they are evaluated initially, a significant number will be awake but will subsequently deteriorate; a patient with mental status changes, focal neurological findings (particularly those suggestive of a seizure), or even a good history for significant head injury must be observed very carefully.

Casualties with open head injuries may appear more frightening, particularly if brain is exposed, but as noted above, they may have less diffuse brain injury than casualties with closed head injuries. Casualties with open head injuries (and even exposed and injured brain) may be wide awake and alert, often with only a minor, focal, neurological deficit—or no deficit at all. On the other hand, a wound to the brain made by a small fragment or a bullet that has not glanced may well have only a small wound of entrance with extremely little surface tissue damage. These wounds are often much more serious than those

involving much more significant surface tissue damage, mostly because in the latter case, the kinetic energy imparted to the head at the wound of entrance is often superficial and tangential. A computed tomography (CT) scan of the former type of wound will often disclose significant intracranial injury that was belied by the relatively benign appearances of the scalp and bone injuries. Such a scan will usually demonstrate significantly greater tissue injury at the wound of exit, especially when the penetrating missile is a bullet. The reason for the greater damage at a bullet's wound of exit is that yaw and tumbling are much more prominent here than at the wound of entrance. For this reason, if the bullet or an irregular fragment has actually exited through the side opposite the wound of entrance, or is lying near the inner surface of the calvarium on the opposite side, that side should generally be addressed first at the time of surgery—as soon as any life-threatening superficial bleeding has been stopped at the wound of entrance. The technique will be discussed in greater detail later in this chapter.

A casualty with a glancing or a relatively shallow injury will often present with much more extensive superficial tissue damage. Here the cerebral damage will be relatively superficial and usually not involve the deeper structures. An example of this would be two different types of self-inflicted gunshot wounds. In the first instance, the individual would point the barrel of the gun directly at his temporal region, then pull the trigger. On leaving the barrel of the gun, the bullet would enter immediately, pass through both hemispheres, and exit the calvarium at the other side. In all likelihood, the greater cerebral damage would be just inside the wound of exit. The superficial damage at the wound of entrance would be relatively slight. In the second instance, consider that the individual flinched at the last instant, turning the barrel so that it was nearly parallel to the plane that was tangential to the skull at the site of the wound. Here, extensive scalp damage would occur and the depressed skull fracture would traverse the the calvarium up to the point where the tangent plane was no longer in contact with the skull. The cerebral damage would be limited to the surface and perhaps a centimeter or so in depth. Keeping these two examples in mind will allow the medical officer to interpret the clinical significance of the appearance of different types of penetrating injury.

GOALS OF NEUROSURGICAL INTERVENTION

Aside from debridement, treatment modalities for casualties with head injuries are, for the most part, nonsurgical. Injury to central nervous system substance cannot be repaired by any primary physical intervention (analogous to suturing for the repair or reanastomosis of a lacerated abdominal organ). If central neural fibers are cut or definitively crushed, there is no treatment, surgical or otherwise, that will restore function. If, on the other hand, the substance of central neural fibers is intact but their function is reversibly impaired (ie, a situation analogous to that of neuropraxia in the peripheral nervous system), certain interventions seem to be capable of altering the physiological environment to make it more conducive to the reversal of the dysfunction. Consequently, nonsurgical treatment of casualties with head injuries is emphasized in this chapter, although several surgical considerations require discussion.

The goal of surgery in closed head injury is generally limited to decompressing the brain parenchyma by removing a mass lesion, which will almost invariably be a hematoma. The only exceptions to this procedure would be the relatively rare circumstances in which (a) a lobectomy would be performed to remove edematous brain or (b) a cerebrospinal fluid leak would be repaired. The former would usually be a delayed procedure, necessitated at the time of peak swelling (48–72 h after the injury); the latter would almost invariably be delayed well beyond that time.

In open or penetrating head injury, the goals are generally very clearly defined; unlike the usual situation with closed head injury, at least some degree of surgical treatment is almost invariably indicated. Perhaps the first truly comprehensive description of modern neurosurgical techniques in open head injury can be found in Harvey Cushing's 106-page article published in 1918 in the *British Journal of Surgery*,⁵ in which he chronicles his extensive experience obtained on the World War I battlefields of France. This, along with his companion article published the same year in the *British Medical Journal*,⁶ set the procedural standards, which remained essentially unchanged (aside from the development of certain new hemostatic techniques) until the work of Arnold M. Meirowsky,⁷ published half a century later in an official history of the U.S. Army Medical Department, and based on his Korean War experience. Meirowsky's article remained

the clear standard until a reasonable update was suggested by Benny Brandvold and colleagues⁸ in 1990, writing of their experiences with casualties evacuated to Rambam Maimonides Medical Center, Haifa, Israel.

The first priority in any trauma surgery is invariably the control of hemorrhage; the second is the removal of contaminated material and nonviable tissue. In this regard, open head injury is no exception and initial hemorrhage control is straightforward. Purely on the basis of cerebral compression, it is virtually impossible to exsanguinate into the substance of the brain (unlike into the abdomen or most other regions of the body). The only exception is in the newborn, with its large intracranial volume relative to the rest of its body, and its widely expansive cranial sutures. It is also impossible for hemorrhage deep in the cerebral substance to continually drain a penetrating wound, as the brain will swell and shut off such egress very rapidly. All exsanguinating hemorrhage in open head injury must then emanate from skin, bone, the dural sinuses, or extremely superficial cortical vessels. The scalp is well vascularized, and exsanguination from a scalp laceration alone is possible. Bleeding from the diploic spaces of fractured calvarium is generally limited to a moderately brisk ooze, at most, but over time this can be significant. Unless instantly tamponaded by a depressed bone fragment, bleeding from a lacerated dural sinus would generally result in exsanguination after a few minutes. The surgeon will therefore see this kind of hemorrhage only after an appropriately situated, depressed fracture is elevated. Brisk bleeding from a cortical (pial) vessel is often seen on elevation of depressed bone.

The first steps of surgical treatment will therefore be the control of hemorrhage from the sources described above. Tricks that help the surgeon depend on a familiarity with the anatomy and character of the vessels in each of these areas. Cessation of scalp bleeding, for example, requires coagulation of individual vessels that are found immediately deep to the galea. Grabbing the galea with forceps will disclose these structures quite easily. Bone hemostasis can readily be obtained by rubbing a small amount of bone wax over the cut edge. Dural sinus bleeding will be extremely profuse as soon as the tamponading bone has been removed, but it can be slowed down considerably by simply elevating the head of the table (ie, putting the patient into a

reverse Trendelenburg's position). Too much elevation, on the other hand, can allow air to be entrained into the sinus, which is rigidly supported by the dura. This can easily lead to an air embolus. The trick, therefore, is to elevate the head of the bed just enough to allow a slow egress of blood, which will permit the surgeon to repair the sinus with direct suture or a patch of cadaver dura or fascia. The bony diploë also contain blood-filled spaces that will entrain air if the head of the bed is high enough. Rapid and accurate use of wax is extremely important here. Bleeding from the superficial cortex is generally from vessels immediately deep to the pia mater, and most of these can be identified and cauterized.

Once life-threatening hemorrhage has been controlled, the surgeon's attention is turned to debridement of the projectile tract. Contaminated cerebral substance that is oozing out of the wound site is totally nonviable and will never regain function. It can therefore be removed without condemning the patient to further deficit. If left in place, it will undoubtedly lead to further necrosis, cerebritis, and abscess formation. All devitalized brain tissue must therefore be removed by suction, which is continued until the tract remains open and does not immediately swell shut.

Meirowsky believed that the entire wound tract absolutely required radical debridement, and that *all* foreign material, aside from the metal itself, required complete removal. For many years, all bone was searched for, even if it meant increasing the deficit, as surgeons believed that any retained bone would unquestionably lead to abscess formation. However, the Vietnam Head Injury Study⁹ has suggested that, although careful debridement is indicated, small amounts of bone left in place may not lead to quite as dangerous a situation as Meirowsky had previously suggested. More recently, experience in the the Israeli-Lebanese War has suggested that with mandatory monitoring a slightly more conservative approach may be acceptable, as long as the threshold for emergent intervention remains low.⁸ Presumably, newer antibiotics and better imaging techniques have been instrumental in this change in philosophy (Figure 16-3).

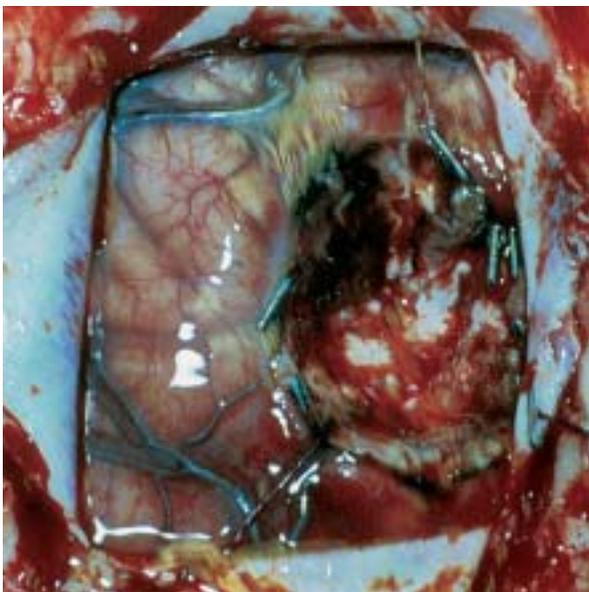
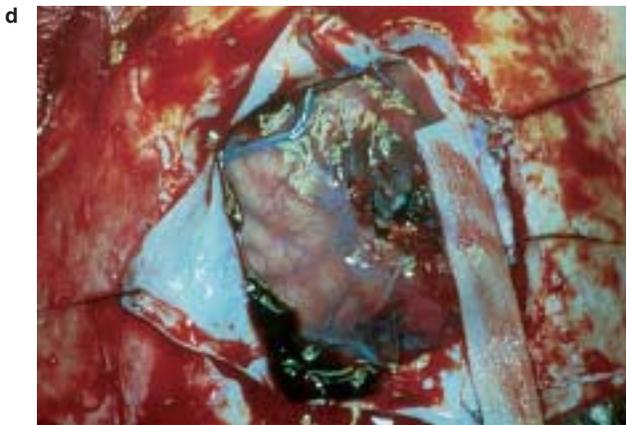
As Cushing pointed out in 1918, failure to close (or patch) the dura will lead to the development of fungus cerebri (ie, an ulcerated cerebral hernia with granulation protruding from the scalp wound). Inattention to dural tears at the base of the skull will lead to cerebrospinal fluid leaks through the nose or



Fig. 16-3. This combat casualty has a typical penetrating head wound (made by a preformed fragment from an improved fragmentation grenade). At the time of operation, the casualty was alert and able to move his extremities normally. (a) The casualty before induction of anesthesia. The wound of entrance is seen in the right frontal region. The radiographs demonstrate that the fragment (b) has crossed the midline and (c) has come to rest in the superficial portion of the casualty's left parietal cortex. (d) A craniectomy has been performed and (e) the missile tract at the wound of entrance has been debrided. (f) The craniectomy site has been closed with a fascial graft. (g) An anteroposterior radiograph of the skull shows the large defect left by the craniectomy. The fragment is still present. The tissue damage and resulting heightened neurological defect caused by its removal would probably not have been justified by the resultant decrease in the risk of complications such as sepsis arising from a retained foreign body. In contrast to bullets, fragments usually cause more tissue damage where they enter the brain substance than they cause in the more distant parts of their pathway. Photographs: Swan Vietnam Surgical Slide Collection.

ear, which may lead to a need for subsequent surgical repair. Closed head injuries may also require delayed surgical treatment for cerebrospinal fluid rhinorrhea or otorrhea.

Once these surgical considerations have been addressed, the nonsurgical interventions, which can improve the physiological environment so as to promote neural survival and recovery (as well as the survival and recovery of the casualty) may be entertained. Future surgical considerations will be limited to unfortunate circumstances such as delayed brain swelling (rarely requiring lobectomy), further cerebrospinal fluid leaks, or abscess formation.



PATHOLOGICAL ANATOMY AND PHYSIOLOGY OF TRAUMATIC BRAIN INJURY

Traumatic brain injuries are diffuse or focal. Comatose trauma patients who demonstrate no focal lesions by CT or magnetic resonance imaging (MRI) during initial diagnostic assessment are said to have diffuse brain injury. Diffuse injury includes diffuse axonal injury, hypoxic brain injury, diffuse brain swelling, and diffuse punctuate brain hemorrhages; focal brain injury includes contusions, avulsions, hematomas, hemorrhages, infarctions, and infections. Diffuse injuries produce coma through damage to the brainstem or cerebral cortex. Focal injuries produce coma by brain compression, brain shift, or herniation. Patients with diffuse brain injury are much less likely than those with focal injuries to have sustained a skull fracture or cerebral contusion, to develop signs of sustained intracranial hypertension, or to have a lucid interval after injury before becoming comatose.²

Hypoxic cerebral damage, a common postmortem finding after blunt head trauma,^{10,11} is associated with arterial hypoxemia or cerebral hypoperfusion, occurring as a consequence of shock, sustained intracranial hypertension, or cerebral vasospasm.

Both cerebral edema and cerebral swelling occur after brain trauma. Vasogenic cerebral edema results when damage to the blood–brain barrier leads to increased accumulation of interstitial water. Cytotoxic edema, characterized by cellular water accumulation, may follow cerebral hypoxia. In contrast, diffuse cerebral swelling represents vascular congestion, evident on CT scan as symmetrical effacement of the lateral ventricles and the basal cisterns, and normal or increased white matter density.

Ten percent of patients with closed head injuries and Glasgow coma scale (GCS) scores of 13 will require surgery for intracranial lesions. This decision, will, of course, be made on a combination of clinical and radiographic (ie, CT) criteria. The most common focal injuries are cerebral contusions, intraaxial (within the substance of the neuraxis) hematomas, and extraaxial (subdural and epidural) hematomas. Extraaxial hematomas are rarely detected in patients who have both a GCS score of 15 and no focal neurological deficits.¹² On CT, subdural hematomas tend to be crescent shaped, spreading out across the brain surface. In contrast, the typical epidural hematoma has a biconvex (ie, lens-shaped) appearance. Delayed intracerebral hematomas may follow a combined traumatic and anoxic insult.

Closed Brain Injury

Most forms of acute brain injury in humans result from sudden cranial impact or angular cranial acceleration. Impact injuries deform and sometimes fracture the skull, initiating shock waves that are transmitted throughout the brain. When brain tissue strikes bony intracranial prominences, disruption of vascular integrity may produce focal injuries. The clinical spectrum of anatomical findings in minor, moderate, and severe diffuse brain injuries has been reproduced experimentally.¹³ Neurological outcome after experimental head trauma correlates with the degree of parenchymal brain injury, especially the extent of primary axonal injury. Traumatic brain injury also alters the cerebral vasculature and disrupts the blood–brain barrier. This disruption may represent the most fundamental response of the brain vasculature to injury and may be a primary mechanism of concussion.¹⁴

The systemic physiological responses to experimental brain injury include an immediate period of apnea, the duration of which increases with increasing severity of injury.¹⁵ Hypertension and bradycardia immediately follow moderate-to-severe injury, even if mechanical ventilation prevents apnea. Intracranial hypertension consistently accompanies the arterial hypertension; however, intracranial pressure (ICP) returns to normal before the arterial blood pressure. Intracranial hypertension is usually not sustained after experimental head injury.

Penetrating Brain Injury

The pathophysiology of experimental penetrating brain injury has been less extensively investigated than closed head injury. The duration of injury-induced apnea increases in proportion to the muzzle energy of the projectile. Blood pressure and cerebral perfusion pressure (CPP) tend to increase steadily after wounding.¹⁶ In monkeys, missile wounds produce dramatic, immediate increases in ICP (to 40–60 mm Hg) that persist for at least 10 minutes after impact. As CPP falls to approximately 50% of preinjury levels, cerebral blood flow (CBF) declines to approximately 60% of baseline.¹⁷

Enhanced Vulnerability to Secondary Ischemic Brain Injury

Current clinical and experimental data leave little doubt that the traumatized brain is uniquely vul-

nerable to posttraumatic ischemic insults. Disability and death arising from insults such as hypotension and hypoxemia are important because they occur after patients have entered the medical care system. Consequently, prevention and effective treatment of secondary ischemic insults should limit morbidity and mortality.

Secondary ischemic brain injury appears to contribute to poor outcome after traumatic brain injury. In one study,¹¹ nearly 90% of patients who died after traumatic brain injury showed ischemic lesions in a variety of patterns. Diffuse ischemic injury was evident in 23.5%, arterial boundary-zone ischemic injury in 7.5%, ischemia in a specific arterial territory in 10.5%, and hippocampal ischemic injury in 45%.

Hypotension and hypoxemia—systemic insults that can induce cerebral hypoperfusion—worsen outcome, both in terms of higher mortality and worse neurological recovery, after head injury. In 1981, researchers² examined the effects of a variety of potential secondary insults on the incidence of unfavorable outcome (ie, severe disability, persistent vegetative state, or death) after severe traumatic brain injury and found that a systolic blood pressure of 90 mm Hg or lower on admission to the hospital was associated with an increase in the incidence of unfavorable outcomes, from 35% to 65%; and hypoxemia (≤ 60 mm Hg on admission) was associated with an increase in unfavorable outcomes, from 35% to 59%. A retrospective review published in 1993¹ of the experience of the Trauma Coma Data Bank even more strongly suggests that hypotension is a powerful predictor of poor outcome after head injury.

Experimental brain injury is followed by a decrease in brain pH and, under certain circumstances, both a decline in high-energy phosphate compounds and an increase in tissue lactate levels. Posttraumatic deterioration in cerebral aerobic metabolism is aggravated by concurrent hypoxia or hypotension. After experimental injury, severe hypoxemia ($\text{PaO}_2 < 40$ mm Hg) worsens neurological deficits and brain ischemia, increases tissue inorganic phosphate, and reduces phosphocreatine and adenosine triphosphate.¹⁸ Similarly, hypotension that alone would not alter the cerebral energy state (assessed by magnetic resonance spectroscopy) reduces brain intracellular pH, adenosine triphosphate, and phosphocreatine, and increases the concentration of inorganic phosphate after brain trauma.¹⁹ On the other hand, a study with animals published in 1993²⁰ demonstrates the existence of an impact level beyond which the head-injured subject never fully recovers from

the initial postinjury drop in blood pressure. The deterioration of brain metabolism that follows the combination of hypoxemia or hypotension with experimental brain injury underscores the necessity for prompt restoration of oxygenation, perfusion pressure, and ventilation in patients with head injuries.

Traumatic brain injury initiates a variety of additional biochemical changes, each of which could contribute to enhanced vulnerability to subsequent vascular or tissue injury. Free oxygen radicals may interfere with the normal ability of the cerebral vasculature to dilate in response to declining blood pressure.²¹ In cats subjected to head injury, hemorrhagic hypotension reduces CBF more than in control animals.^{22,23} Compensatory cerebral vasodilation also fails to occur in response to hemodilutional resuscitation after hemorrhage and head trauma.²⁴

Increases in excitatory amino acids such as glutamate and aspartate, which activate *N*-methyl-D-aspartate (NMDA) receptors (among others), correlate with the severity of brain injury in rats and with reduced availability of high-energy phosphate compounds.²⁵ Evidence also suggests that cholinergic mechanisms mediate enhanced vulnerability to global cerebral ischemia after experimental head injury.²⁶ The metabolism of cell membrane lipids generates prostaglandins, leukotrienes, and oxygen free radicals. The presence of extravascular blood also generates free radicals. Experimental brain injury increases thromboxane A_2 , a powerful vasoconstrictor and platelet aggregator, and also increases brain levels of prostaglandin E_2 , a compound that has been associated with reduced motor activity.²⁷ Finally, some classes of endogenous and exogenous opioid agonists and antagonists may aggravate, while others may limit, brain injury. For example, naloxone, a nonspecific opiate antagonist, significantly worsens outcome after fluid-percussion head injury in rats, whereas morphine reduces deficits.²⁸

Although preadmission hypotension and hypoxemia are associated with worse outcome, such insults continue to occur after admission to the hospital, raising the possibility of limiting secondary ischemic injury, either through effective prevention or through preischemic pharmacological intervention. Among a series of 100 patients in an intensive care unit after mild, moderate, or severe traumatic brain injury, hypotension (mean arterial pressure [MAP] ≤ 70 mm Hg) occurred in 76%; reduced CPP (≤ 60 mm Hg), defined as MAP minus ICP, occurred in 78%; and hypoxemia ($\leq 90\%$ hemoglobin saturation) occurred in 43%.²⁹ In eight patients who un-

derwent ICP monitoring after severe traumatic brain injury without evidence on cranial CT of intracranial hypertension, ICP exceeded 20 mm Hg for more than 5 minutes in seven patients, and CPP was less than 60 mm Hg for more than 5 minutes in five patients.³⁰ Therefore, systemic insults that could worsen outcome by inducing cerebral ischemia occur commonly in patients with head injuries, both at the time of admission to the hospital and during intensive care.

To further support the likelihood that postadmission cerebral ischemia contributes to poor outcome after traumatic brain injury, patients who survived head injury demonstrated evidence of ischemic injury on CT. In 1987, researchers³¹ monitored the cerebral metabolic rate for oxygen (CMRO₂) and the cerebral metabolic rate for lactate in 44 patients with severe head injuries, and correlated those results with changes in cranial CT scans. Although 27 patients developed low-density areas surrounding intracranial hematomas, 6 others developed evidence of cerebral infarction not associated with hematomas. Low CMRO₂ and net cerebral lactate production were found to be weak predictors of later infarction.

Recognition (via continuous monitoring) of cerebral ischemia during intensive care offers the possibility of prompt intervention. Several investigators have examined the ability of jugular venous bulb monitoring to detect ischemia. The jugular venous bulb provides "mixed" cerebral venous blood, relatively uncontaminated by extracranial venous effluent. Like mixed systemic venous sampling, cerebral venous oxygenation provides information that suggests but does not prove cerebral ischemia. In 45 patients cared for in intensive care units after traumatic brain injury, 33 episodes of jugular desaturation below 50% were documented.³² One researcher³³ reported 121 episodes of combined arterial and jugular venous desaturation during intensive monitoring of 69 patients with severe head injuries; of the 121 episodes, 32 (26.5%) responded poorly to treatment. Prolonged desaturation was associated with deterioration of the GCS score.³³ More importantly, among 102 patients with GCS scores below 9 on admission or on the first day thereafter, one episode of jugular venous desaturation (< 50% for > 10 min) was associated with good recovery or moderate disability in 23%, more than one episode with good recovery or moderate disability in 12%, and the absence of jugular venous desaturation with good recovery or moderate disability in 46%.³⁴

Systemic and Organ-System Responses

Clinical head injury is associated with sympathetically mediated hypertension, tachycardia, and increased cardiac output. The more severe the level of isolated clinical head injury (ie, the lower the GCS score), the greater the increase in plasma norepinephrine levels and heart rate.³⁵ However, experimental data²⁰ suggest that beyond a threshold impact level, the increase in catecholamines no longer linearly correlates with the intensity of the impact. In sympathetically stimulated patients with head injuries, β -adrenergic blockade reduces heart rate, cardiac index, and circulating catecholamines.³⁶

In addition to the characteristic hyperdynamic response associated with head injury, hypertension also accompanies severe increases in ICP, a response first recognized by Cushing in 1903³⁷ and now known as the Cushing phenomenon. If permitted to progress and depending on the direction in which the brain expands, increasing intracranial hypertension may produce medullary compression and precipitous systemic hypotension. Management of the Cushing phenomenon consists of therapy to reduce ICP, thereby interrupting the reflex response. If systemic hypertension is secondary to the Cushing phenomenon, attempts to control blood pressure using vasodilators may both reduce MAP and increase ICP, thereby further compromising cerebral perfusion.

Patients with head injuries also undergo stress-induced hyperglycemia and accelerated protein wasting. The frequent occurrence of fever and hypoalbuminemia has been attributed to endothelial cell injury.³⁸

Pulmonary Effects

Acute head injury is associated with a variety of respiratory problems, some potentially fatal (eg, apnea, central neurogenic pulmonary edema, and altered breathing patterns), as a result of associated injuries, fat embolism syndrome, pulmonary contusion, and hemopneumothorax. Hypoxemia may occur even in patients who lack auscultatory or radiographic evidence of pulmonary compromise, apparently because of failure of mechanisms that regulate ventilation-perfusion matching.³⁹ Central neurogenic pulmonary edema, a rare, acutely life-threatening complication usually seen accompanying severe injury, may result from acute left atrial hypertension or increased pulmonary capillary permeability.⁴⁰ Management of central neurogenic

pulmonary edema necessitates prompt correction of intracranial hypertension, if present, in addition to therapy to improve oxygenation.

Coagulopathy

Laboratory evidence of disseminated intravascular coagulation is reported in nearly one fourth of patients with head injuries.⁴¹ Patients with higher concentrations of fibrin degradation products have poorer functional outcomes and are more likely to develop the adult respiratory distress syndrome.⁴²

Cerebral Circulatory Responses

Acute head injury of sufficient severity to produce coma is associated with moderately decreased CBF, markedly depressed $CMRO_2$, and highly variable autoregulation and carbon dioxide reactivity. The uninjured cerebral circulation is responsive to changes in metabolic demand; CPP; the partial pressure of carbon dioxide, arterial ($Paco_2$); and the partial pressure of oxygen, arterial (Pao_2). Cerebral blood flow normally is coupled to $CMRO_2$, which varies directly with body temperature and the level of brain activation; that is, $CMRO_2$ is increased by fever, seizures, or pain. Cerebral blood flow remains constant (approximately 50 mL/100 g/min in adults) in the normal brain as CPP changes over a range of approximately 50 to approximately 130 mm Hg. In normal individuals, over a range of $Paco_2$ from 20 to 80 mm Hg, CBF will be acutely halved if $Paco_2$ is halved and will double if $Paco_2$ is doubled. That relationship is apparently intact in most patients with head injuries. Pao_2 exerts little effect on CBF unless Pao_2 declines below 60 mm Hg (hemoglobin saturation < 90%). Below that level, CBF increases abruptly.

In 1991, one group of researchers⁴³ reported that CBF was less than a critical value of 18 mL/100 g/min in one third of measurements made within 6 hours of injury in 106 patients with head injuries. Moreover, the average difference in cerebral arteriovenous oxygen content ($AVDO_2$) was greater than normal during this critical early interval (Figure 16-4), suggesting that CBF was indeed inadequate.⁴³ Later, in the days after injury, some patients with head injury demonstrate depressed levels of both $CMRO_2$ and CBF, while others demonstrate uncoupling, with CBF substantially in excess of $CMRO_2$ (Figure 16-5).⁴⁴ Nearly 90% of patients younger than 18 years of age demonstrate relative cerebral hyperemia, defined as CBF that exceeds metabolic

demand, or even exceeds normal values, at some point during intensive monitoring.⁴⁵

Experimental and clinical data⁴⁶ demonstrate that CBF after head trauma frequently is MAP dependent rather than MAP independent (Figure 16-6). Most patients with mass lesions demonstrate defective autoregulation; conversely, autoregulation remains intact in many patients without intracranial mass lesions.⁴⁶ In patients with head injuries in whom autoregulation is intact, mannitol reduces ICP and does not change CBF; if autoregulation is defective, ICP changes little and CBF increases.⁴⁷

In many patients, reduced CBF appears to represent appropriate coupling between low $CMRO_2$ and low CBF rather than cerebral ischemia.⁴⁴ However, patients with low but coupled CBF may be vulnerable to excessive vasoconstriction during acute hyperventilation. Nearly 20% of patients develop a wide cerebral $AVDO_2$ during hyperventilation, sug-

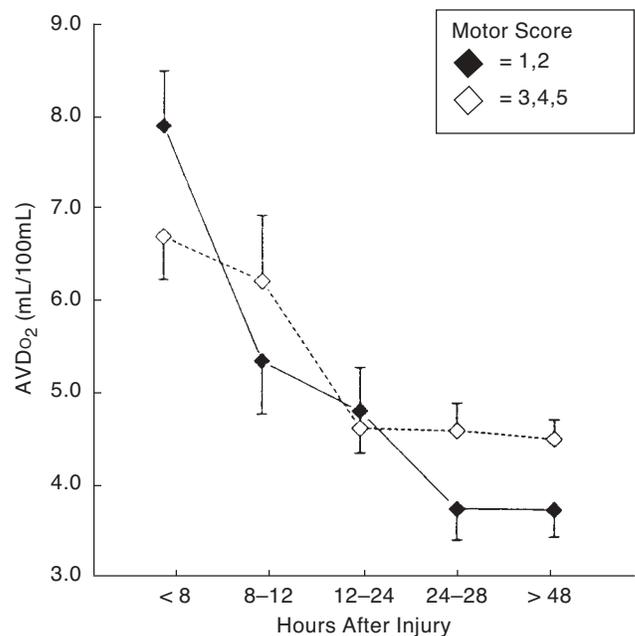


Fig. 16-4. Time course of mean arteriovenous oxygen difference ($AVDO_2$) in patients with head injuries and with motor scores of 1 or 2, and those with motor scores of 3 to 5 (motor score is measured as the best response to a painful stimulus: 1 = no response; 2 = extension; 3 = abnormal flexion; 4 = flexion withdrawal; 5 = localizing response). Error bars represent the standard error of the mean. Reprinted with permission from Bouma GJ, Muizelaar JP, Choi SC, Newlon PG, Young HF. Cerebral circulation and metabolism after severe traumatic brain injury: The elusive role of ischemia. *J Neurosurg.* 1991;75:688.

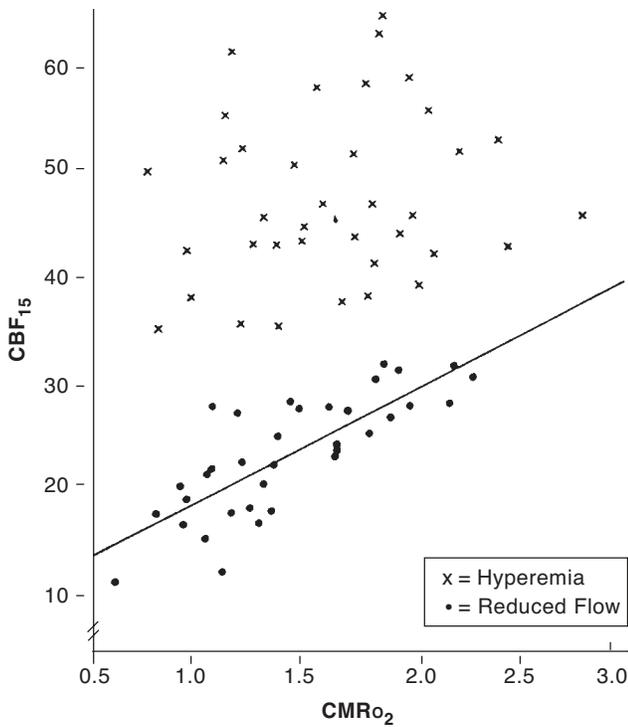


Fig. 16-5. In patients who have sustained closed head injury, both the cerebral metabolic rate of oxygen consumption ($CMRO_2$, the abscissa) and cerebral blood flow (CBF, based on ^{133}Xe clearance integrated over 15 min, the ordinate) are reduced. (Normal values are 3.4 mL/100 g/min and 50 mL/100 g/min, respectively). In some patients (closed circles), $CMRO_2$ and CBF appear to be reduced to a similar extent (ie, coupled). In others (represented by the x's), global CBF is higher than apparently is necessary to meet metabolic demand (uncoupled). Reprinted with permission from Obrist WD, Langfitt TW, Jaggi JL, Cruz J, Gennarelli TA. Cerebral blood flow and metabolism in comatose patients with acute head injury: Relationship to intracranial hypertension. *J Neurosurg.* 1984;61:247.

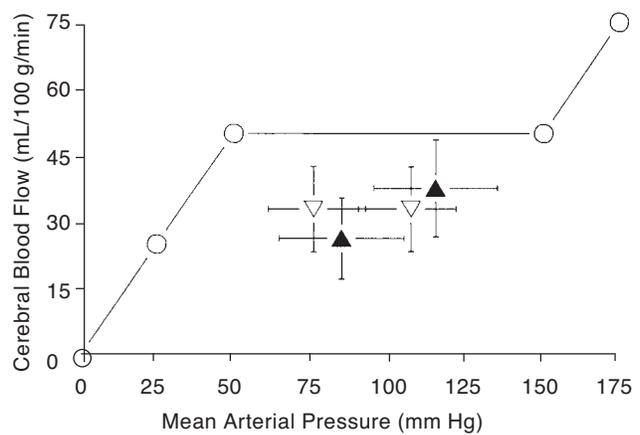


Fig. 16-6. In comparison to the normal autoregulatory curve (open circles), adult patients who have sustained closed head injury have reduced flow, and in some cases (closed triangles), impaired autoregulation. Other patients have reduced flow and preserved autoregulation (open triangles). Data source: Muizelaar JP, Lutz HA III, Becker DP. Effect of mannitol on ICP and CBF and correlation with pressure autoregulation in severely head-injured patients. *J Neurosurg.* 1984;61:700–706.

gesting that hyperventilation therapy occasionally may adversely influence cerebral perfusion (Table 16-1).⁴⁴ If increased ICP develops in patients with a wide cerebral $AVDO_2$, other measures to reduce ICP, such as sedation or osmotic diuresis, produce better matching of cerebral oxygen supply and demand than further hyperventilation. If CBF measurements are unavailable, the calculation of the cerebral oxygen extraction or the lactate extraction may provide clinically useful information regarding the adequacy of cerebral oxygen delivery.^{31–33,48–50}

TABLE 16-1

FINDINGS IN 10 PATIENTS WITH WIDE ARTERIOVENOUS OXYGEN DIFFERENCES AFTER HEAD INJURY*

| Hemodynamic Variable | Hyperventilated Patients* | Normal Value ($Paco_2 \approx 40$ mm Hg) |
|----------------------|---------------------------|---|
| $CMRO_2$ | 1.9 ± 0.5 | 3.3 ± 0.4 |
| CBF_{15} | 18.6 ± 4.4 | 53.3 ± 6.8 |
| $AVDO_2$ | 10.5 ± 0.7 | 6.3 ± 1.2 |
| VPo_2 | 22.3 ± 1.8 | 37.5 ± 5.6 |

*Values are means \pm standard deviations obtained 10 to 85 h after the injury
 $AVDO_2$: arteriovenous oxygen differences (in vol %); CBF_{15} : mean cerebral blood flow based on ^{133}Xe clearance integrated over 15 min; $CMRO_2$: cerebral metabolic rate for oxygen (in mL/100 g/min); VPo_2 : jugular venous oxygen tension (in mm Hg)
 Reprinted with permission from Obrist WD, Langfitt TW, Jaggi JL, Cruz J, Gennarelli TA. Cerebral blood flow and metabolism in comatose patients with acute head injury: Relationship to intracranial hypertension. *J Neurosurg.* 1984;61:251.

CARE OF THE PATIENT WITH TRAUMATIC BRAIN INJURY

Initial Care

Two assessment systems can be used to understand the reports that characterize the initial management of traumatic brain injury. The first, the GCS, was originally developed to permit comparisons among series of patients with traumatic brain injuries, based on their initial clinical presentation (Table 16-2).⁵¹ Consisting of three major categories (eye opening [1–4 points], verbal responses [1–5 points], and motor function [1–6 points, assessed in the best responding limb]), the GCS score ranges from 3 to 15. Mild head injury is defined as scores ranging from 13 to 15, moderate as scores from 9 to 12, and severe as 8 or less. The GCS constitutes a reliable index of overall brain function with minimal interobserver variability. The second system, the Glasgow outcome scale (GOS) defines five categories: good recovery, moderate

disability, severe disability, persistent vegetative state, and death (Table 16-3).⁵² Defined in these terms, about 2,000 to 3,000 patients per million population per year come to medical attention; 85% to 90% are in the mild category, 5% to 10% in the moderate, and 5% in the severe.²⁹ The GCS is a powerful predictor of the final outcome (Table 16-4).⁵³

Prompt application of basic life support may prevent secondary hypoxic brain damage.⁵⁴ Secondary insults such as anemia, hypotension, hypoxemia, and hypercarbia, alone or in combination, occur in nearly 50% of comatose patients with head injuries (Table 16-5).² Thus, tracheal intubation, positive-pressure ventilation with oxygen, and systemic hemodynamic resuscitation should be initiated promptly when indicated, and should not be

TABLE 16-2
GLASGOW COMA SCORE

| Component | Response | Score |
|------------------------------------|---------------------------|---------------|
| Eye Opening | Spontaneously | 4 |
| | To verbal command | 3 |
| | To pain | 2 |
| | None | 1 |
| | Subtotal | (1–4) |
| Motor Response (best extremity) | Obeys verbal command | 6 |
| | Localizes pain | 5 |
| | Flexion-withdrawal | 4 |
| | Flexion (decortication) | 3 |
| | Extension (decerebration) | 2 |
| | No response (flaccid) | 1 |
| | Subtotal | (1–6) |
| Best Verbal Response | Oriented and converses | 5 |
| | Disoriented and converses | 4 |
| | Inappropriate words | 3 |
| | Incomprehensible sounds | 2 |
| | No verbal response | 1 |
| | Subtotal | (1–5) |
| | Total | (3–15) |

Reprinted with permission from Teasdale G, Jennett B. Assessment of coma and impaired consciousness. *Lancet*. 1974;2:81–84.

TABLE 16-3
GLASGOW OUTCOME SCALE

| Patient Outcome | Description |
|-----------------------------|---|
| Good Recovery | Normal or minimally impaired patients who have returned to school, university, or former occupation, or are capable of managing their households |
| Moderate Disability | Patients who can perform the tasks of daily living but are no longer able to work or attend school |
| Severe Disability | Patients who require assistance to perform the tasks of daily living but do not require institutional care |
| Persistent Vegetative State | Patients remain unresponsive and speechless for weeks or months; absent function in the cerebral cortex (as judged behaviorally) although cortex may appear structurally intact |
| Death | Patients do not regain consciousness; most deaths occur within 48 h |

Reprinted with permission from Jennett B, Bond M. Assessment of outcome after severe brain damage: A practical scale. *Lancet*. 1975;1:480–484.

TABLE 16-4
RELATION OF ACUTE GLASGOW COMA SCALE SCORE TO GLASGOW OUTCOME SCALE SCORE*

| GOS Score | GCS Score | GCS Score | GCS Score | Total Cases |
|------------------|------------|------------|------------|-------------|
| | 3-4 | 5-6 | 7-9 | |
| No. (%) of Cases | | | | |
| Dead/PVS | 15 (78.9%) | 19 (45.2%) | 9 (25.7%) | 43 |
| SD/MD/GR | 4 (21.2%) | 23 (54.8%) | 26 (74.3%) | 53 |
| Total Cases | 19 | 42 | 35 | 96 |

*Significance: $P < .0001$ (chi-square)

GCS: Glasgow Coma Scale; GOS: Glasgow Outcome Scale; PVS: persistent vegetative state; SD: severe disability; MD: moderate disability; GR: good recovery

Reprinted with permission from Jaggi JL, Obrist WD, Gennarelli TA, Langfitt TW. Relationship of early cerebral blood flow and metabolism to outcome in acute head injury. *J Neurosurg.* 1990;72:178.

deferred until the patient arrives at a trauma center.

Intubation of patients with head injuries, who also may have unrecognized cervical spine injuries, presents a challenge in which several therapeutic conflicts must be balanced. Inadequate ventilation and oxygenation must be promptly reversed. Ideally, profound sedation and muscle relaxation limit increases in ICP associated with endotracheal intubation; however, hypovolemia resulting from associated injuries may contraindicate effective sedation. Intubation may be technically difficult owing to trauma to facial structures or to trismus. In such

cases (and if time permits), it may be practical to secure an airway using a fiberoptic endoscope. Occasionally, cricothyroidotomy, transcricothyroid oxygen insufflation, or formal tracheostomy may be required. Facial fractures constitute a relative contraindication to nasotracheal intubation.⁵⁵

Despite legitimate concern that the depolarizing muscle relaxant succinylcholine may increase ICP,⁵⁶ its more rapid onset of action, compared with that of nondepolarizing muscle relaxants, may make it necessary to use succinylcholine for immediate intubation of an unstable patient. After intubation, the patient should be ventilated with an oxygen-

TABLE 16-5
INFLUENCE OF REMEDIABLE CAUSES OF SECONDARY INJURY ON OUTCOME AFTER HEAD INJURY

| Secondary Insult | Definition | Poor Outcome* (%) |
|---------------------------|----------------------------|-------------------|
| Hypoxemia | $PaO_2 < 60$ mm Hg | 59 |
| Hypotension | $SBP < 90$ mm Hg | 65 |
| Anemia | Hct $< 30\%$ | 62 |
| Hypercarbia | $Paco_2 > 45$ mm Hg | 78 |
| Intracranial hypertension | ICP > 20 , reducible | 45 |
| | ICP > 20 , not reducible | 95 |

*Poor outcome: severe disability, persistent vegetative state, or death

Hct: hematocrit; ICP: intracranial pressure; SBP: systolic blood pressure

Adapted with permission from Miller JD, Butterworth JF, Gudeman SK, et al. Further experience in the management of severe head injury. *J Neurosurg.* 1981;54:292.

enriched gas mixture. Minute ventilation should be adjusted to maintain P_{aCO_2} at or below 40 mm Hg unless clinical evaluation or monitoring indicates severe, acute, intracranial hypertension. Routine hyperventilation may not improve outcome and may even be deleterious.⁵⁷

No resuscitation fluid has proven ideal in patients with multiple trauma and head injury. Several studies with animals have demonstrated lower ICP or higher CBF when hypertonic saline solutions are substituted for isotonic fluids (Figure 16-7).^{58,59} Clearly, hypotonic solutions, which increase brain tissue volume, should be avoided. The role of colloid-containing solutions is controversial. Although for years clinicians have considered colloids less likely to increase ICP after head injury, data suggest that changes in colloid osmotic pressure exert minimal effects on brain water or ICP in animals both with and without intracranial pathol-

ogy.^{60,61} Although rapid resuscitation with 6.0% hydroxyethyl starch increases ICP less than hemodynamically comparable resuscitation using lactated Ringer's solution,⁶² the difference is probably due to the fact that lactated Ringer's solution is slightly hypotonic.

The neurological examination, preferably conducted before induction of neuromuscular paralysis, should assign a GCS score and, in all comatose patients, assess brainstem function, (ie, pupillary light responses and oculocephalic or vestibuloocular reflexes). Patients should undergo a more complete neurological examination as time permits, but as expeditiously as possible. Changes in the examination may be localizing and may dictate intervention, even if they do not change the GCS score.

Clearly, the highest priority for comatose patients with head injuries is to determine the need for craniotomy. Patients with open or penetrating inju-

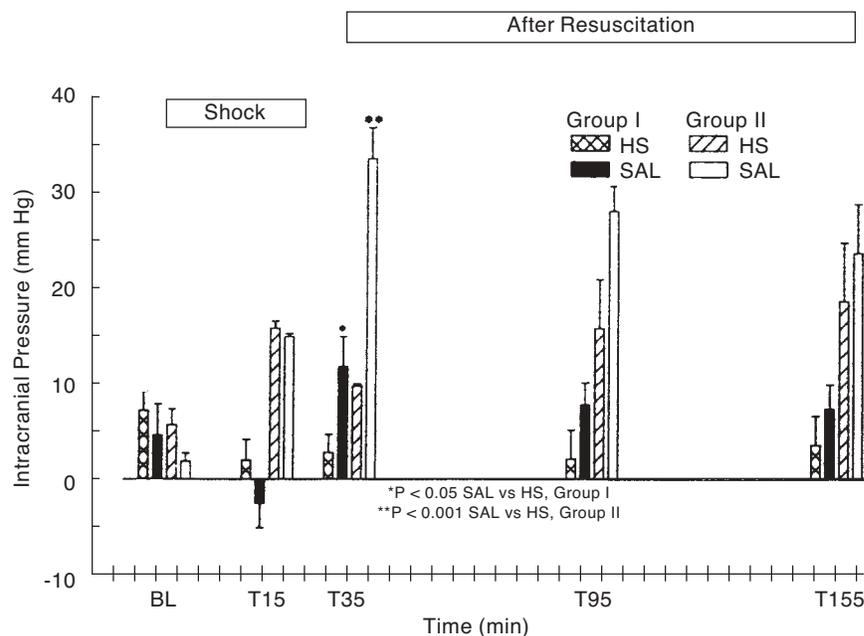


Fig. 16-7. Response of intracranial pressure (ICP) to resuscitation from shock with 7.2% hypertonic saline (HS) or 0.8% saline (SAL) in animals without (Group 1) and with (Group 2) a right hemispheric intracranial mass. Intracranial hypertension (15 mm Hg) in Group 2 was induced by inflation of a right-hemispheric subdural balloon to which additional saline was added as necessary to maintain ICP at 15 mm Hg during shock. Shock extended from 0 time to T30 (30 min after initiation of shock). BL denotes baseline; T15 refers to 15 min after the beginning of a 30-min shock interval; T35, T95, and T155 refer to 5, 60, and 120 min after a single-bolus resuscitation. After a 30-min interval of hemorrhagic shock at a mean cerebral perfusion pressure of 35 mm Hg, rapid resuscitation consisted of 0.8% SAL (54 mL/kg) or 7.2% HS (6.0 mL/kg). In animals both with and without intracranial mass lesions, ICP was significantly greater immediately after resuscitation in animals resuscitated with 0.8% SAL than in those resuscitated with 7.2% HS; differences quickly became statistically insignificant. Reprinted with permission from Prough DS, Whitley JW, Taylor CL, Deal DD, DeWitt DS. Regional cerebral blood flow following resuscitation from hemorrhagic shock with hypertonic saline: Influence of a subdural mass. *Anesthesiology*. 1991;75:323.

ries require prompt surgical debridement or, if local facilities are limited and the patient is sufficiently stable to transport, transfer to a secondary- or tertiary-care facility. All patients who do not obey simple commands (in the absence of drug or alcohol intoxication) require prompt CT scanning to exclude intracranial mass lesions. Patients who require evacuation of subdural hematomas but whose craniotomy is delayed have a far worse outcome than those patients whose hematomas are promptly evacuated.⁶³ Before the widespread availability of CT and MRI, neuroradiological evaluation could rapidly be accomplished through either cerebral angiography or air ventriculography. These latter two techniques, now rarely used, should be considered if immediate scanning is unavailable, as may well be true under battlefield conditions. In such circumstances, a single-shot arteriogram, performed on initial evaluation of a patient with a high likelihood of an intracranial mass lesion, could be life-saving. The use of near-infrared spectroscopy to diagnose acute intracranial hematomas is yet to be completely evaluated, although preliminary data⁶⁴ are encouraging.

Adult patients who have *briefly* lost consciousness, but who subsequently have a completely normal neurological examination, probably do not need a CT scan in the absence of other clinical indications. The management of patients who have intermediate levels of injury requires repeated evaluation and careful observation.

Diagnostic priorities for other organ systems should proceed as indicated by the description of the traumatic event and by the general physical examination. In all comatose patients with head injuries, unless they are known to have suffered *only* an isolated head injury, abdominal CT scan or diagnostic peritoneal lavage⁶⁵ should be performed to detect occult intraabdominal injury. Patients should be considered to have cervical spine fractures until this possibility has been excluded by a cross-table lateral neck radiograph (or a cervical CT scan) that adequately shows all cervical vertebrae. Some degree of immobilization (eg, using a hard collar) should be maintained until the patient is sufficiently cooperative to perform his own neck manipulation for flexion and extension films.

Although shock sometimes occurs in association with an isolated head injury, other causes of hypotension that must be excluded⁶⁵ include intraabdominal hemorrhage (evaluated by CT scan or diagnostic peritoneal lavage), intrathoracic hemorrhage (evaluated by chest radiography, CT scan, or aortography), long-bone fractures (assessed using a

skeletal survey), myocardial ischemia or cardiac decompensation (evaluated using electrocardiography or pulmonary arterial catheterization), and pericardial tamponade (suggested by a paradoxical pulse, central venous or pulmonary artery pressure monitoring, chest radiography, echocardiography, or pericardiocentesis). Untreated shock portends a worse prognosis.¹

Anesthetic Management

Military trauma anesthesia personnel may be confronted with a spectrum of casualties with head injuries, from those who arrive from the battlefield only moments after injury to those who have been thoroughly evaluated and stabilized. The patient may be intubated, sedated, and paralyzed, or may be awake but combative. Surgery may be required for emergent evacuation of an intracranial hematoma or may be directed at treatment of injuries to other organ systems. Therefore, it is obvious that anesthesia personnel participate in a continuum of care that includes the considerations discussed elsewhere in this chapter. However, for purposes of brevity, this section will address questions of anesthetic management as they apply to unintubated patients presenting for surgery with moderate-to-severe head injuries.

Preoperatively, little time may be available for evaluation. Therefore, the following assumptions are appropriate. Airway obstruction, hypoventilation, and hypoxia are likely, and oxygen requirements are increased. Both depression of consciousness and injury increase the risk of aspiration of stomach contents. Hypotension, uncommon in a patient with an isolated head injury, implies hemorrhage from associated injuries or spinal cord injury and markedly increases the risk of secondary cerebral ischemia in patients with head injuries. Many patients with head injuries are hypertensive because of catecholamine release. Some patients are hypertensive because of physiological reflexes that attempt to maintain CPP as ICP increases. Only severe hypertension necessitates treatment; reflex hypertension caused by intracranial hypertension requires treatment of increased ICP. Direct vasodilators (ie, nitroglycerine, nitroprusside, and hydralazine) increase CBF and ICP. β -Adrenergic blocking agents such as esmolol or propranolol or mixed α - and β -adrenergic blocking agents such as labetalol minimally affect ICP.

Monitoring should be determined by the severity of the injuries and the availability of technologically sophisticated equipment. Resuscitation and

stabilization should not be delayed in an effort to establish better monitoring. If time and circumstances permit, direct arterial pressure monitoring and central venous pressure monitoring are valuable. Pulse oximetry and capnography provide early warning of deterioration in gas exchange. Automated, noninvasive blood pressure monitoring may free anesthesia personnel for other activities. If practical, ICP monitoring may be useful if a patient with severe head injuries requires extensive surgery for other injuries.

Volume resuscitation should not be constrained by fears of increasing cerebral edema. At the present time, the most appropriate fluid for the initial treatment of hemorrhagic shock in a patient with head injuries is 0.9% saline, a slightly hypertonic solution that should result in no increase in brain water or ICP in areas of brain in which the blood-brain barrier is intact. Blood products should be given as indicated and available. More highly hypertonic solutions, which decrease brain water and ICP in experimental animals and decrease ICP in humans, may become the treatment of choice in the future.

Anesthetic induction must preserve CPP. Avoidance of hypotension is more important than reduction of small increases in ICP. Potential induction agents include thiopental, etomidate, benzodiazepines, and ketamine. Barbiturates decrease $CMRO_2$, reduce CBF, and reduce ICP. However, large dosages of barbiturates may reduce blood pressure, especially in hypovolemic patients, and may slow emergence from general anesthesia. Etomidate provides prompt induction and is usually well tolerated even in moderately hypovolemic patients. Benzodiazepines decrease $CMRO_2$ and CBF, and usually preserve blood pressure. Large dosages may slow emergence. Ketamine, which increases CBF and ICP, would rarely be appropriate in a patient with head injuries. Cricoid pressure should be applied during induction and intubation.

In a patient who is hemodynamically resuscitated, a reasonable sequence is to administer succinylcholine (or a large dose of a nondepolarizing agent), thiopental, and fentanyl while maintaining cricoid pressure. In a severely hypovolemic patient, succinylcholine and a small dose of a narcotic or intravenous lidocaine to blunt the hemodynamic response to intubation would be preferable. Although succinylcholine may increase ICP, expeditious control of the airway is more important.

Volatile anesthetics decrease $CMRO_2$, but they also increase CBF and should only be administered

to patients at risk of intracranial hypertension after hyperventilation has been established. Isoflurane appears to be most satisfactory. Although nitrous oxide causes small increases in CBF and $CMRO_2$, the reduced fraction of inspired oxygen (FIO_2) necessitated by nitrous oxide administration limits its use in patients with lung injury.

Management in the Intensive Care Unit

The primary goals of management in the intensive care unit are to prevent secondary neurological injury and to limit complications that develop in other organ systems.

Cerebral Circulatory Considerations

Much of the management of casualties with acute head injuries is intended to maintain adequate cerebral perfusion. Because CBF is not routinely measured, most cerebral circulatory information is inferred from measurements of $Paco_2$, blood pressure, and ICP. Measurement of ICP, combined with measurement of MAP, permits calculation of CPP according to the equation:

$$CPP = MAP - ICP$$

Cerebral ischemia, defined as inadequate cerebral oxygen delivery (CDO_2), can result from a critical reduction of any of the components, including CBF, hemoglobin concentration, and arterial hemoglobin saturation (So_2). Uniquely susceptible to oxygen deprivation, the brain constitutes only approximately 2% of total body weight, but it receives 15% of the cardiac output and accounts for 15% to 20% of total oxygen consumption.

Brain Monitoring

Most global cerebral insults, occurring secondary to hypotension, hypoxemia, or cardiac arrest, are readily detected by systemic monitors. Therefore, brain-specific monitors can provide additional information primarily in situations such as cerebral trauma, in which regional cerebral oxygenation may be impaired despite adequate systemic oxygenation and perfusion.

Monitoring devices facilitate early recognition of physiological derangements that would produce complications unless effective treatment were provided. The application of brain-monitoring devices in patients with head injuries therefore presupposes certain assumptions:

1. Reduced C_{DO_2} ($CBF \cdot \text{arterial } O_2 \text{ content}$) is associated with avoidable neurological morbidity.
2. The proportion of patients who will develop avoidable injury is sufficiently large to justify extensive application of brain-monitoring devices.
3. Thresholds for intervention can be defined based on experimental and clinical evidence.

Brain monitors assess, directly or indirectly, cerebral function, cerebral perfusion, or brain metabolism (Exhibit 16-1). The role of brain monitoring has been incompletely defined in acute head injury. Many brain monitors, such as those that record CBF, evoked potentials, and computer-processed electroencephalograms, are technically challenging and difficult to apply in combat situations unless specifically trained personnel are available. More importantly, no generally accepted protocols have been developed that establish clinically important thresholds for monitored variables other than ICP, define appropriate interventions to improve the monitored variables, and have been proven to improve outcome.

Operational Characteristics of Monitors

Thresholds of CBF that correlate with various clinical outcomes, physiological changes, and changes in monitored variables have been defined (Table 16-6). It is impossible to predict with certainty if even severe changes in function will be followed by further cerebral damage. Moreover, because various brain regions are selectively vul-

EXHIBIT 16-1
TYPES OF CEREBRAL MONITORING

Function

- Evoked potentials
- Electroencephalogram
 - Raw
 - Processed

Perfusion

- Cerebral blood flow
- Velocity
- Intracranial pressure

Metabolism

- Oxygen extraction
- Jugular bulb saturation
- Near-infrared spectroscopy

nerable to injury, regional ischemia and infarction may develop without producing changes in monitored variables. The complexity and heterogeneity of brain tissue virtually preclude development of a single, perfectly predictive brain monitor.

Evoked Potentials. Sensory evoked potentials (EPs), which include somatosensory evoked potentials (SSEPs), brainstem auditory evoked potentials (BAEPs), and visual evoked potentials (VEPs), and, more recently, motor evoked potentials (MEPs), provide a visual representation of the response of

TABLE 16-6
CLINICAL, PATHOPHYSIOLOGICAL, AND MONITORING THRESHOLDS IN CEREBRAL ISCHEMIA

| Cerebral Blood Flow (mL/100 g/min) | Clinical Findings | Pathophysiological Changes | Electrophysiological Changes |
|------------------------------------|----------------------|--------------------------------|------------------------------|
| 50 | Normal | | |
| 23 | Reversible paralysis | | EEG slowing, EP change |
| 20 | | Na^+/K^+ pump dysfunction | |
| 18 | Permanent paralysis | | EEG flat |
| 15 | | | EP absent |
| 10 | | K^+ efflux, Ca^{++} influx | |

EEG: electroencephalogram; EP: evoked potentials

neural structures to stimulation. EPs are generated by integrated neural networks, including the initial sensory structure, the transmitting pathways, and cortical and subcortical stimulus-processing centers. As the response to a stimulus is transmitted centrally, characteristic waveforms are generated that correspond to structures through which the stimulus passes. EPs, especially BAEPs and MEPs, are relatively refractory to insult, although they are modified by sedatives, narcotics, and anesthetics, as well as by trauma, hypoxia, or ischemia. Because obliteration of EPs occurs only under conditions of profound cerebral ischemia or mechanical trauma, EP monitoring is one of the most specific ways to assess neurological integrity. However, EPs are less sensitive to smaller changes in cerebral (or spinal cord) oxygen availability.

In patients with head trauma^{66,67} or spinal cord injury,^{68,69} EP monitoring has been employed primarily as a diagnostic and prognostic aid. Multimodality EPs improve the prognostic accuracy of the clinical examination and measurement of ICP in patients with head injuries.⁶⁷ With impending brain death, cortical SSEPs disappear first; BAEPs disappear only when brain death is imminent.⁷⁰ Because BAEPs are resistant to the effects of barbiturates, EPs assist in assessing neurological status even in patients in barbiturate coma.

Electroencephalographic Monitoring. Computerized compression of electroencephalographic (EEG) data permits frequent, repetitive assessment with a minimum of specific training. In the most commonly employed software programs, the data are displayed as a compressed spectral array (CSA) or density spectral array (DSA). Because the EEG is sensitive to drug effects, either unprocessed or processed EEG monitoring can be used to assess sedation in critically ill patients as well as to provide early evidence of seizure activity or cerebral ischemia.

This equipment remains relatively expensive, depends on the availability of dedicated technicians, and requires a modest level of sophistication for interpretation of changes. Like EP monitoring, EEG monitoring appears to have little value for either surveillance or goal-directed therapy in patients with head injuries, in contrast to its possible value for intraoperative monitoring.

Cerebral Blood Flow Monitoring. Although measuring the cerebral blood flow via xenon 133 clearance is a powerful research tool, these measurements do not yet constitute a clinically useful monitor. Despite the prognostic value of CBF mea-

surements in patients who have suffered closed head injury, no role has developed for primary diagnosis, surveillance, or goal-directed management. Among the obstacles to wider use are the necessity for administering a radionuclide; the technically demanding nature of the measurements; and the relatively long interval (5–15 min) of stable conditions required to perform a single measurement, after which an additional several minutes must pass before subsequent measurements can be performed.

Intracranial Pressure Monitoring. Because ICP functions as the outflow pressure for the cerebral circulation (assuming jugular venous pressure is lower), ICP bears an important relationship to CBF, although CBF cannot be directly inferred from knowledge of ICP and MAP. In more than one third of patients with head injuries, ICP exceeds 20 mm Hg, a level thought to be a critical threshold for clinical intervention.⁷¹ If untreated, intracranial hypertension will increase brain injury by causing herniation or cerebral hypoperfusion. Although ICP monitoring has been credited by some investigators⁷² with an improving prognosis from acute closed head injury, others⁷³ question whether concurrent improvements in management, rather than ICP monitoring, explain the improvement. Although the subarachnoid screw⁷⁴ occasionally generates erroneous information,⁷⁵ measurement of ICP is usually considered to be a fundamental part of the care of patients with severe closed head injury (ie, GCS score \leq 8).⁷⁶ A practical, reliable fiberoptic ICP monitor has been developed (manufactured by Camino Laboratories, San Diego, Calif.) that works well as an intraventricular, subdural, or brain tissue monitor⁷⁷ and has relatively little drift of calibration over periods of 5 days or less.⁷⁸

Unlike the other modalities previously discussed, ICP monitoring has been used for surveillance and for goal-directed therapy. The information is especially valuable in patients in whom neuromuscular blocking agents are administered as part of treatment to reduce ICP, because such agents preclude a comprehensive neurological examination. Clinicians have applied systematic, although institutionally specific, protocols for avoidance of intracranial hypertension and for reduction of increased ICP when a threshold of 15 or 20 mm Hg is exceeded. Decisions about diuretics, hyperventilation, changes in the patient's position, and additional diagnostic procedures may be influenced by ICP information. If intracranial hypertension is refractory to conventional therapy, ICP monitoring is one of the alterna-

tive techniques used to titrate barbiturate coma,⁷⁹ although prophylactic barbiturate coma has failed to improve neurological outcome after head injury.⁸⁰ Whenever ICP monitoring is employed, the potential complications, the most important of which is infection, must be considered. The risk of intracranial infection appears to be less with subarachnoid bolts than with ventriculostomies, although subcutaneous tunneling may diminish the risk of infection in the latter procedure.

Brain Metabolic Monitoring. Two forms of brain metabolic monitoring are available: jugular venous saturation and near-infrared spectroscopy. Blood obtained from the jugular venous bulb provides the cerebral equivalent of "mixed venous" blood. Clinical venipuncture of the jugular venous bulb was first performed more than 60 years ago.⁸¹ Today, retrograde cannulation of the jugular bulb is a safe, technically simple procedure.⁸² CBF, CMRO₂, arterial oxygen content (CaO₂), and jugular venous oxygen content (CjvO₂) are related according to the following equation:

$$(CaO_2 - CjvO_2) \cdot CBF = CMRO_2$$

Rearranged, the equation becomes

$$CjvO_2 = CaO_2 - CMRO_2 \div CBF$$

Therefore, CjvO₂ is a function of CBF, CMRO₂, and arterial oxygenation. SjvO₂, which, together with hemoglobin, determines CjvO₂, is a potential monitor of the cerebral circulation. Mixed cerebral venous blood, like mixed systemic blood, is a global average of effluent from a variety of brain regions and may not reflect even severe regional hypoperfusion. Therefore, low SjvO₂ is abnormal and suggests the possibility of cerebral ischemia; but while normal or elevated SjvO₂ is reassuring, it is not adequate evidence of satisfactory cerebral perfusion. Recent experience with jugular venous bulb monitoring suggests that the technique may be appropriate to guide management.^{31-33,48-50}

Near-infrared spectroscopy, a noninvasive technique that quantifies brain hemoglobin saturation, may eventually offer the opportunity to provide effective monitoring for surveillance or goal-directed therapy. Near-infrared light penetrates the skull and, during transmission through or reflection from brain tissue, undergoes changes in optical density proportional to the relative concentrations of oxygenated and deoxygenated hemoglobin in the tissue beneath the field.⁸³ Although consider-

able animal and clinical data demonstrate the sensitivity of the technique for the detection of qualitative changes in brain oxygenation,⁸⁴ clinical application has been delayed because of uncertainties regarding quantification of the signal. Recent data⁸⁵ suggest that quantification of the signal may be practical and that a noninvasive, continuous monitor of cerebral circulatory adequacy will be possible. The availability of an inexpensive, simple-to-operate surveillance monitor as well as a monitor that could be used for goal-directed therapy provides an opportunity to manage the cerebral circulation as comprehensively as the systemic circulation can now be managed.

General Supportive Care

The essential elements of general supportive care include airway support, administration of maintenance fluids, provision of sedation and analgesia, careful head positioning, and frequent turning. If airway reflexes are impaired, intubation may prevent aspiration and reduce the likelihood of hypoxemia or hypocapnia. Pulse oximetry and capnography facilitate prompt detection of hypoxemia or hypercarbia. Fluid restriction to 50% to 75% of calculated maintenance has long been considered appropriate. Recently, this belief has been challenged,⁶¹ and greater emphasis is now placed on osmolality rather than fluid restriction. Because agitation or pain increases CMRO₂ and CBF, and can precipitate intracranial hypertension, adequate analgesia is particularly important in patients with not only head injuries but also other painful injuries (eg, surgical wounds or fractured long bones or ribs). In noncomatose patients in whom repeated neurological evaluation or direct observation of mental status is essential, sedatives and analgesics must be used with great caution. Neuromuscular blockade necessitates generous, empirical administration of sedatives and narcotic analgesics.

The head should not be rotated excessively. Although compression of the jugular venous system by head rotation produces no change in ICP in normal individuals, it may impede cerebral venous drainage and increase ICP in those with reduced intracranial compliance. Despite the demonstration that head elevation actually reduces CPP in some patients,⁸⁶ most patients appear to tolerate a 15° to 30° elevated-head position well.⁸⁷

An essential aspect of the nursing care of patients with head injuries is frequent, systematic turning:

from the right lateral decubitus position, to the supine, to the left lateral decubitus position. This turning reduces decubitus ulceration and pulmonary retention of secretions. If turning-induced increases in ICP prevent adequate positioning, the use of a laterally rotating kinetic bed may both facilitate pulmonary toilet and limit the incidence of decubitus ulcers. In patients who do not require the kinetic bed, appropriate skin care includes the use of a low-pressure mattress pad.

Management of Intracranial Hypertension

Because of the spatial constraints imposed by the rigid skull, the brain, cerebrospinal fluid, and cerebral blood volume have little room to expand without increasing ICP. Head injury may increase intracranial volume, and therefore ICP, by the mechanisms listed in Table 16-7. Through extensive monitoring of ventricular fluid pressure in patients who had chronic intracranial mass lesions such as gliomas, Nils Lundberg and associates⁸⁸ demonstrated in 1965 that ICP is not static but rather is subject to periodic wave phenomena, the most dangerous of which is the plateau wave. Plateau waves (sometimes called Lundberg waves), which often precipitate acute neurological deterioration, appear to result from cerebral vasodilation⁸⁹ (Figure 16-8) and increases in cerebral

hemispheric blood volume accompanied by a decline in CBF.⁹⁰

Intracranial hypertension can be managed using a variety of strategies, all of which are based on the central concept that reduction of ICP (or improvement of intracranial compliance) can be accomplished by reducing one of the three intracranial constituents: blood volume, tissue volume, or cerebrospinal fluid volume.

Cerebral blood volume can be reduced using several techniques. Endotracheal intubation limits the likelihood of increases in CBF induced by hypoxemia or hypercarbia. Neuromuscular blockade prevents increases in cerebral venous volume as a consequence of coughing, straining, or actively exhaling, but neuromuscular blockade has not been associated with improved outcome in clinical head injury.⁹¹ Head elevation facilitates cerebral venous drainage but, by reducing venous return, cardiac output, and MAP, may actually reduce CPP.⁸⁶ Adequate sedation and analgesia attenuate increases in CMRO₂ and CBF that are produced by painful or noxious stimulation. Control of fever limits increases in CMRO₂ and the accompanying increases in CBF. Passive hyperventilation, probably overused in the past, acutely reduces CBF and cerebral blood volume, although CBF tends to return to original levels despite continued hyperventilation. Selective diminution of blood volume in the pial

TABLE 16-7
CAUSES OF INTRACRANIAL HYPERTENSION AFTER HEAD TRAUMA

| Cause | Mechanism |
|------------------------|---|
| Mass Lesions | Local expansion |
| Intracerebral hematoma | |
| Extraaxial hematoma | |
| Brain Swelling | Vascular congestion, hyperemia |
| Brain Edema | |
| Cytotoxic | Cellular swelling secondary to hypoxia or ischemia |
| Vasogenic | Breakdown of blood-brain barrier, interstitial accumulation of protein |
| Interstitial | Hydrocephalus |
| Secondary Vasodilation | |
| Hypercarbia | Increased extracellular hydrogen ion concentration |
| Hypoxia | Mechanism unclear; possibly increased local metabolite (adenosine?) concentration |
| Hypertension | Impaired autoregulation |

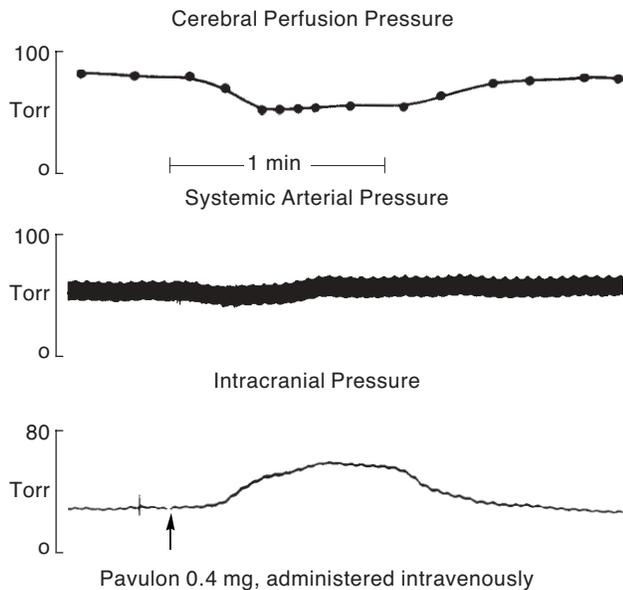


Fig. 16-8. Plateau wave during intracranial pressure recording. Shortly after receiving pancuronium (Pavulon, manufactured by Organon, Inc, West Orange, NJ) 0.4 mg administered intravenously, systemic arterial pressure declined slightly. Almost immediately, intracranial pressure increased dramatically, causing a marked decline in cerebral perfusion pressure. Reprinted with permission from Rosner MJ, Becker DP. Origin and evolution of plateau waves: Experimental observations and a theoretical model. *J Neurosurg.* 1984;60:317.

veins has been demonstrated in experimental animals with α_2 -adrenergic receptor agonists.⁹²

Reduction of brain tissue volume is conventionally achieved by diuresis. Osmotic diuresis, usually accomplished using mannitol, reduces brain water primarily in normal brain. A continuous intravenous infusion of mannitol significantly increases regional CBF without increasing blood pressure or reducing ICP.⁹³ Maximal reduction of ICP appears to be achieved by larger dosages of mannitol administered rapidly, particularly if followed by furosemide.⁹⁴ Fluid restriction, a conventional part of therapy, does not reduce brain tissue volume or ICP in experimental animals.⁶¹ Another method that has been used to reduce brain tissue water and therefore to manage refractory intracranial hypertension is the administration of hypertonic saline. In anecdotal reports,⁹⁵ small volumes of a saline solution approximating 30% (5.0 mmol/mL) have produced a prolonged reduction in ICP and improved prerenal azotemia. In children with head injuries, a 3.0% saline solution reduced ICP significantly.⁹⁶ If intracranial hypertension is refractory to

pharmacological therapy, subtemporal decompression may reduce mortality.⁹⁷ Glucocorticoids, expected to reduce cerebral edema and ICP, have been ineffective in clinical trials.^{98,99}

Regardless of whether ICP can be reduced, an intervention that can reduce cerebral oxygen consumption should be therapeutically desirable. Barbiturates in sufficient dosages suppress both $CMRO_2$ and CBF.⁹³⁻¹⁰⁰ Although barbiturates have been used to control ICP in patients with head injuries, routine administration of barbiturates did not reduce the incidence of intracranial hypertension or improve outcome in a well-designed randomized clinical trial⁸⁰; in fact, the occurrence of barbiturate-induced hypotension prompted the investigators to suggest caution. Barbiturates still may have a role in selected patients who have refractory intracranial hypertension.⁷⁹

Systemic Circulatory Management

Treatment of hypertension requires careful distinction of the life-threatening Cushing response from hypertension associated with the typical hyperdynamic response to head injury. Many commonly used antihypertensive agents, including sodium nitroprusside, nitroglycerin, and hydralazine, may increase ICP. Although those drugs may be used if ICP is monitored, alternative agents (such as the long-acting α -adrenergic and β -adrenergic antagonist, labetalol) may be preferable. Labetalol reduces blood pressure without increasing ICP. Acutely, esmolol may provide rapidly reversible control of blood pressure and heart rate. Adequate sedation and analgesia may also reduce hypertension and tachycardia.

Ventilatory Management

Although many patients with severe head injury will ventilate and oxygenate normally if they are provided with a secure airway, many clinicians prefer mechanical ventilation. Hyperventilation, though useful in reducing life-threatening intracranial hypertension, may lead to inadequate CBF as evidenced by excessive cerebral oxygen extraction⁴⁴ and, when used as routine therapy, actually worsen neurological outcome and mortality in comparison to normocarbia.⁵⁷ Positive end-expiratory pressure (PEEP) may be used to manage hypoxemia in patients with head injuries. PEEP-related increases in ICP are avoided both clinically and experimentally by head elevation.^{101,102}

Many clinicians in North America routinely paralyze patients for at least the first several days of intensive care to prevent decerebrate and decorticate posturing and active expiration, each of which is associated with increases in MAP, central venous pressure, and ICP. The primary disadvantage of neuromuscular blockade is that it interferes with clinical neurological examination. However, recent reports¹⁰³ warn of prolonged neuromuscular dysfunction in patients who have been paralyzed for extended intervals. More importantly, evidence⁹² suggests that neuromuscular blockade may extend the duration of intensive care of patients with head injuries without improving outcome.

Seizure Prophylaxis

Although the overall incidence of posttraumatic seizures is low, most clinicians provide seizure prophylaxis in the acute interval after head injury, particularly for patients with skull fractures or intracranial hematomas.¹⁰⁴ In adults, the prophylactic seizure medication most commonly employed is phenytoin, in a dose of 100 mg administered three times daily (therapeutic serum concentration 10–20 µg/mL). Phenobarbital is commonly added. For the management of acute seizures, diazepam, lorazepam, or phenytoin are usually chosen.

MEDICAL COMPLICATIONS OF HEAD TRAUMA

In addition to their head trauma per se, casualties with brain injuries are also at risk for gastrointestinal and pulmonary complications, infections, and electrolyte and fluid imbalances. Enteral or parenteral nutritional support is usually required.

The incidence of stress-induced gastrointestinal bleeding in patients with head injuries is directly proportional to the severity of the injury.¹⁰⁵ Although aggressive antacid therapy or frequent administration of histamine type 2 blocking agents may be necessary to increase gastric pH, recent data¹⁰⁶ demonstrate that gastric bacterial overgrowth accompanies loss of gastric acidity. Sucralfate, which provides effective prophylaxis without causing gastric alkalinization, may be preferable for ulcer prophylaxis.¹⁰⁷

Patients with severe head injury are at risk for aspiration pneumonitis, noncardiac pulmonary edema, pulmonary embolism, and barotrauma. Prevention of aspiration requires gastric decompression and the use of an appropriately inflated, cuffed endotracheal tube. To limit secretion retention, atelectasis, and secondary infection, mandatory turning and positioning are essential.

An occasional patient with severe head injury develops the adult respiratory distress syndrome secondary to either infection or severe intracranial injury (in which case the syndrome is often called central neurogenic pulmonary edema). The management of adult respiratory distress syndrome consists of ventilatory support with PEEP and relief of intracranial hypertension, if present.

Patients with severe head injury also are at risk for nonpulmonary infections. Urinary tract infection may result from urinary catheterization. Maxillary sinusitis is surprisingly common among

nasotracheally intubated patients with head trauma,¹⁰⁸ although a prospective trial showed that orally intubated patients also have a substantial incidence.¹⁰⁹ Ventriculitis and meningitis complicate trauma, surgical procedures that violate the dura, and indwelling intraventricular monitoring devices.

Abnormalities of sodium metabolism are common in patients with acute head injury. Hypernatremia may result from diabetes insipidus (DI) secondary to trauma or transection of the pituitary stalk. Typically, traumatic DI follows a three-phase course, with polyuria occurring initially, followed by reduced urinary output as stored antidiuretic hormone is released from the damaged pituitary, followed finally by prolonged DI.¹¹⁰ Partial deficits of antidiuretic hormone also occur. The diagnosis of DI necessitates the demonstration that polyuria is a physiologically inappropriate response, which is usually confirmed by the development of hypovolemia or hypernatremia. If hypernatremia develops, the free-water deficit should be corrected slowly. Antidiuretic hormone replacement can be provided in the form of either aqueous vasopressin (5–10 units administered intramuscularly every 6–8 h), lysine vasopressin, or desmopressin (DDAVP, 0.1 mL administered intranasally every 12 to 24 h).

Patients with head injuries frequently develop hyponatremia, occasionally as the result of the syndrome of inappropriate antidiuretic hormone secretion (SIADH)¹¹¹ or excessive intravenous administration of free water, or occasionally, excessive production of atrial natriuretic peptide.¹¹² The treatment of hyponatremia depends on its cause. Hyponatremia associated with hypovolemia should be managed with intravenous fluid administration of

isotonic fluids. Conversely, patients with SIADH usually respond to water restriction. Occasionally, demeclocycline may be necessary to correct SIADH.¹¹³

Despite extensive documentation of hypermetabolism after head injury, the appropriate route and composition of nutritional support have yet to be defined. Many questions remain unanswered. However, most clinicians tend to initiate either enteral or parenteral nutrition as soon as possible after acute head injury, generally preferring enteral nutrition if tolerated. Because patients with a GCS score of 4 to 5 require nearly 70% more than normal

predicted resting metabolic requirements,¹¹⁴ positive nitrogen balance may be difficult to achieve.¹¹⁵ Fever further increases metabolic requirements. Glucocorticoids increase nitrogen wasting.¹¹⁶ When feeding patients with head injuries, the possibility of hyperglycemia should be anticipated and glucose should be controlled. Although only descriptive data¹¹⁷ implicate hyperglycemia in worse neurological outcome after head trauma, considerable experimental and clinical data¹¹⁸ suggest that hyperglycemia worsens injury in some, but not all, models of focal and global neurological ischemia.

RECOVERY AND OUTCOME FOLLOWING TRAUMATIC BRAIN INJURY

Despite good neurological recovery, patients with head injuries often have persistent cognitive or emotional deficits or personality changes, particularly if posttraumatic coma results from diffuse brain injury rather than discrete focal lesions. Recovery of the verbal intelligence quotient usually begins within a few months and becomes stable more rapidly than the performance intelligence quotient, which may continue to improve for a year or more.¹¹⁹ Memory deficits, which may impair social interactions and interfere with work performance, usually persist only briefly after minor injuries. In contrast, patients with severe injuries, particularly those involving both temporal lobes, may have persisting, profound difficulty with both long- and short-term memory.¹²⁰

The most common major neurological findings in patients who survive head injury are unilateral cerebral hemispheric dysfunction and cranial nerve palsies. Factors associated with an increased risk of epilepsy after closed head injury include intracranial hematomas and depressed skull fractures. Of patients who develop seizures within 5 years of injury, nearly 75% will have their first seizure during the first year after injury.¹⁰⁴

Good recovery to moderate disability can be anticipated for approximately 50% of adult patients who enter the hospital with GCS scores of 8 or lower.^{2,72} Studies consistently demonstrate increasing mortality with declining GCS scores.^{2,67,121} Overall, the level of consciousness (as assessed by the GCS) and brainstem reflexes (pupillary light responses and vestibuloocular or oculocephalic reflexes) correlate highly with patient outcome.^{2,67} Bilaterally impaired pupillary light responses or impaired eye movements are associated with a mortality exceeding 75%.^{2,67} In patients with intra-

cranial hematomas, signs of brainstem failure, which would ordinarily predict a dismal outcome, may reverse rapidly after evacuation of a hematoma.^{2,67,122} Factors unrelated to the neurological examination that adversely influence outcome include advanced age, which also correlates with an increasing risk of medical complications; hypoxemia, hypocarbia, hypotension, or anemia on admission to the hospital; and the presence of an intracranial mass lesion on CT scan.^{1,2,67}

Patients who sustain focal lesions tend to have a worse outcome than those presenting with diffuse injury and a similar level of consciousness.¹²³ Patients in whom subdural hematomas are promptly evacuated (ie, within 4 h) have a 30% mortality; those operated on later have a 90% mortality.⁶³ More timely diagnosis of epidural hematoma using CT appears to have reduced mortality.²⁸ More prolonged and severe intracranial hypertension is associated with worse outcome after all head injuries. Patients in whom ICP consistently exceeds 20 mm Hg have a mortality exceeding 50%, whereas patients in whom ICP remains continuously lower than 20 mm Hg have a mortality of only 16%.^{2,72} Mortality approaches 100% if intracranial hypertension cannot be controlled medically.

To increase cost-effectiveness, future research should seek to identify those patients who are so severely impaired that intensive therapy offers no benefit. A recently tested prognostic scale successfully predicted a fatal outcome (without a single falsely pessimistic prediction) in 23 of 52 severely patients with head injuries.¹²⁴ If confirmed in larger studies, such data would permit physicians to make informed, ethical decisions to withhold therapy from certain well-defined categories of patients, thereby more effectively allocating increasingly scarce resources.

SPINAL CORD INJURY

Each year in the United States, 10,000 victims of acute spinal cord injury become paraplegic or quadriplegic. The economic cost of caring for 180,000 acutely and chronically disabled persons is \$4 billion per year.¹²⁵ Most victims of spinal cord injury are young, usually between 15 and 35 years of age,¹²⁶ and male. Patients whose neurological level of injury is above C-7 are unlikely to return to an independent existence (Table 16-8).¹²⁷

Three factors appear to have reduced the acute mortality of patients with spinal cord injury and have improved life expectancy¹²⁷:

1. Patients are more quickly given emergency care.
2. They are then frequently transferred to specialized trauma centers, followed by transfer to specialized rehabilitation facilities.
3. The medical management of cardiovascular, pulmonary, gastrointestinal, and urinary systems has improved, as has skin care and the management of infections.

Spinal anatomy can be divided into three biomechanical categories.¹²⁶ The first portion, the vertebral unit, includes the bones, ligaments, and muscles. The second unit is composed of the spinal cord, nerve roots, and membranous elements, including the dura; and the third, the spinal cord vasculature. Although these arbitrary divisions are not strictly accurate, they are useful for conceptualizing spinal cord injury that results from anatomical damage to one or more elements.

The vertebral unit of the spinal column is composed of 7 cervical, 12 thoracic, 5 lumbar, and 5 fused sacral vertebrae, and 1 coccygeal element formed by the fusion of 4 separate bodies.¹²⁶ With the exception of the first two cervical and the sacral vertebrae, articulation is accomplished by the intervertebral discs, which stabilize the synarthroses between the vertebral bodies; and the posterolateral joints, which are stabilized by their capsules as well as the intrasupraspinous ligaments and the ligamentum flava. The structure promotes stability over a wide range of motion.

The segmental organization of the spinal cord, an extension of the brainstem, is apparent from the

TABLE 16-8

FUNCTIONAL ABILITY BASED ON LEVEL OF NEUROLOGICAL INJURY

| Level of Injury | Functional Ability |
|-----------------|---|
| C-2–C-4 | No movement of upper extremities; some neck control C-2 requires ventilatory support |
| C-5 | Use of biceps allows feeding and grooming with use of special equipment |
| C-6 | Use of wrist allows independent grooming, driving, and simple meal preparation |
| C-7 | Ability to extend arm can permit independent living |
| T-1–T-6 | Normal hand ability; usually capable of independent living in wheelchair-adapted environment |
| T-12 | Complete trunk control with good sitting balance Ambulation with long leg braces possible |
| L-4 | Use of hip flexors and quadriceps Ambulation possible with short leg braces Spinal reflex activity regulating bladder and bowel usually present in T-12 or above but absent in L-1 or below |

Reprinted with permission from Levinson W, Ward G. Care of spinal-cord injured patients after the acute period. *J Gen Int Med.* 1989;4:337.

paired spinal nerves, which are formed from the anterior (motor) roots and the posterior (sensory) roots, then exit through the intervertebral foramina. The spinal cord is suspended within the spinal fluid by nerve roots and ligaments. The pia mater, arachnoid, and dura mater constitute the spinal meninges. The dura, a fibrous membrane extending from the cranium, is firmly attached circumferentially at the foramen magnum, usually closely apposed to the posterior surfaces of the first and second cervical vertebrae, and loosely associated with the posterolongitudinal ligament in the lumbar and cervical regions. The 21 dentate ligaments, which extend from above the first cervical roots to the region of the last thoracic roots, attach to the inner surface of the dura and the lateral surface of the cord, securing and suspending the cord within the dura. Interposed between the spinal dura and the vertebral column is the epidural space, which contains veins, fatty tissue, and ligaments.

The vascular supply of the proximal spinal cord originates from the vertebral and posterior cerebellar arteries, supplemented regionally by branches from the thoracic and abdominal aorta and the deep cervical, intercostal, lumbar, and lateral sacral arteries. These arteries and branches give origin to lateral spinal arteries, which, in turn, give rise to anterior and posterior radicular arteries. The paired anterior radicular arteries enter the spinal cord with each anterior (motor) root and pass caudad to join the anterior spinal artery.

The severity of spinal cord injury is determined by two pathophysiological events: primary mechanical injury and secondary injury. Disruption of the bony and ligamentous elements of the spinal canal through flexion, extension, compression, rotation, shear, or traction injures vascular structures and axons in direct proportion to the magnitude of the disruptive force. Secondary injury worsens the primary mechanical injury as a result of the deleterious effects of hypoxia, hypotension, and progressive vascular and biochemical events that follow injury. Shortly after experimental cord compression, hyperemia and small hemorrhages appear in the central gray matter. These areas subsequently expand, further compromising cord perfusion. Within 8 hours, edema and infarction spread to the white matter. The end result of this sequence is swelling, edema, necrosis, extensive demyelination, and severe neuronal loss in the central gray matter.¹²⁸

Many of the factors implicated in head injury have also been implicated in spinal cord injury (Figure 16-9), primary among which is the failure of

cellular adenosine 5'-triphosphate (ATP) stores, followed by intracellular influx of calcium. With the influx of calcium, phospholipase A₂ is activated, forming arachidonic acid from membrane phospholipids derived from injured cells. Arachidonic acid disrupts cellular integrity; decreases mitochondrial ATP synthesis; and is metabolized to form prostaglandins, thromboxanes, leukotrienes, and free radicals. The free radicals then act directly to destroy normal cellular components, mediate platelet aggregation, initiate vasospasm, inhibit neurotransmitter release, and cause the release of lysosomal enzymes.¹²⁹ The complex pathophysiological effects of secondary injury have suggested a multiplicity of possible therapeutic interventions.

Experimental evidence¹³⁰ suggests that hyperglycemia is associated with worsened neurological outcome after experimental spinal cord ischemia, as it is with cerebral ischemia. In contrast, insulin-induced hypoglycemia improves recovery of electrophysiological function after spinal cord ischemia.¹³¹ Ethanol, commonly involved in spinal cord injury, interferes with spinal cord autoregulation, thereby worsening neurological outcome in ethanol-intoxicated animals.¹³²

After trauma to the vertebral column, neurologically injured patients may be asymptomatic or they may have complete or incomplete spinal cord injury syndromes. The four most common incomplete syndromes are the Brown-Sequard, acute central cord, anterior cord, and posterior cord syndromes (Table 16-9).¹³³

Physiological Responses

Cardiovascular

The immediate cardiovascular response to spinal cord injury is sympathetically mediated tachycardia and hypertension, followed by hypotension secondary to acute sympathectomy if the lesion is above T-5. Acute sympathectomy results in slightly decreased systemic vascular resistance and marked dilation of the venous capacitance vessels. If the cord lesion is cephalad to T-4, the input to cardiac accelerator fibers also is lost, reducing or eliminating compensatory increases in heart rate in response to hypotension. At times, bradycardia produced by unopposed vagal tone may result in cardiac arrest, particularly in association with hypoxemia.¹³⁴

Pulmonary edema occurs in up to 50% of patients with cervical spinal cord injury. In experimental animals, pulmonary edema may result from severe vasoconstriction and acute pulmonary venous hy-

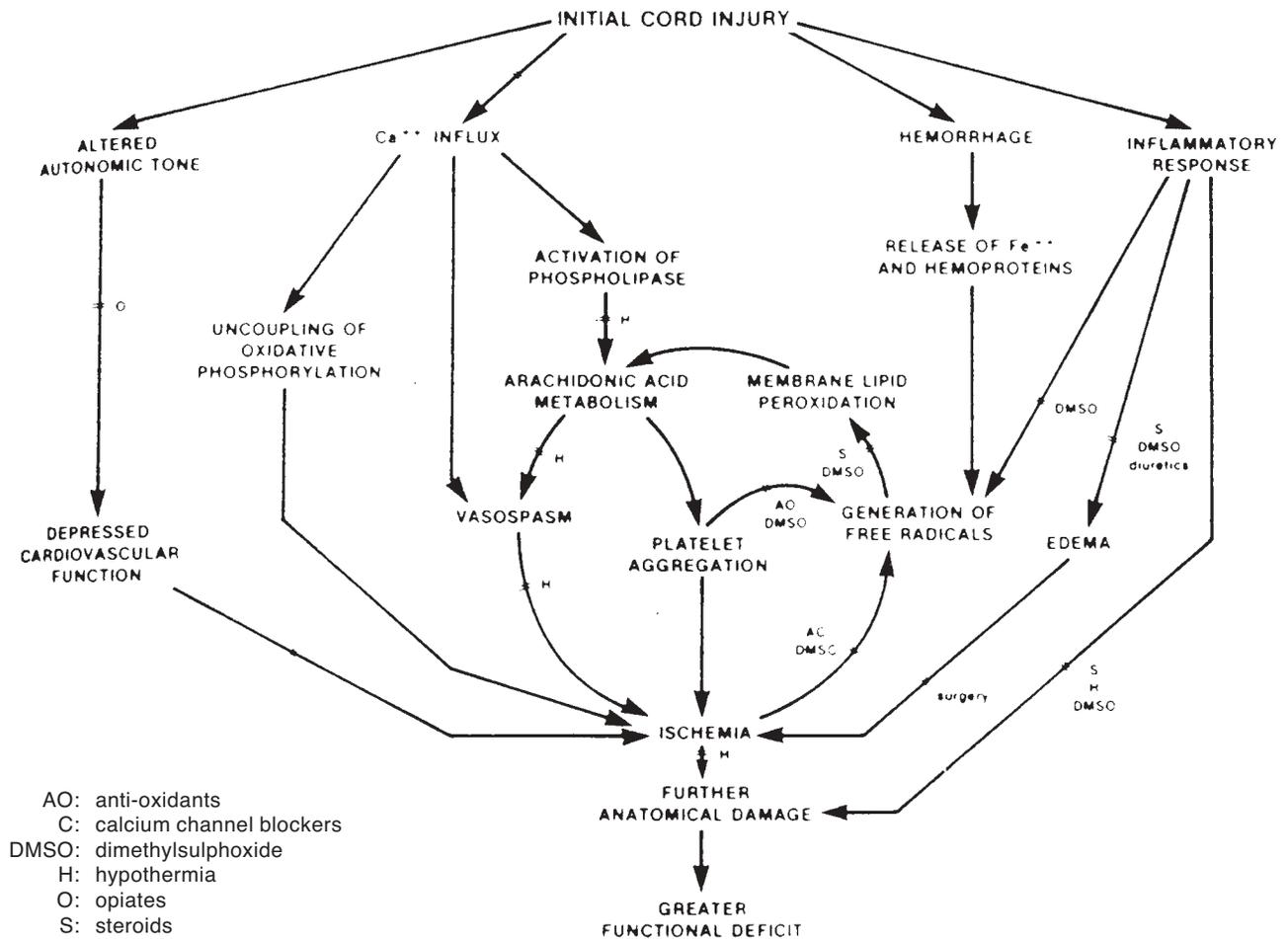


Fig. 16-9. Schematic illustration of proposed mechanisms of secondary spinal cord injury. The processes interact, ultimately producing a greater deficit than could be attributed to the primary injury alone. Potential sites at which these interactive pathways could be interrupted are illustrated. Reprinted with permission from Janssen L, Hansebout RR. Pathogenesis of spinal cord injury and newer treatments: A review. *Spine*. 1991;14:25.

pertension accompanying massive sympathetic discharge after high spinal cord injury.¹³⁵ Researchers¹³⁶ have demonstrated that experimental transection of the spinal cord at the level of C-4 initiates a transient, sympathetically mediated response characterized by a marked increase in MAP followed by hypotension and increases in pulmonary artery occlusion pressure and extravascular lung water. A variety of electrocardiographic changes have been reported, including subendocardial ischemia, shifting pacemaker, atrial fibrillation, ventricular tachycardia, ST-T wave changes, and multifocal premature ventricular contractions.

Although acute spinal cord injury exerts no direct effects on myocardial function, the loss of sympathetic innervation reduces the ability of the myocardium to tolerate sudden increases in preload and

afterload. In patients in whom hemodynamic problems persist, particularly if pulmonary edema is present, pulmonary artery catheterization may clarify the need for additional intravascular volume replacement versus the need for inotropic support.¹³⁷

Autonomic Hyperreflexia. As sympathetic nervous system function begins to recover and spinal shock resolves, usually within 2 to 3 weeks after injury, patients with spinal cord injury cephalad to the level of splanchnic sympathetic innervation (T-4-T-6) may manifest autonomic hyperreflexia (Exhibit 16-2).¹³⁸ The pathogenesis of this disorder is the loss of neurogenic control distal to the level of the lesion, which, in response to noxious stimuli (usually visceral) below the level of the lesion, leads to profound vasoconstriction. Subsequent reflex

TABLE 16-9
NEUROLOGICAL SYNDROMES POSSIBLE WITH CERVICAL SPINE INJURY

| Syndromes | Findings |
|--------------------|--|
| Brown-Sequard | Ipsilateral paralysis Ipsilateral position and vibration sense loss Contralateral temperature and pain sense loss |
| Acute Central Cord | Bilateral motor loss, weakness in upper extremities exceeds weakness in lower extremities Bladder control loss Variable sensory loss |
| Anterior Cord | Bilateral motor, pain, and temperature loss Preserved position and vibration sense Hyperesthesia below the level of the lesion |
| Posterior Cord | All motor and sensory functions preserved except touch and temperature |

Adapted with permission from Hastings RH, Marks JD. Airway management for trauma patients with potential cervical spine injuries. *Anesth Analg*. 1991;73:473.

EXHIBIT 16-2
SIGNS AND SYMPTOMS OF AUTONOMIC HYPERREFLEXIA

- Hypertension
 - Vasoconstriction (below lesion)
 - Vasodilation (above lesion)
- Bradycardia
- Dysrhythmia (ectopic beats occurring during period of heart block)
- Sweating (below lesion)
- Piloerection (below lesion)
- Headache
- Shortness of breath
- Flushed face and neck
- Nausea
- Blurred vision
- Muscle fasciculations
- Convulsions
- Loss of consciousness

Adapted with permission from Schreiberman DL, Mackenzie CF. The trauma victim with an acute spinal cord injury. *Problems in Anesthesia*. 1990;4:472

TABLE 16-10
PULMONARY AND RESPIRATORY MUSCLE FUNCTION IN QUADRIPLÉGIC PATIENTS WITH CERVICAL SPINAL CORD INJURY AT C-4 THROUGH C-7

| Variable | Predicted Normal Value (%) |
|---|----------------------------|
| Vital capacity | 52 ± 11 |
| FEV ₁ /FVC (%) | 85 ± 3 |
| Inspiratory capacity | 71 ± 16 |
| Expiratory reserve volume | 21 ± 12 |
| Total lung capacity | 70 ± 4 |
| Functional residual capacity | 86 ± 14 |
| Residual volume | 141 ± 20 |
| Maximum voluntary ventilation | 49 ± 10 |
| PI _{max} (cm H ₂ O) | 64 ± 12* |
| PE _{max} (cm H ₂ O) | 41 ± 22* |

*Values are expressed as absolute units, not as percentages of predicted normal values

FEV₁: forced expiratory volume in 1 s; FEV: forced vital capacity; PI_{max}: maximum inspiratory pressure; PE_{max}: maximum expiratory pressure

Adapted with permission from Findley LJ, Rochester DF. The lungs and neuromuscular and chest wall diseases. In: Murray JF, Nadel JA, eds. *Textbook of Respiratory Medicine*. Philadelphia, Pa: WB Saunders; 1988: 1949.

activity of the carotid and aortic baroreceptors produces vasodilation above the level of the lesion, ventricular dysrhythmias, bradycardia, and, occasionally, heart block. Of patients with lesions above T-7, 66% to 85% manifest hyperreflexia at some time.^{138,139} Although the potential for autonomic hyperreflexia is maximal at approximately 4 weeks after injury, any patient with spinal cord injury above T-7 is at risk of exposure to a variety of visceral stimuli, most commonly distention of a hollow viscus.¹⁴⁰⁻¹⁴³ Medical management is first directed toward eliminating the causative stimuli, using direct-acting vasodilators such as sodium nitroprusside, controlling cardiac arrhythmias, or, during surgery, deepening anesthesia.

Pulmonary System

A lesion at the level of T-1 eliminates the contributions of the intercostal muscles to both inspiration and expiration and of the abdominal oblique muscles to expiration. Inspiratory reserve volume and total lung capacity are thereby reduced, as are expiratory reserve volume and forced vital capacity (FVC). Table 16-10 illustrates typical pulmonary function tests from patients with acute injury to the mid-to-lower spinal cord.¹⁴⁴ Although functional residual capacity increases in both normal and quadriplegic patients as they are tilted upward, the increase in normal persons is accomplished by an increase in expiratory reserve volume, whereas in quadriplegic patients, who are unable to assist expiration by actively contracting the abdominal musculature, the increase is produced by an increase in residual volume.¹⁴⁵ Within the first few days after acute spinal cord injury, FVC typically decreases somewhat, then begins to recover. Five months later, both peak expiratory flow rates and FVC are substantially greater than during the first week after injury (Figure 16-10).¹⁴⁶

Despite considerable compromise of the respiratory musculature, most patients who are able to generate a normal or nearly normal tidal volume remain normocarbic. However, hypoxemia occurs when FVC is lower than 15 mL/kg because coughing and sighing are inadequate. Acute onset of aspiration, atelectasis, or pneumonia may result in sudden respiratory compromise.

Spinal Cord Blood Flow

Despite extensive experimental work demonstrating the relationship between acute spinal cord injury and reduced spinal cord blood flow (SCBF), the

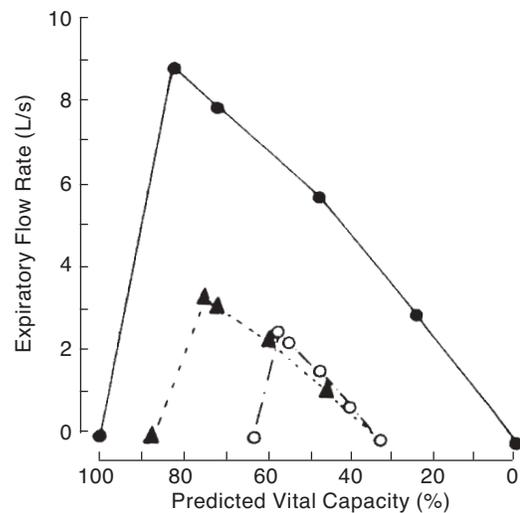


Fig. 16-10. A comparison of predicted expiratory flow rates as a function of predicted vital capacity (closed circles). Open circles represent data from 11 patients in the first week after injury; closed triangles represent data from the same 11 patients 5 months after injury. Reprinted with permission from Ledson JR, Sharp JN. Pulmonary function in acute cervical cord injury. *Am Rev Respir Dis.* 1981;124:43.

relationship between spinal cord ischemia and the progression of neurological deficits is unclear. Because of the unique circulation of the spinal cord, “watershed” areas exist at midpoints of the spinal arterial circulation, equidistant from the radicular arteries. Arterial blood flow in the spinal cord comes from opposite directions, from the cervical cord vessels above and the paired anterior and posterior spinal arteries below.¹⁴⁷ The lower cervical area, most distant from collateral pathways, is most vulnerable to ischemia. Researchers¹⁴⁸ demonstrated that in monkeys, the area most vulnerable to ischemia was at the level of C-6, resulting from the compartmental division of blood flow up and down the anterior spinal artery. Because of limited collateral flow from the vertebral and intercostal arteries, the cervical cord between C-5 and C-8 would be the area most vulnerable to ischemia when MAP falls below the lower limit for autoregulation. Another area of special concern is the variable portion of the thoracic spinal cord supplied by the radicularis magna.

SCBF autoregulation has been described as similar to that of cerebral autoregulation, with the range of 60 to 120 mm Hg representing the autoregulatory plateau (Figure 16-11).¹⁴⁹ Although it is unclear whether autoregulation of SCBF is impaired after cord injury, it is likely that the traumatized spinal

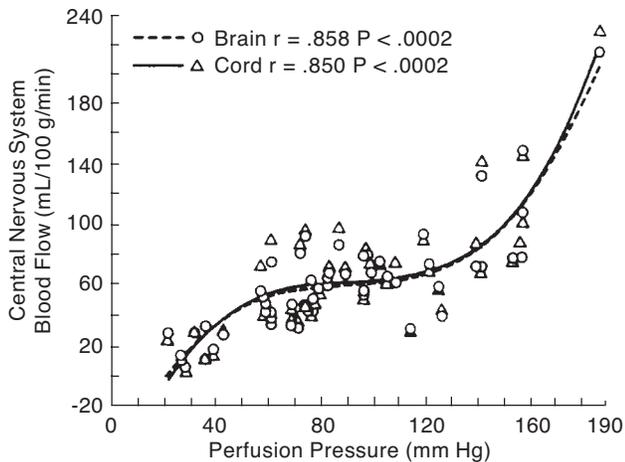


Fig. 16-11. Autoregulation of blood flow is similar in both spinal cord and brain in rats anesthetized with pentobarbital. In both tissues, the autoregulatory plateau extends from 60 to 120 mm Hg perfusion pressure. Reprinted with permission from Hickey R, Albin MS, Bunegin L, Gelineau J. Autoregulation of spinal cord blood flow: Is the cord a microcosm of the brain? *Stroke*. 1986;17:1184.

cord, like the traumatized brain, is extremely vulnerable to hypotension.

Initial Care

Initial care of the patient with potential or evident spinal cord injury consists of three parts: clinical evaluation and stabilization, radiological evaluation, and, when necessary, airway management. Of course, depending on the severity of respiratory compromise, securing the airway may be the first priority.

Clinical Evaluation and Stabilization

Meticulously detailed neurological examinations must be performed as soon as possible. Serial examinations will characterize the initial spinal cord insult, herald extension of injury, and provide prognostic information. Based on knowledge of the neurological level of injury, physiological responses can be anticipated.

It is imperative that patients with spinal cord injury rapidly enter a system in which care is provided by adequately trained, technically proficient personnel who are attuned to the importance of the basic principles of trauma care and the importance of immobilization of the spine, especially if airway intervention is required. Respiratory support and the prompt reversal of shock by the intravenous

administration of fluids are essential to reducing morbidity and mortality. During evaluation and management, appropriate use of cervical collars, backboards or vacuum mattresses, sand bags, and 3-in. tape will immobilize the spine and minimize exacerbation of the injury to the cord until adequate radiological studies have ruled out injury. Such studies may not be possible for days or weeks in patients with multiple trauma.

Once the level of the neurological deficit is established, problems can be anticipated and managed. Among these, the acute level-dependent systemic complications of acute spinal cord injury include, as discussed earlier, loss of sympathetic control of the vascular tree, left ventricular dysfunction, and respiratory compromise. Long-term problems include autonomic hyperreflexia, decreased gastrointestinal motility, nutritional depletion, genitourinary problems, and metabolic dysfunction.

After treating any life-threatening conditions recognized in the primary survey, the medical officer should then proceed to a secondary survey and meticulous head-to-toe physical examination. In comatose patients, concomitant spinal cord injury may be overlooked, especially if a meticulous neurological exam is not conducted. However, neck pain is a sensitive, specific indication of cervical spine injury in awake, cooperative patients (Table 16-11).³³ Because 25% to 60% of patients who have sustained vertebral column injuries have other injuries,¹³⁷ a high index of suspicion must be maintained, even when evaluating an alert, cooperative patient, because the patient may be insensate to an injury below the level of the injury to the spinal cord. Of patients who have spinal cord injuries, 49% also have a concomitant closed head injury.¹⁵⁰ Retrospective studies detect a much lower incidence (6%) of total head injuries associated with spinal cord injuries, and approximately a 2% incidence of severe closed head injury (GCS score \leq 8).¹⁵¹ Injuries of the thoracic spine are associated with multiple rib fractures, flail chest, or aortic disruption.

In summary, during initial evaluation, a rapid assessment of spinal cord, head, and other injuries should be made; the airway and adequate ventilation assured; supplemental oxygen applied; intravenous access secured; fluid resuscitation initiated; and expeditious transport arranged to a definitive care facility, ideally a trauma or spinal cord injury center.

Radiological Evaluation

In the recent past, a single, cross-table lateral, plain cervical spine X-ray examination, which in-

TABLE 16-11
CERVICAL SPINE INJURY AND NECK PAIN IN ALERT TRAUMA PATIENTS

| Primary Study | Number of Patients | | Study | Number of Patients | |
|------------------------------|---------------------|------------------|----------------|------------------------|-------------------|
| | With C-Spine Injury | With Neck Pain | | Primary C-Spine Injury | Without Neck Pain |
| Fisher (1984)* | 5 | 5 | Fisher | 0 | 328 |
| Roberge (1988) | 6 | 6 | Roberge | 0 | 141 |
| Bayless (1989) | 2 | 2 | Bayless | 0 | 122 |
| Bachulis (1987) | 65 | 65 | Neifeld (1988) | 0 | 145 |
| Ringenberg (1988) | 253 | 247 [†] | Kreipke (1989) | 0 | 324 |
| Lally (1989) [‡] | 16 | 16 | | | |
| Rachesky (1987) [‡] | 21 | 21 | | | |

*Numbers in parentheses are the years in which the primary source was published

[†]Six patients with other painful injuries did not report neck pain

[‡]Pediatric trauma

Adapted with permission from Hastings RH, Marks JD. Airway management for trauma patients with potential cervical spine injuries. *Anesth Analg*. 1991;73:473.

cluded all seven cervical vertebrae and the C-7-T-1 interspace, was considered sufficient to assess cervical spine integrity. However, the sensitivity of this single view is only 74% to 92% (Table 16-12). A three-view evaluation, including anteroposterior and odontoid views, permits better assessment of vertical alignment of the spinous and articu-

lar process and of abnormalities in the joint and disk spaces.¹³³ If a patient is able to follow commands, lateral flexion-extension views should be obtained, as the patient controls head motion. Until then, rigid immobilization with a hard cervical collar (eg, the Philadelphia collar, manufactured by Philadelphia Collar, Westville, N.J.) is

TABLE 16-12
SENSIVITY OF CERVICAL SPINE X-RAY STUDIES

| Primary Study | View | Number of Patients With Cervical Spine Injury | Sensitivity* (%) |
|----------------------------------|---------|---|------------------|
| Streitwieser (1983) [†] | CTL | 44 | 82 |
| | 3 Views | 38 | 92 |
| Shaffer (1981) | CTL | 35 | 74 |
| Bachulis (1987) | CTL | 90 | 77 |
| Ross (1987) | CTL | 13 | 85 |
| | 3 Views | 13 | 92 |
| MacDonald (1990) | CTL | 92 | 92 |
| Freemyer (1989) | 3 Views | 33 | 91 |

*Sensitivity = true positives ÷ (true positives + false negatives) • 100

[†]Numbers in parentheses are the years in which the primary sources were published

CTL: cross-table lateral view; 3 views: cross-table lateral, anteroposterior, and open-mouth

Adapted with permission from Hastings RH, Marks JD. Airway management for trauma patients with potential cervical spine injuries. *Anesth Analg*. 1991;73:476.

indicated, if there is any clinical suspicion of neck injury.

Computed or conventional tomography supplements plain radiographs that are equivocal or negative despite clinical evidence of cord injury.¹⁵²⁻¹⁵⁴ The occurrence of spinal cord injury without radiographic evidence of vertebral injury or instability can be as high as 15% to 70%.^{155,156} Although MRI is superior for evaluation of injury to soft tissues, including paravertebral tissue, intervertebral discs and ligaments, the complexity involved in obtaining MRI scans precludes its use in most acute circumstances.

Airway Management

Despite the simultaneous risks of inadequate pulmonary function and aggravation of spinal cord damage, there are no universally accepted approaches to airway management in patients with acute spinal cord injury. The method chosen in a specific situation is determined by a variety of factors, among them the severity of physiological compromise, the clinician's experience and ability, the availability of specialized equipment, the magnitude and type of associated injuries, the presence of hemodynamic stability, and the availability of definitive diagnostic information about the cervical spine.^{133,157} Because the need to secure the airway emergently cannot be predicted in advance, a cart containing necessary equipment should be readily available (Exhibit 16-3).

The choice among various methods of airway management depends on the severity of physiological compromise. If a patient is unable to maintain normoxia and normocarbida, or is actively vomiting and unable to protect the airway, then direct visualization and orotracheal intubation should be attempted first. Adjunctive medications, such as sedatives, induction agents, or muscle relaxants, should be used as dictated by the patient's hemodynamic status. Cricoid pressure may be used if necessary, but its vigorous application could produce subluxation at the injury site. Immobilization of the cervical spine can be accomplished with manual in-line axial traction (MIAT),¹⁵⁷ a method associated with no evidence of new or worsened neurological injury resulting from intubation in more than 3,000 patients.¹⁵⁸ Gardner-Wells tongs can also be used for cervical spine immobilization. If the oral route is impractical because of injury, edema, or oropharyngeal bleeding, and an artificial airway is required, emergent surgical cricothyrotomy can be performed; alternatively, percutaneous needle cricothyrotomy can be used as a temporizing measure.

If the need for an artificial airway is less acute, nasotracheal intubation may be performed either blindly or using fiberoptic guidance. The nasal route should not be used in the presence of basilar skull fracture, midface instability, or nasoseptal injury because of the risk that the endotracheal tube will enter the brain⁵⁵ or cause serious hemorrhage. Vasoconstriction of the nasal mucosa can be achieved with the application of nasal decongestant spray (eg, Afrin, manufactured by Shearing-Plough Health Care Products, Inc., Memphis, Tenn.) or phenylephrine. Topical anesthesia may alleviate discomfort even in a semicomatose patient who might otherwise react vigorously to noxious airway stimulation, thereby precipitating extension of the spinal cord injury. Fiberoptic bronchoscopy can be used to facilitate nasotracheal intubation, assuming that the intubator is skilled and that there is little blood or foreign material in the oropharynx.

The nasal route does have some important disadvantages, however. Any mechanical airway manipulation may provoke hypertension, a particular hazard in a patient with closed head injury and decreased intracranial compliance. Epistaxis may be severe, especially in patients who have coagulopathies. When performed blindly, nasotracheal intubation may damage the larynx. Either blind or fiberoptically guided nasotracheal intubation may be time-consuming. Semicomatose,

EXHIBIT 16-3

NECESSARY EQUIPMENT FOR EMERGENCY AIRWAY MANAGEMENT

- Laryngoscope with blades of various sizes and shapes
- Endotracheal tubes of various sizes
- Endotracheal tube stylet
- Oral and nasal airways
- Face masks of various sizes
- Tonsil-tipped suction handle or suction source
- Ambu bag (attached to oxygen source)
- Pharmacological adjuncts (drugs drawn up in syringes)

Reprinted with permission from Grande CM. Airway management of the trauma patient in the resuscitation area of the trauma center. *Trauma Q.* 1988;5(1):38.

inebriated, or frightened patients may react combatively, risking further injury to the spinal cord. Prolonged nasotracheal intubation is associated with maxillary sinusitis and occasional sepsis.

Other methods of placing oral tracheal tubes such as retrograde wires may be challenging because of associated injuries. Thus, even in circumstances that are not emergent, the method of choice for tracheal intubation may be orotracheal intubation in conjunction with MIAT, after denitrogenation, supplemented by a carefully titrated dose of thiopental, midazolam, or etomidate, and a neuromuscular blocking agent. Succinylcholine is appropriate if the injury is less than 48 hours old; after 48 hours, the risk of succinylcholine-induced hyperkalemia, potentially a fatal event,¹⁵⁹ increases dramatically. A nondepolarizing agent such as vecuronium is preferable if more than 48 hours have elapsed.

Anesthetic Management

Anesthesia personnel may be confronted with patients with spinal cord injury at any stage of resuscitation, just as they may with patients with head injuries. This section addresses the anesthetic management of the patient with spinal cord injuries who does not have a secure airway.

Preoperatively, the most important considerations relate to respiratory compromise, hypotension, and the possibility of associated injuries. Respiratory compromise may not be evident on initial examination but may be apparent if the patient is asked to perform a vital-capacity maneuver. Hypotension is secondary to venodilation and impairment of the usual reflex increase in heart rate that occurs with hypotension in patients in whom sympathetic pathways are intact. Associated injuries below the level of the injury may not be recognized because of the absence of pain. Before induction of anesthesia, blood pressure should be restored with the use of volume expansion. Pressors may occasionally be necessary. Dopamine tends to constrict the venous system and increase heart rate. Ephedrine may be useful for short-term stabilization. Atropine is especially useful if bradycardia is a major factor in hypotension or if severe bradycardia follows airway stimulation.

Monitoring during anesthesia should be determined by the severity of associated injuries and the availability of equipment. If time and circumstances permit, direct arterial pressure monitoring will warn promptly of hypotension. Central venous pressure monitoring may provide information about the re-

lationship between blood volume and expanded venous capacitance. Pulse oximetry and capnography provide early warning of deterioration in gas exchange. Automated, noninvasive blood pressure monitoring may free anesthesia personnel for other activities.

Anesthetic induction must preserve hemodynamic stability while securing the airway with minimal risk of aggravating the cervical spine injury. The initial decision is whether to intubate the patient awake (fiberoptically or blindly) or whether to proceed with a rapid-sequence induction. Either should be accompanied by MIAT. Awake intubation, in the absence of MIAT, risks cervical movement when the endotracheal tube enters the trachea and stimulates coughing. On the other hand, vigorous head movements during direct laryngoscopy also risk excessive cervical motion.

Potential anesthesia induction agents include thiopental, etomidate, benzodiazepines, and ketamine. The effects of anesthetics on perfusion of the injured spinal cord are not well known. Most clinicians assume that the effects are similar to the effects of agents on CBF and $CMRO_2$. Large dosages of barbiturates may reduce blood pressure, especially in hypovolemic patients. Etomidate provides prompt induction and is usually well tolerated even in some hypotensive patients. Benzodiazepines usually preserve blood pressure. Large dosages may slow emergence. Cricoid pressure should be applied during induction and intubation. In a hemodynamically resuscitated patient with acute spinal cord injuries, a reasonable sequence is to administer thiopental and fentanyl followed by succinylcholine, all while maintaining cricoid pressure. Hyperkalemia resulting from succinylcholine administration is unlikely after recent (< 24-h old) spinal cord injury. The incidence increases after 24 hours and plateaus by 12 days. For maintenance, the effects of volatile anesthetics on spinal perfusion also appear to parallel their effects on CBF and $CMRO_2$.

Postoperatively, the ability to extubate the patient with a spinal cord injury depends on preoperative respiratory compromise and on the magnitude and site of surgery. Before extubating the patient with a cervical cord injury, it is wise to determine FVC and peak negative pressure.

Management in the Intensive Care Unit

Management in the intensive care unit is intended to limit neurological disability and reduce the morbidity and mortality of the complica-

TABLE 16-13
CAUSE OF DEATH IN THE FIRST MONTH AFTER SPINAL CORD INJURY

| Cause of Death | Actual Deaths | SMR | 95% Confidence Limit of SMR |
|-------------------------------------|---------------|-------|-----------------------------|
| Septicemia | 5 | 500.0 | 61.7–938.3 |
| Cancer | 0 | 0.0 | 0.0–0.0 |
| Ischemic heart disease | 4 | 7.7 | 0.2–15.2 |
| Other heart disease | 8 | 133.3 | 40.9–225.7 |
| Cerebrovascular disease | 4 | 33.3 | 0.6–66.0 |
| Diseases of arteries | 2 | 50.0 | 0.0–119.3 |
| Venous thrombosis and embolism | 10 | 500.0 | 190.1–809.9 |
| Influenza and pneumonia | 12 | 300.0 | 130.3–469.7 |
| Other respiratory disease | 4 | 66.7 | 1.4–132.0 |
| Diseases of digestive system | 4 | 66.7 | 1.4–132.0 |
| Diseases of urinary system | 4 | 200.0 | 4.0–396.0 |
| Symptoms and ill-defined conditions | 11 | 275.0 | 112.5–437.5 |
| Unintentional injuries and suicides | 0 | 0.0 | 0.0–0.0 |
| Residual | 7 | 24.1 | 6.2–42.0 |
| Unknown | 1 | | |

SMR: standardized mortality ratio (actual deaths ÷ predicted deaths • 100)

Adapted with permission from DeVivo MJ, Kartus PL, Stover SL, Rutt RD, Fine PR. Cause of death for patients with spinal cord injuries. *Arch Intern Med.* 1989;149:1765.

tions that accompany acute spinal cord injury. It is necessary to prevent further damage to the patient's spinal cord, manage cardiovascular and pulmonary complications, and control pain. In the first month after spinal cord injury, which includes the time during which patients are most likely to be admitted to the intensive care unit, pneumonia, pulmonary embolism, and sepsis are the most common factors contributing to death (Table 16-13).¹⁶⁰

In the intensive care unit, monitoring of patients with acute spinal cord injury consists of careful cardiovascular monitoring, monitoring of gas exchange, and the use of EPs for diagnosis and prognosis. Pulmonary monitoring is the same as that used for other critically ill patients, consisting of pulse oximetry, capnography, and, as necessary, arterial catheterization to obtain arterial blood gases. Pulse oximetry may be of particular value in those patients who become bradycardic in response to hypoxemia.

Somatosensory evoked potentials (SSEPs), noninvasive tests of the integrity of spinal cord transmission, have developed into important diagnostic and prognostic tools in patients with acute

spinal cord injury.^{161,162} Strictly speaking, SSEPs cannot be used to monitor acute injuries. Not surprisingly, preservation of SSEPs after injury is consistent with incomplete spinal cord transection and portends a more favorable outcome than loss of SSEPs.¹⁶³ When combined with clinical evaluation of motor function, sensation to pinprick, and joint position, SSEPs provide greater prognostic value.⁶⁹ Although techniques for the use of MEPs have been developed for humans, few data quantify the likely diagnostic and prognostic value of this modality.¹⁶⁴

Spinal Cord Protection

Because of the progressive hemorrhagic infarction of the spinal cord that sometimes follows acute trauma, the pathophysiology of secondary, progressive injury has been extensively investigated (see Figure 16-9). Of the great variety of interventions that have been attempted after acute spinal cord injury, several have received particularly intense scrutiny, including localized spinal cord hypothermia, endogenous opioid antagonists, glucocorticoids, free radical scavengers, and G_{M1} ganglioside.

Topical Hypothermia. Topical hypothermia was first investigated more than 25 years ago,¹⁶⁵ based on the assumption that hypothermia would reduce spinal cord metabolism, thereby decreasing the risk of infarction, and presumably decreasing the rate of formation of toxic metabolites.¹⁶⁶ Despite experimental evidence in favor of topical hypothermia, the results in humans have been inconsistent and discouraging,¹²⁶ perhaps because of the heterogeneity of human spinal cord injury and the substantial delay before most patients reach treatment facilities.

Opiate Antagonists. Researchers^{167,168} first reported in the 1980s that endorphins (endogenous opioids) are involved in the progression of spinal cord injury and that the administration of naloxone, an opiate antagonist, increased blood pressure and SCBF and improved neurological recovery in comparison to results in animals given a saline control. Thyrotropin-releasing hormone, an opiate antago-

nist that does not reverse opioid-mediated analgesia, also exerts a beneficial effect in experimental spinal cord injury.^{168,169} However, other researchers¹⁷⁰ conclusively demonstrated that, in humans, naloxone (given as a bolus of 5.4 mg/kg, followed by an infusion at a rate of 4.0 mg/kg/h for 23 h) failed to improve neurological outcome in comparison with placebo and methylprednisolone (discussed below) (Table 16-14).

Glucocorticoids. In animals subjected to spinal cord trauma, glucocorticoids, including dexamethasone and methylprednisolone, have generally exerted therapeutic benefit. As a consequence of promising data from studies with animals, a multicenter, double-blind, randomized clinical trial¹⁷¹ compared patients who received "standard"-dose glucocorticoids (100 mg as a bolus followed by daily administration of 100 mg for 10 d) and patients who received "high"-dose glucocorticoids (a 1,000-mg bolus followed by 1,000 mg daily for 10 d). There were

TABLE 16-14

NEUROLOGICAL OUTCOME AT 6 WEEKS AND 6 MONTHS IN PATIENTS TREATED WITHIN 8 HOURS OF INJURY

| Category of Injury and Measure* | 6 Weeks | | | 6 Months | | |
|------------------------------------|-------------------|-------------------|----------------------|-------------------|----------|----------------------|
| | MP | Naloxone | Placebo [†] | MP | Naloxone | Placebo [†] |
| Plegic with total sensory loss | | | | | | |
| <i>Number of patients</i> | 47 | 37 | 46 | 45 | 34 | 44 |
| Motor | 6.2 [‡] | 3.2 | 1.3 | 10.5 [‡] | 7.5 | 4.2 |
| Pinprick | 5.9 | 3.0 | 2.2 | 9.4 [‡] | 4.2 | 4.0 |
| Touch | 6.8 [‡] | 3.7 | 2.6 | 9.7 [‡] | 7.1 | 4.7 |
| Plegic with partial sensory loss | | | | | | |
| <i>Number of patients</i> | 5 | 12 | 6 | 5 | 11 | 6 |
| Motor | 14.4 | 14.1 | 18.0 | 23.0 | 28.9 | 26.5 |
| Pinprick | 11.8 | 13.9 [‡] | 4.0 | 11.6 | 18.4 | 9.8 |
| Touch | 4.4 | 7.1 | 0.3 | 0.0 | 13.5 | 5.2 |
| Paretic with variable sensory loss | | | | | | |
| <i>Number of patients</i> | 14 | 12 | 17 | 12 | 11 | 17 |
| Motor | 18.3 [‡] | 12.7 | 10.8 | 24.3 [‡] | 14.5 | 12.9 |
| Pinprick | 10.7 | 8.2 | 7.5 | 14.3 | 9.6 | 7.5 |
| Touch | 3.8 | 6.1 | 1.2 | 7.6 | 6.2 | 1.0 |

*Scores for motor function range from 0 to 70; scores for sensations of pinprick and touch each range from 29 to 87

[†]Placebo value is used as the control (reference value) to which MP and naloxone were compared, using an analysis of variance

[‡] $P < .05$

MP: methylprednisolone

Adapted with permission from Bracken MB, Shephard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. *N Engl J Med.* 1990;322:1409.

no differences in neurological recovery between the two groups, but wound infections were more prevalent in the patients receiving the higher dose.

A second, randomized, controlled clinical trial¹⁷⁰ compared placebo, naloxone, and methylprednisolone given as a bolus of 30 mg/kg, followed by a 23-hour infusion at a rate of 5.4 mg/kg/h. The protocol differed from the earlier trial¹⁷¹ in two key respects. First, the initial daily dose was much greater. Second, the drug was given for only 1 day, based on the rationale that short-term administration would be associated with fewer wound complications and infections, but would still provide maximal glucocorticoid effect during the most critical interval. The subjects who received methylprednisolone within 8 hours of injury showed a significantly greater improvement in motor function and sensation to pinprick and touch than those who received the placebo, naloxone, or methylprednisolone more than 8 hours after injury (see Table 16-14).¹⁷⁰ Although the differences in the second clinical methylprednisolone trial were statistically significant, they were not dramatic; however, even small differences in motor function may result in major differences in the rehabilitation and quality of life of patients with spinal cord injury. In this study, the short-term administration of glucocorticoid was not associated with increased risk of infection or gastrointestinal bleeding.

Free Radical Scavengers. Considerable experimental evidence suggests that postischemic oxidative injury may contribute to neurological damage. A variety of methods have been used in an attempt to reduce oxidative stress, particularly to reduce the accumulation of oxygen free radicals in injured tissue. One of the more effective experimental interventions has been the use of 21-aminosteroids, compounds that are derived chemically from glucocorticoids, but lack glucocorticoid activity and are potent inhibitors of iron-dependent lipid peroxidation.¹⁷²

G_{M1} Ganglioside. Most of the pharmacological interventions that have been directed at acute spinal cord injury have been designed to reduce the rapid hemorrhage and infarction of the spinal gray matter that occur in the first few hours after injury. However, an alternative strategy is to attempt to preserve the axons passing through white matter adjacent to the site of injury.¹⁷³ Experimental data suggest that preservation of as few as 6% of axons prevents distal muscle paralysis and preserves normal movement.¹⁷⁴ Enhanced recovery or repair of damage to long tracts in the spinal cord potentially can be initiated 72 hours or more after the primary injury.¹⁷⁵ In a prospective, randomized, placebo-

controlled, double-blind trial, G_{M1} ganglioside (monosialotetrahexosylganglioside), a compound that enhances neurological recovery after experimental spinal cord injury,¹⁷⁶ substantially enhanced improvement, assessed using the Frankel scale and the American Spinal Injury Association (ASIA) motor scores, after 1 year of follow-up.¹⁷³ The improvement appeared to be attributable to restoration of useful motor function in initially paralyzed muscles rather than to improving strength in paretic muscles. The overall improvement in ASIA motor score (Figure 16-12) reflects a trend toward greater improvement in lower extremity function than in upper extremity function (Figure 16-13). The magnitude of the beneficial effect of G_{M1} ganglioside in this study was approximately twice as great as that reported earlier with high-dose methylprednisolone. Because the patients in the G_{M1} ganglioside trial received relatively small, probably ineffective, dosages of methylprednisolone, these data raise the exciting possibility that the combination of acutely administered high-dose glucocorticoids and the subsequent administration of G_{M1} ganglioside could produce greater improvement than either agent alone.

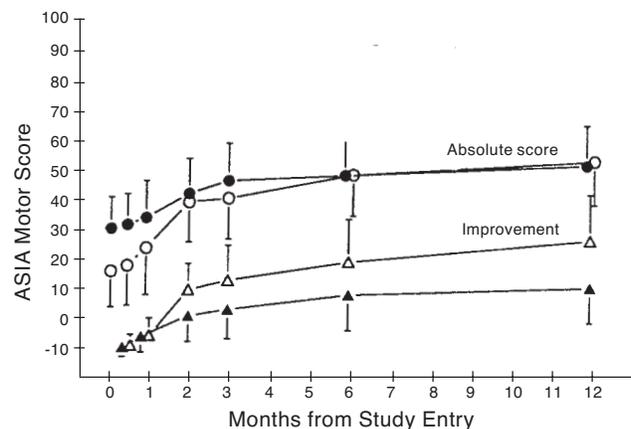


Fig. 16-12. Changes in the American Spinal Injury Association (ASIA) motor score in patients treated with G_{M1} ganglioside (open symbols) and a placebo (solid symbols) in the 12 months after their entry into the study. The upper curves (circles) show unadjusted improvement and demonstrate that the two groups were significantly different at admission into the study (ie, the patients receiving G_{M1} ganglioside were more severely injured). After adjustment for the imbalance in scores at entry, the improvement is apparent. The error bars represent 95% confidence intervals. Reprinted with permission from Geisler FH, Dorsey FC, Coleman WP. Recovery of motor function after spinal-cord injury—a randomized, placebo-controlled trial with GM-1 ganglioside. *N Engl J Med.* 1991;324:1835.

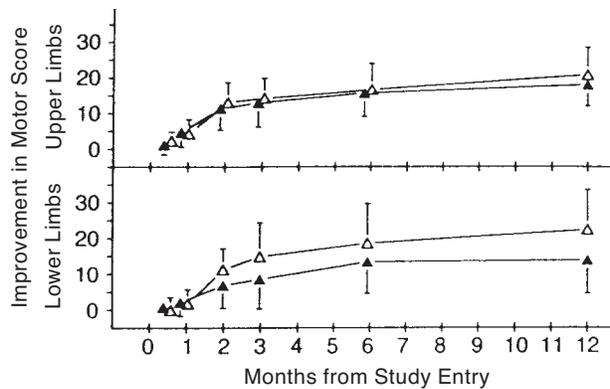


Fig. 16-13. Comparison in the improvement of motor score, assessed using the American Spinal Injuries Association (ASIA) score, in the upper and lower limbs in patients with cervical injuries who were randomized to receive G_{M1} ganglioside (open triangles) and a placebo (solid triangles). A trend toward a drug effect in the score for the lower extremities was not observed in the upper extremities. The error bars represent 95% confidence intervals. Reprinted with permission from Geisler FH, Dorsey FC, Coleman WP. Recovery of motor function after spinal-cord injury—a randomized, placebo-controlled trial with G_{M1} ganglioside. *N Engl J Med.* 1991;324:1836.

Cardiovascular Management

Cardiovascular management in the intensive care unit is directed at maintaining intravascular volume, providing inotropic support as needed, avoiding excessive fluid administration, and carefully avoiding or promptly treating bradycardia—especially that associated with suctioning in hypoxic patients. The syndrome of spinal shock, which may continue to be a problem for as long as 6 weeks after injury, requires adequate intravascular volume expansion and, in some cases, vasoactive drugs such as dopamine (which, even in low dosages, acts as a vasoconstrictor). Particular care should be taken with the positioning of patients in the spinal shock phase of acute spinal cord injury, because sudden changes in position, particularly to the upright position, may result in sudden hemodynamic instability.^{177,178} Hemodynamic deterioration resulting from bradycardia may necessitate intermittent or prophylactic treatment with atropine, especially during vagotonic maneuvers such as tracheal suctioning, endotracheal intubation, or rectal examination.

In the acute phase of management, an arterial catheter may be useful for blood pressure monitoring and management of blood gases. Pulmonary artery catheterization may be useful for short-term

assessment of intervascular volume and cardiac output during the acute phase of spinal shock. However, because of the significant contribution of infection to morbidity and mortality in patients with spinal cord injury, invasive monitoring should be used judiciously. Fluid challenges have been recommended to assess cardiac function in conjunction with pulmonary artery catheterization.¹³⁷ Using this approach, fluid is administered in 250-mL increments at 50 mL/min until cardiac filling pressures rise and remain at least 2.0 mm Hg greater than the preinfusion level. Fluid challenges help to define the need for inotropic support as well as for additional volume administration in hypotensive patients.

Pulmonary Management

In patients with spinal cord injury, the major cause of early death—respiratory failure secondary to muscle paralysis—depends on the level of injury, and may either progress or regress over time. Frequently, diaphragmatic function gradually and progressively deteriorates for up to 5 days after injury, often accelerated by other pulmonary complications such as pulmonary edema or aspiration of vomitus. Serial measurements of FVC and peak negative inspiratory pressure permit early recognition of deterioration and prompt intubation and mechanical ventilation before hypoxemia and hypercarbia occur. In general, patients with spinal cord injury require ventilatory assistance when FVC is lower than 15 mL/kg or when a peak negative inspiratory pressure of -25 cm H_2O cannot be attained. Likewise, measurements of FVC and peak negative inspiratory pressure used to guide weaning from mechanical ventilation (Table 16-15).¹³⁸

Nosocomial pulmonary infections occur not only as a result of respiratory compromise but also because of the relative immunocompromise that accompanies severe trauma. Close attention to pulmonary toilet, including chest physiotherapy, positioning, and tracheal suctioning, is essential,¹⁷⁹ especially in those patients in whom postinjury FVC barely exceeds the threshold for intubation (15 mL/kg). The use of rotating beds, especially for patients in cervical traction, may promote better pulmonary toilet, although controlled studies¹⁸⁰ of this expensive intervention in patients with head injuries have been less encouraging than earlier, uncontrolled studies. Care must be taken during rotational therapy to maintain hemodynamic stability in patients who have high cervical lesions or spinal shock.

TABLE 16-15
WEANING CRITERIA FOR REMOVAL FROM MECHANICAL VENTILATION

| Weaning Criterion | Acceptable Value |
|------------------------------------|-----------------------------|
| Maximum inspiratory force | > -20 cm H ₂ O |
| Maximum expiratory force | > +20 cm H ₂ O |
| Vital capacity | > 1,000 mL |
| Expiratory value | > 10 L/s (level dependent) |
| PaO ₂ /FIO ₂ | > 250 |
| Lung/thoracic cage compliance | > 30 mL/cm H ₂ O |

Adapted with permission from Schreiberman DL, Mackenzie CF. The trauma victim with an acute spinal cord injury. *Problems in Anesthesia*. 1990;4:471.

For patients who require intubation for pulmonary support, the choice of neuromuscular blocking agents is important. After the first 24 to 48 hours after injury, succinylcholine-induced hyperkalemia can be life threatening.^{159,181} Although the exact onset of this response in humans has not been elucidated, studies¹⁸² in a primate model suggest that the critical time may be as soon as 4 days after injury, with half of the peak increases (approximately 2.8 mEq/L above baseline) in serum potassium occurring at 8 days, and peak increases (approximately 5.5 mEq/L above baseline) occurring at 14 days after injury. The interval during which patients with spinal cord injury remain vulnerable to the hyperkalemic response to succinylcholine is unclear but certainly extends more than 6 months after injury.

Prevention of venous thrombosis and pulmonary embolism, major causes of morbidity and mortality after spinal cord injury,¹⁷⁷ is an important part of the management of patients with acute injury to the spinal cord. Low-molecular-weight heparin is safer and more effective than conventional heparin in the prevention of thromboembolism in patients who have complete motor paralysis.¹⁸³

Pain Syndromes

Three types of pain syndromes commonly develop in the postacute phase of spinal cord injury^{127,184}:

1. Pain at the site of trauma is usually well localized, tender and responds well to analgesics, heat, and transcutaneous electrical nerve stimulation (TENS).
2. Radicular or dermatomal pain presumably results from irritation of nerve roots by adhesive arachnoiditis. Variable symptoms include sharp, burning pain with hypersensitivity in a radicular distribution. TENS may be effective. Carbamazepine is also often effective. Other analgesics are sometimes helpful, but often not.
3. Posttraumatic spinal deafferentation pain is a diffuse burning or dysesthesia below the level of injury. Often difficult to control, deafferentation pain is resistant to analgesics but may respond to phenytoin, carbamazepine, or amitriptyline. Approaches such as dorsal root entry zone (DREZ) surgery are reserved for refractory cases.

The specific management of pain associated with spinal injury beyond the acute stages is beyond the scope of this chapter, which deals with trauma anesthesia for combat casualties, not their rehabilitation. However, *Rehabilitation of the Injured Soldier*,¹⁸⁵ a volume in the *Textbook of Military Medicine* series, discusses the problem in detail.

Medical Complications

The sequelae of spinal cord injury include numerous and varied medical complications, such as those of the gastrointestinal and genitourinary systems, electrolyte abnormalities, decubitus ulcers, and the need for nutritional and emotional support.

Gastrointestinal System

Gastric atony usually accompanies spinal shock; intestinal motility may be reduced for several weeks after injury. Because air swallowing leading to gastric distention is frequently a problem in the early course of hospitalization after spinal cord injury, placement of a nasogastric tube is mandatory. Replacement of nasogastric drainage is important to prevent hypokalemic, hypochloremic metabolic alkalosis. Prophylaxis against stress gastritis and ulceration can be accomplished using antacids, H₂ blocking agents, or sucralfate. In 1989,

researchers¹⁸⁶ reported that early provision of total energy requirements through adequate oral or parenteral nutrition decreased the incidence of gastrointestinal bleeding in patients with spinal cord injury.

In cervical and high-thoracic spinal cord injury, the sympathetic control of the transverse colon and rectum is disrupted, as is parasympathetic control of the descending and rectosigmoid colon. Loss of voluntary control of the external anal sphincter, pelvic floor, and abdominal wall impairs normal bowel evacuation. Early after the injury, reflex emptying of the colon usually occurs. Later, fecal impaction secondary to ineffective distal colonic peristalsis can manifest as absent bowel movements or diarrheal flow around an obstructing fecal mass.¹²⁷ Rarely, colonic distention may result in autonomic hyperreflexia. A high-fiber diet, generous fluid intake, and stool softeners usually adequately prevent fecal impaction.

Genitourinary System

All patients with complete spinal cord injury and many patients with incomplete cord injury syndromes require urinary bladder drainage to relieve distention. Because of recurrent urinary tract infections associated with long-term, indwelling bladder catheters, intermittent bladder catheterization is initiated after initial stabilization. Recurrent, chronic urinary tract infections remain a cause of morbidity and mortality.^{127,177,178} Renal function often deteriorates as a result of repeated urinary tract infections and stone formation, which may be secondary to either infection or hypercalciuria. Pyelonephritis and chronic renal insufficiency or failure may lead to proteinuria, electrolyte losses, and uremia.

Electrolyte Abnormalities

Immobility stimulates calcium release from bone and, therefore, hypercalcemia usually manifests within 2 weeks after spinal cord injury; hypercalcemia eventually leads to nephrolithiasis and frequently becomes a chronic problem. Because of osteoporosis, a consequence of bone demineralization, patients with spinal cord injuries are vulnerable to fractures that occur with minimal trauma. Care must be taken when these patients are transferred and positioned in association with their routine care, examinations, or surgery.

Hyponatremia, a mild, common complication after spinal cord injury, occasionally results in severe water intoxication.¹⁸⁷ In part, this reflects a tendency of patients with spinal cord injuries toward high fluid intake, in addition to impaired free-water excretion.

Decubitus Ulceration

Pressure sores are a common, potentially expensive, and morbid complication of spinal cord injury.¹²⁷ Typically occurring over bony prominences, decubitus ulcers can be prevented by proper skin care, compulsive skin inspection, and judicious application of weight-relieving equipment. Patients in the intensive care unit should be repositioned every 2 hours, both to prevent decubitus ulceration and to prevent stasis of secretions. Air-bladder beds such as Roto-rest R and Kin-air R beds (manufactured by Hill-ROM, Charlotte, N.C.) are usually the best solution.

Nutritional Support

Adequate provision of calories and nitrogen prevents muscle wasting and therefore facilitates management of pulmonary status and later rehabilitation. Although not specifically demonstrated in patients with spinal cord injury, improved nutritional status and limitation of catabolism should reduce morbidity and mortality. Early use of the gastrointestinal tract, which can be accomplished with small nasoenteric tubes, feeding jejunostomy tubes placed during surgery, or, in some instances, conventional nasogastric tubes, is preferable to intravenous hyperalimentation. Metoclopramide may facilitate feeding by improving gastric emptying. Intravenous hyperalimentation should be considered only a short-term alternative used to reduce catabolism in patients unable to tolerate enteral feedings.

Emotional Support

The psychological impact of spinal cord injury cannot be overstated. Most casualties with spinal cord injury are young, previously healthy, active individuals; they all require psychological and emotional support in dealing with sudden disability and prolonged rehabilitation. Early involvement of patients, friends, and family is imperative.¹⁸⁸ Without consistent support, rehabilitation may prove difficult or futile.

SUMMARY

Advances in strategic planning, logistical support, and the miniaturization of devices permit the modern fighting force to have relatively sophisticated medical services in close support of forward operations. Despite the fact that mobile army surgical hospitals or equivalent hospital units may be located close to the front lines of engagement, thereby reducing evacuation time and facilitating the initiation of definitive care, combat conditions do not always permit immediate evacuation. Initial triage and treatment may be required in technologically constrained circumstances. At this level, the art of medicine must be most critically practiced within the strict guidelines of military triage. Thus, when confronted with the neurologically injured patient, time or other injuries may already have rendered the casualty with head or spinal cord injuries unsalvageable owing to intervening hypoxia, hypercarbia, or hypotension. Regardless of the apparent prognosis, resuscitation should proceed aggressively, keeping in mind the vulnerability of the acutely injured central nervous system to systemic insults.

Airway obstruction, hypoventilation, and hypoxia should be presumed to be present in any comatose patient with a head injury. Concomitant spinal cord injury should be suspected. The airway should be secured quickly. Oral intubation is best accomplished with accompanying manual in-line cervical traction. Nasal intubation is relatively contraindicated in the patient with apparent facial fractures. During intubation, cricoid pressure may limit the likelihood of aspiration of stomach contents. Ventilation should account for increased oxygen consumption.

Hemodynamic resuscitation must be accomplished promptly. Cerebral ischemia or intracranial hypertension may result from inadequately treated hypotension. Volume administration, especially in the form of 0.9% saline or more hypertonic solutions, is unlikely to increase ICP. If hypotension and bradycardia are related to acute spinal cord injury, then volume expansion, ephedrine, and atropine will restore perfusion pressure. Hypertension, a common physiological response to acute head injury, usually requires no treatment and may represent a physiological reflex that maintains CPP in the face of increased ICP.

If intracranial hypertension is suspected, then hyperventilation should be used; however, routine hyperventilation is unnecessary and ineffective. Mannitol, with or without furosemide, helps to maintain high serum osmolality, which is the key to reducing the volume of normal brain tissue, thereby lowering ICP.

Although barbiturates (which decrease $CMRO_2$, act as cerebral vasoconstrictors, and reduce ICP) may be useful in inducing anesthesia in normovolemic patients, they do not improve outcome if used routinely in all patients with head injuries. Benzodiazepines or etomidate represent appropriate alternatives for induction of anesthesia or sedation during intubation. Etomidate may be particularly useful in patients who are hypovolemic.

Working in an austere combat environment with limited material and equipment can be stressful to military medical personnel; however, the lives of many critically injured individuals can be saved and their neurological function preserved.

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