

GENERAL PRINCIPLES OF HEAD INJURY MANAGEMENT

INTRODUCTION

Traumatic brain injury continues to pose a serious health care challenge throughout the world. In the United States, trauma is the leading cause of death in individuals under 45 years of age and is a major cause of death and disability in older age groups as well. Brain injury results in more trauma deaths than do injuries to any other specific body region. Half of the 150,000 injury-related deaths that occur annually in the United States involve a serious brain injury that is primarily responsible for the patient's demise. Overall, approximately 500,000 head injuries per year in the United States are serious enough to require admission to a hospital. Despite intensive intervention, long-term disability or death occurs in the majority of severely head-injured patients. Significant neuropsychological sequelae and physical disabilities also are common in patients sustaining milder injuries.

Meaningful recovery of function after head injury is possible if patients are rapidly and effectively resuscitated, if surgical mass lesions are emergently evacuated, and if secondary insults are prevented or minimized. A key factor in recovery is the heightened vulnerability of the brain milieu following injury. After focal or diffuse insults, many neuronal, glial, and endothelial cell populations are functionally impaired. If conditions are favorable, these cells will recover with time. However, if such events as hypotension, hypoxia, or intracranial hypertension go unchecked, many vulnerable cells will succumb. Optimizing conditions for cellular recovery by maintaining adequate cerebral perfusion, normalizing intracranial pressure (ICP), and averting additional secondary insults is essential in the overall treatment strategy for the head-injured patient. Improvements in resuscitation, diagnosis, and surgical treatment may be reaching a plateau as means of further reducing morbidity and mortality. However, refinements in critical care continue to offer new avenues for enhancing outcome. Monitoring cerebral blood flow and metabolism for detection and prevention of ischemia, as well as new neuroprotective pharmacological therapies hold the greatest promise for achieving further meaningful recovery after head injury. This chapter will provide an overview of the essential principles involved in optimizing outcome after traumatic brain injury, including aspects of prehospital and emergency room care, operative and perioperative strategies, and intensive care monitoring and intervention.

PREVENTION

Clearly, the most effective means of addressing central nervous system trauma is through prevention. In the United States, Canada, and elsewhere, public education initiatives on brain and spinal cord injury, such as the Think First Program, and enactment of safety legislation have begun to help. From 1982 to 1992 seat-belt use among drivers and passengers in the United States increased from approximately 11 to 66 percent; legislation now mandates their use in 46 of 50 states. Since 1990, all new cars manufactured in the United States are equipped with automatic seat belts and/or a driver's side air bag. In 1992, air bags were estimated to have saved over 550 lives and prevented approximately 40,000 serious injuries. Motorcycle helmet laws have been implemented in 47 of 50 states. Fatalities from motorcycle use have fallen from over 4600 in 1982 to approximately 2400 in 1992. Given that head injury is the primary cause of death in most motorcycle fatalities, these laws appear to be having a beneficial effect on reducing mortality. Similarly, infant restraint seats are now mandatory in automobiles throughout the United States. Increasing use of these devices from 1982 to 1992 was estimated to have saved over 2000 infant lives. The pervasive problem of alcohol-related traffic accidents is also starting to diminish owing to broad educational efforts and strict enforcement of drunk driving laws. The legal drinking age is now 21 throughout the United States. Between 1982 and 1992, alcohol-related traffic deaths fell by over 30 percent.

despite a rising number of vehicles on the road. Overall these efforts have reduce the incidence of certain types of head injury, but further progress can still be made from broader implementation of such preemptive strategies.

SECONDARY BRAIN INJURY AND CELLULAR VULNERABILITY

Function recovery of traumatic brain injury is detemined by the severity of the initial trauma and by the occurrence of secondary insults. By the denifition, primary injury occurs immediately on impact and may leads to irreversible damage as a result of direct mechanical cell disruption. Secondary insults are physiological events that can occur within minutes, hours, or days after the primary injury and can lead to further damage of nervous tissue, prolonging and/or contributing to permanent neurological dsyfunction. Following the initial injury, many cells are functionally compromised, but can recover if provided an optimal enviroment. Such cells, however, are particularly vulnerable to the physiological challenges imposed by secondary insults. This state of vulnerability was illistrated by Jenkins and coworkers in a series of animal experiments in which a fluid percussion brain injury was followed 1 h later by a brief period of global ischemia. Each insult alone produced no morphological cell death. However, in animals sustaining concussion followed by transient ischemia, extensive hippocampal neuronal cell death occured. Such ischemic insults following human head injury appear to occur relatively frequently. Bouma and collagues documented regional of global ischemia, defined as a cerebral blood flow of 18 ml/100 g/ min or less by the xenon-133 method or by stable xenon- CT in approximately one-third of severe head injury patients evaluated within 6 to 8 h of injury. Each ischemia correlated significantly with early mortality. Hypotension and/or hypoxia was documented in 57 percent of the severe head injury patients in the Traumatic Coma Data Bank (TCDB) cohort. A single episode of hypotension was associated with an 85 percent increase in mortality.

The importance of secondary injury has been recognized for almost two decades. Graham and associates in 1978 documented ischemia brain damage in 92 percent of 151 head injury victims at autopsy, illustrating the ultimate consequences of postinjury hypotension, hypoxia and intracranial hypertension. Contemporaneous work by Jennett and Carlin suggested that further improvements in outcome after head injury would hingelargely on reversing or preventing such insults. The concept of “early diagnosis and intensive management” to pre-empt secondary insults and thus improve outcome after severe head injury was emphasized by Becker and colleagues in 1977. This aggressive medical and surgical approach, stressing maintenance of normothermia, optimizing hemodynamic function, treatment of hypoxia, cerebral ischemia, and intracranial hematomas, remains the basic therapeutic strategy fof the 1990s.

RECENT THERAPEUTIC MODIFICATIONS

Although the essential treatment approach for head injury developed in the 1970s endures, the understanding of injury pathophysiology and the resultant therapeutic concepts have evolved substantially over the last decade. These modifications appear to affect longterm neurological recovery favorably. The most important changes are a greater emphasis on maintaining an adequate cerebral perfusion pressure in relation to ICP as a means of averting cerebral ischemia, and the recognition that aggressive hyperventilation to control ICP may promote cerebral ischemia. Recent investigation have delineated other cause of secondary brain injury, including excitatory amino acid release, coagulopathy, hyperthermia, hyperglycemia, and electrolyte disturbances. Such insights have resulted in an intensified effort to prevent or minimize the impact of these insults. The following discussion on cerebral perfusion pressure, ICP, cerebral blood flow, autoregulation, and the use of hyperventilation will provide the background and rationale for current management strategies in the patient with traumatic brain injury.

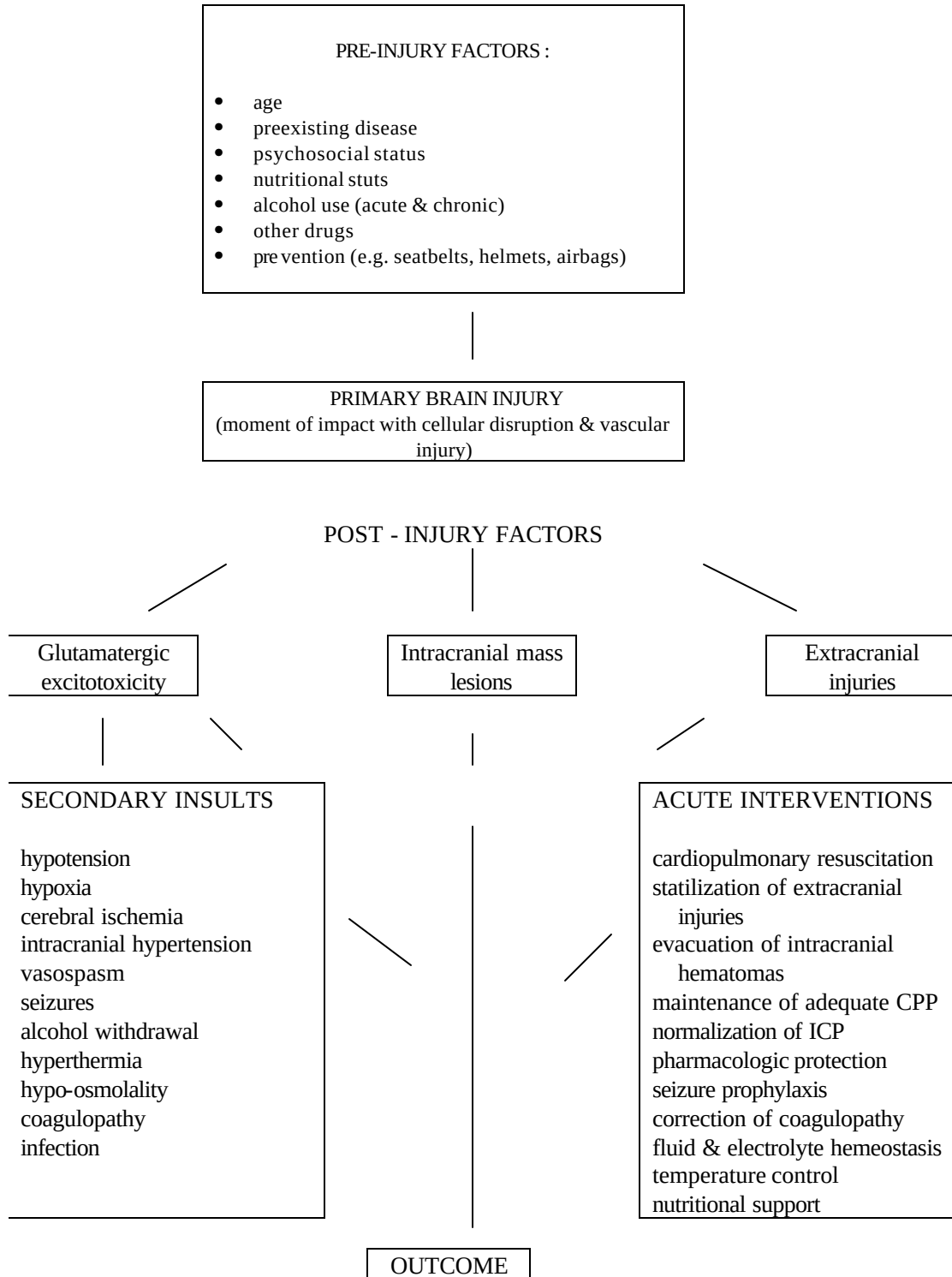


Figure 6-1. Schematic representation of factors and interventions that influence outcome before and after traumatic brain injury. (CPP = cerebral perfusion pressure)

OUTCOME IN RELATION CEREBRAL PERFUSION PRESSURE (CPP)

Randomized trials have not been performed in patients with severe head injury by using different perfusion pressure goals. A retrospective report by McGraw of over 180 closed head injury patients demonstrated significantly better outcome and lower mortality when CPP was above 80 mm Hg rather than below 80 mmHg during the first 48 h after injury. In 166 severe head injury patients, Gopinath and coworkers demonstrated that mean and lowest recorder CPP correlated significantly with outcome. The same investigation demonstrated in 163 severe head injury patients that a CPP of 60 mmHg was the critical level below which outcome was significantly worsened.

Rosner and Rosner and Marion and colleagues studied outcome after severe closed head injury (Glasgow Coma Scale 7 or less) when pressure was maintained at 70 mmHg of greater and $P_{a_{CO_2}}$ was kept in the range of 35 mmHg. Rosner reported a favorable outcome (good recovery or moderate disability) in 59 percent of 158 patients and a mortality rate of 29 percent. Marion and colleagues documented favorable outcome in 51 percent of 84 patients at 6 months postinjury, with 20 percent mortality. These two reports compare favorably with the TCDB cohort in which specific CPP goal was not established and hyperventilation to a $P_{a_{CO_2}}$ below 30 mmHg was typically employed. In patients with an initial GCS of 7 or less analyzed by the TCDB, outcome was favorable in 37 percent, while mortality was 40 percent. Even more significant is a comparison of results in patients with traumatic hematomas requiring surgery who were treated by these two different management strategies. In the series by Rosner and Rosner, favorable outcome was achieved in 59 percent of operated patients; in the TCDB patients, a favorable outcome was seen in 23 percent of operated patients.

	Favorable (%)	Unfavorable (%)	Mortality (%)
Rosner et al	59	41	(29)
Marion et al	51	49	(20)
TCDB	37	63	(40)

TABLE 6-1 Outcome after severe Closed Head Injury (GCS 7 or less) Comparing Cerebral Perfusion Pressure Therapy With Minimal Hyperventilation (*) to Traditional Management (TCDB cohort)

AUTOREGULATION, CEREBRAL PERFUSION PRESSURE AND ICP

Understanding the relationship between CPP and autoregulation is of fundamental importance in managing the head injured patient. Autoregulation of cerebral blood flow is defined as the mechanism by which a constant flow is delivered to the brain over a wide range of systemic blood pressure of CPP. CPP is the difference between the mean arterial pressure and the mean ICP. In normal subjects, mean arterial pressure ranges from 80 to 100 mmHg, ICP is 5 to 10 mmHg, and CPP ranges from 70 to 95 mmHg. Cerebral pressure autoregulation normally has a lower perfusion pressure limit of approximately 50 mmHg and an upper limit of approximately 140 mmHg. Below a perfusion pressure of 50 mmHg, compensatory cerebral arteriole vasodilation is exhausted, vessel collapse may occur, and cerebral ischemia ensues. Above a perfusion pressure of 140 mmHg, cerebral vasoconstriction is overcome, and cerebral blood flow increases passively with perfusion pressure, resulting in blood brain barrier damage and brain edema.

After traumatic brain injury, pressure autoregulation is often disturbed, yet there is considerable debate concerning the nature of this derangement. Recent studies by Bouma and Muizelaar indicate that one-third to one-half of severely head injured individuals will have some degree of autoregulatory impairment. The most clinically relevant aspect of this derangement appears to be an elevation of the lower limit of perfusion pressure at which autoregulation will function. Earlier studies suggested that a CPP of 50 mmHg to 60 mmHg

was sufficient to achieve this autoregulation threshold. More recent investigations, however, indicate that more severe head injury patients require a perfusion pressure of at least 60 mmHg to 70 mmHg to achieve autoregulatory function and to reduce ischemia insults. When perfusion pressure is maintained above this level, cerebral blood flow does not correlate with CPP. Only when perfusion pressure falls below the lower autoregulatory threshold will cerebral blood flow become directly linked to perfusion pressure.

A concern maintaining relatively high CPP has been the exacerbation of intracranial hypertension through passive increases in cerebral blood flow in patients with impaired autoregulation. In earlier studies, an association between "hyperemia" and intracranial hypertension was demonstrated in both adults and children after severe head injury. However, more recent reports suggest that blood pressure elevations do not worsen intracranial hypertension in most head injury victims and that maintaining higher CPP may, in fact, facilitate ICP control.

In a series of 66 head-injured adults, Kelly and coworkers documented hyperemia (cerebral blood flow > 55 ml/100 g/min) in 37 of 213 (17.4 percent) blood flow studies in 21 of 66 patients (31 percent). Simultaneous intracranial hypertension occurred in 21.6 percent of the studies with hyperemia compared to 11.9 percent of the studies in which blood flow was below hyperemic levels. Although the incidence of intracranial hypertension was greater with simultaneous hyperemia, in approximately 80 percent raised ICP occurred in the setting of normal or low cerebral blood flow. This study suggests that hyperemia may be one of several causes of intracranial hypertension, but in the majority of head-injured patients high cerebral blood flow is not associated with high ICP. Investigations by Muizelaar and colleagues and Robertson and coworkers have also demonstrated a lack of correlation between elevated blood flow and intracranial hypertension. In 158 severe head injury patients, Rosner and Rosner also found no positive correlation between CPP and intracranial hypertension. On average, ICP decreased after CPP reached 72 mmHg and continued to decrease up to a perfusion pressure of 112 mmHg. In individual patients, however, this critical level of CPP above which ICP began to decline ranged widely from 43 to 130 mmHg.

In two reports of severe head injury patients by Bouma and associates autoregulation was directly tested and changes in cerebral blood flow and ICP were assessed in response to induced hypertension or hypotension. These two studies demonstrated that, for the majority of patients with impaired autoregulation, induced hypertension will result in an increase in cerebral blood flow with a relatively low risk of significant intracranial hypertension. When individuals with impaired autoregulation were subjected to induced hypotension, a reduction in ICP and cerebral blood flow occurred. Equally important was the finding that, in patients with intact autoregulation, induced hypertension did not cause changes in cerebral blood flow or ICP, however, a reduction of blood pressure resulted in a significant rise in ICP.

These investigations have important therapeutic implications. When autoregulation is intact, maintenance of an adequate perfusion pressure will help prevent intracranial hypertension. A fall in blood pressure generally will result in an increase in ICP because of reflex vasodilation and an increase in cerebral blood volume. An increase in blood pressure or perfusion pressure will cause a decrease or at least a stabilization of ICP due to reflex vasoconstriction. The vasodilatory and vasoconstriction cascade models described by Rosner are helpful in understanding this process. Cyclic ICP waves originally defined by Lundberg, can be understood on the basis of these models. Both Lundberg A Waves (plateau waves) and the shorter duration B waves appear to be initiated by decrements in systemic arterial pressure. When such pressure waves occur, efforts to rapidly improve perfusion pressure should be made by ICP reduction, blood pressure elevation or both.

The vasoconstriction and vasodilation models do not apply when perfusion pressure falls below the lower autoregulation threshold or exceeds the upper autoregulatory limit. In the latter situation, an increase in CPP may cause a significant rise in cerebral blood flow. The

effect on ICP, however, is less dramatic, in significant exacerbation of intracranial hypertension does not commonly occur. The fact that ICP changed minimally with induced hypertension in the majority of patients with "impaired autoregulation" in the studies by Bouma and associates may indicate that a degree of autoregulatory control was achieved when higher perfusion pressures were reached.

While the lower perfusion pressure autoregulatory limit appears to be in the range of 70 mmHg in most severe head injury patients, the upper limit is not well defined. Perfusion pressure in the range of 105 mmHg appear to have been well tolerated for relatively brief periods in the patients studied by Bouma and associates. Whether perfusion pressure of this level are ultimately beneficial when sustained for hours or days remains to be determined. Persistent induced hypertension to maintain perfusion pressure at this level may promote cerebral edema through a damaged blood brain barrier and ultimately may prolong the period of intracranial hypertension. Additionally, systemic complications may result from prolonged vasopressor therapy utilized to maintain high perfusion pressure. In patients with significant autoregulatory impairment, induced hypertension may exacerbate intracranial hypertension through passive blood flow increases, although, as described above, this appears to be uncommon. High cerebral blood flow seen in the early injury period may also be partially attributable to transient glucose hypermetabolism, which could in turn exacerbate intracranial hypertension. These forms of hyperemia-associated intracranial hypertension, although uncommon, are probably optimally treated by suppression of the cerebral metabolic rate using high-dose pentobarbital or propofol, while still maintaining an adequate CPP. If cerebral blood flow measurements are available, this modality can help determine if hyperemia is in fact contributing to elevated ICP.

In summary, maintaining CPP at or above 70 mmHg after severe head injury is recommended because it appears to reduce the risk of cerebral ischemia, facilitates ICP control in the majority of patients and substantially improves long-term outcome compared with historical controls. This threshold, however, appears to have a rather wide range from patient to patient and has a variable time course. Some patients will maintain cerebral blood flow well above ischemic thresholds at a perfusion pressure below 70 mmHg; others may require perfusion pressure in the range of 80 to 100 mmHg. Although the upper safe range for perfusion pressure remains ill-defined, sustained levels of 90 to 100 mmHg appear to be well tolerated and may be beneficial in some patients for achieving autoregulatory function and control of ICP. When ICP and CPP cannot be maintained at normal levels (i.e., ICP below 20 mmHg and perfusion pressure above 70 mmHg), maintenance of an adequate perfusion pressure should still be aggressively pursued. With an adequate CPP, modest intracranial hypertension appears to be well tolerated if clinical status and CT findings do not suggest impending herniation from a surgically amenable mass lesion. The precise level and duration of raised ICP that does not adversely affect outcome, however, remains to be defined. Given the strong correlation between elevated ICP and poor outcome, an aggressive attempt to maintain ICP below 20 mmHg should still be made.

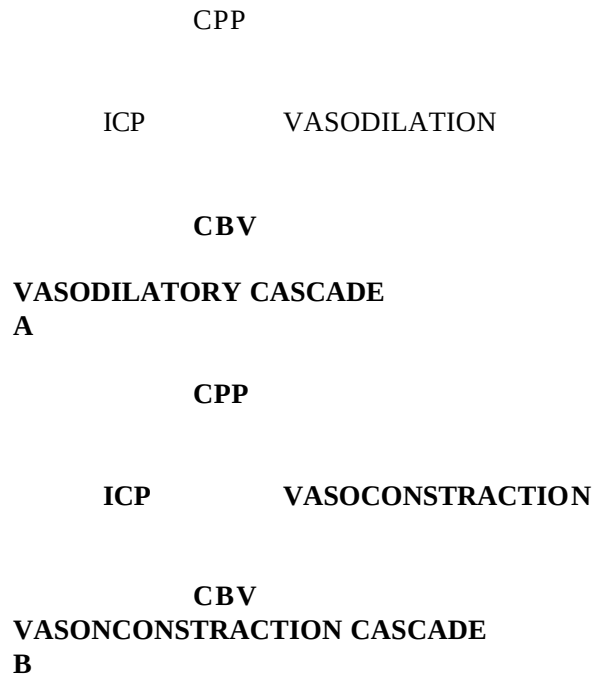


Figure 6-5 The vasodilatory and vasoconstriction cascade assume pressure autoregulatory mechanisms are intact. A. The vasodilatory cascade model illustrates how a decrease in cerebral perfusion pressure can result in cerebral vasodilation with an increase in cerebral blood volume and a rise in ICP. A drop in systemic arterial pressure is probably the most common cause of initiating the cascade. If blood pressure is not increased, perfusion pressure will continue to increase until vasodilation is maximal. B. The vasoconstriction cascade demonstrates the therapeutic effect on ICP of maintaining a higher cerebral perfusion pressure. Elevations of blood pressure with volume expansion, blood transfusion, bolus mannitol administration, and vasopressor therapy can all help initiate the cascade.

HYPERVENTILATION

Despite the effectiveness of hypocapnia in reducing cerebral blood volume and ICP, its use has recently been curtailed in severe head injury patients. This change is based on evidence that maintaining pa_{CO_2} in the range of 25 to 30 mmHg can result in significant cerebral vasoconstriction and marked reductions in blood flow. In a randomized trial by Muizelaar and coworkers of severe head injury patients maintained at an average pa_{CO_2} of either 25 or 35 mmHg, the patients treated with normocapnia had a significantly better outcome at 3 and 6 months postinjury. This difference in outcome, however, was seen only in patients with Glasgow motor scores of 4 or 5 and was not present 1 year after injury.

Gopinath and colleagues studied that effects of hypocapnia in 116 severe head injury patients with continuous jugular venous oxygen saturation (Sjv_{O_2}) monitoring. In patients with episodes of oxygen desaturation, the cause was attributed to a pa_{CO_2} of less than 29 mmHg in 27 percent. Single or multiple episodes of desaturation to an Sjv_{O_2} of less than 50 percent strongly correlated with poor outcome. The good outcome in severe head injury patients reported by Rosner and Rosner and Marion and coworkers with perfusion pressure management and maintenance of normocapnia, also suggests that hyperventilation is not essential for achieving a high rate of functional survivors.

Hyperventilation probably has its most deleterious effect during the first 24 h after severe head injury, when cerebral blood flow is typically at its lowest. It is therefore recommended that pa_{CO_2} be maintained at approximately 35 mmHg within the first 24 h after injury, and in the range of 30 to 35 mmHg after that time if ICP control is problematic. Although hyperventilation is effective in lowering ICP at least transiently, it is generally not essential if other standard methods are utilized. Dissemination of this information to emergency room physicians and paramedics is warranted, given that significant hyperventilation is still commonly practiced in the initial management of the comatose head-injured patient.

INITIAL MANAGEMENT

PREHOSPITAL AND EMERGENCY ROOM CARE

Assessment and stabilization of the head injury victim begins at the injury scene by emergency medical personnel. Their tasks include securing the patient's airway, initiating fluid resuscitation, stabilizing the cervical and thoracolumbar spine, identifying and stabilizing extracranial injuries, and assessing the patient's level of consciousness. Obtaining information on the mechanism of injury and providing rapid transport to a qualified medical facility are also critical components of initial management.

Once the victim is in the hospital, care should progress in a rapid and systematic manner, with diagnostic and therapeutic maneuvers proceeding simultaneously. This approach is best facilitated by a designated trauma team, which has become the standard of care for acute management of the trauma victim. As in the prehospital setting, the most immediate concerns are establishing a secure airway. Providing adequate ventilation, and correcting or preventing hypoxia and hypotension. Adequate venous access with a minimum of two large-bore peripheral intravenous lines (16-gauge or larger) is essential in managing the hypotensive, hypovolemic trauma patient. Simultaneously, a general survey is performed to assess for major extracranial injuries. Life-threatening insults such as tension pneumothorax, cardiac tamponade, or major vascular injuries with hypovolemic shock take precedence over neurological injury and are addressed immediately. Initial radiological studies in the trauma patient include a three-view cervical spine series plus chest and pelvic films. An indwelling catheter to monitor urine output and an oral or nasogastric tube are placed. Contraindications to nasogastric tube insertion include suspected anterior cranial base or midface fractures. Initial laboratory studies should include a type and cross-match for possible blood transfusion, complete blood count, serum electrolytes, glucose, creatinine, arterial blood gas, prothrombin time and partial thromboplastin time, and urinalysis. A blood alcohol level and a urine toxicology screen for narcotics, benzodiazepines, amphetamines, cocaine, and phencyclidine (PCP) are also useful.

HYPOXIA AND AIRWAY MANAGEMENT

Hypoxia during resuscitation was documented in 46 percent of patients in the TCDB cohort and was significantly associated with poor outcome. Transient or prolonged apnea after concussive brain injury is a well recognized phenomenon and a major contributor to postinjury hypoxia. Associated injuries such as flail chest, hemothorax or pneumothorax, upper airway trauma, and cervical spinal cord injury will also impair ventilation and oxygenation. Intoxication from alcohol or other sedative-hypnotics will diminish respiratory drive and protective airway reflexes as well.

In patients with poor airway protection or inadequate ventilatory effort, prompt endotracheal intubation is required. In recent years, as emergency medical technicians have become more highly trained, endotracheal intubation at the injury scene has become commonly practiced. Specific indications for intubation in the head-injured patient include inability to maintain adequate ventilation, impending airway loss from neck or pharyngeal injury, poor airway protection associated with depressed level consciousness, or potential for neurological deterioration when being transported out of the emergency room. All patients who are not verbalizing and who cannot follow commands should be intubated promptly. In general, this means that all patients with GCS of 8 or less require intubation and assisted ventilation.

The vast majority of head injury victims can be safely intubated via the orotracheal route with in-line stabilization of the cervical spine, even prior to obtaining radiographs of the cervical spine. This method of intubation, with strict maintenance of a neutral head position and slight to no axial traction, cause minimal movement of the potentially unstable cervical spine and has not been associated with new neurologic deficit in three clinical series. The nasotracheal route of intubation is contraindicated in patients with possible fractures of the anterior cranial base and/or midface, requires considerably more experience and time, and has a lower success rate and a higher complication rate than does oral intubation. In patients with major facial or upper airway trauma, a surgical airway by cricothyroidotomy may be required, although the complication rate in the emergency setting may be as high as 32 percent.

Orotracheal intubation in the head-injured patient is best facilitated by rapid sequence induction utilizing thiopental, 3 to 5 mg/kg, and succinylcholine, 1 to 2 mg/kg. In recent years, as an alternative to thiopental, the ultra-short-acting hypnotic anesthetic agent etomidate appears to be safe and effective for rapid sequence induction. Once intubated, adequate sedation and paralysis are essential to help prevent dangerous spikes in ICP caused by "bucking" on the endotracheal tube or excessive motor activity. Narcotic sedation with morphine or fentanyl is effective, and neuromuscular blockade with vecuronium or pancuronium can be added if excessive motor activity persists.

HYPOTENSION AND FLUID RESUSCITATION

Hypotension was documented in almost 35 percent of the severe head injury patients in the TCDB cohort and was an even more ominous predictor of poor outcome than was hypoxia. Klauber and colleagues documented a mortality of 35 percent in head-injured patients admitted with a systolic blood pressure of less than 85 mmHg compared with a mortality of only 6 percent in patients presenting with higher systolic blood pressure. Hypotension or hypoxia also have been shown to correlate with diffuse brain swelling on CT.

Most head injury victims with hypotension and multisystem trauma have sustained significant volume loss and require rapid fluid resuscitation. Volume replacement can significantly improve cerebral perfusion and often results in a dramatic improvement in neurological status. The optimal method of fluid resuscitation in the hypotensive, hypovolemic brain injured patient remains controversial. Isotonic saline (0.9 % Na Cl) is the most commonly used and least expensive preparation for volume resuscitation. A concern when using isotonic saline has been the development of brain edema. Experimental and clinical studies suggest that the risk of aggravating brain edema with isotonic crystalloid fluid resuscitation is minimal provided hypo-osmolality does not develop. However, infusion of even mildly hypotonic saline for fluid resuscitation has been associated with increased brain edema in experimental studies. Because commercially available Ringer's lactate solution is significantly more hypo-osmolar than its calculated osmolality of 273 mOsm/kg, use of 0.9 % normal saline with an osmolality of 308 mOsm/kg is recommended.

Recently, hypertonic saline or colloid infusion have been use in the acute resuscitative phase after head injury. Such solution are potent volume expanders and may help prevent deveploment of brain edema and intracranial hypertension by creating a favorable gradient for the movement of free water from the extravascular to the intravascular compartment. Both experimental and clinical studies suggest that hypertonic saline may prove to be an effective form of early volume expansion in the head injured patient. The use of colloid for acute volume expansion is appealing because compounds such as albumin for 6 % hetastarch have a high molecular weight and a relatively low vascular permeability. With longer intravascular retention time their effect on volume expansion is more sustained compared to isotonic crystalloid, and theoretically the risk of aggravating brain edema is diminished. However, reviews of randomized trials comparing crystalloid versus albumin for fliud resuscitation have shown no difference in survival and a significantly greater cost when using albumin.

Currently, neither hypertonic saline nor colloid preparations appear to have a distinct advantage over isotonic crystalloid as a means of achieving prompt volume expansion in the head-injured hypovolemic patient. Rapid infusion of 1 to 2 liters of 0.9% NaCl generally is effective in hemodynamically stabilizing most trauma victims. If hypotension is not reversed after 1 to 2 liters of crystalloid and/or colloid, a central venous line should be inserted, and transfusion of packed red blood cells is indicated. Despite their widespread use, no studies have demonstrated an improved survival with the use of military antishock trousers (MAST) in the multiple trauma petient.

NEUROLOGICAL EVALUATION

THE GLASGOW COMA SCALE

One of the most important factors in early treatment decisions and in long-term outcome after head injury is the patient's initial level of consciousness. Although many methods of defining level of consciouness exist, the most widely used measure is the Glasgow Coma Scale (GCS) first presented by Teasdale and Jennett in 1974. The utility of this scaling system is in its objectivity, reproducibility, and simplicity. It provides paremedics, nurse and physicians with a rapid a measure of level of consciousness, obviating ambiguous terminology such as "lethargic," "stuporous ," and "obtunded". Because the level of consciousness can be lowered independent of head injury by severe factors, including shock, hypoxia, hypothermia, alcohol intoxication, the postictal state, and administering of sedatives or narcotics, the postresuscitation GCS is generally, considered more reliable in providing an initial assessment of injury severity. Although not consistently defined, this score generally refers to the best GCS obtsined within the first 6 to 8 h of injury following nonsurgical resuscitation. Injury severity is generally categorized into three levels based on the postresuscitation GCS. Minor or mild head injury includes patients whose initial GCS is 13 to 15; moderate injury includes patients with a GCS of 9 to 12; severe injury refers to a GCS of 3 to 8 or a subsequent deterioration to a GCS of 8 or less. These categorizations, although arbitrary, have helped to define the spectrum of traumatic brain injury and are useful in guiding therapy and providing important prognostic information.

Glasgow Coma Scale

EYE OPENING

- 4 = Spontaneously**
- 3 = To voice**
- 2 = To pain**
- 1 = None**

VERBAL RESPONSE

- 5 = Oriental**
- 4 = Confused**
- 3 = Inappropriate words**
- 2 = Incomprehensible sounds**
- 1 = None**

MOTOR RESPONSE

- 6 = Follows commands**
- 5 = Localizes to pain**
- 4 = Withdrawal to pain**
- 3 = Abnormal flexion**
- 2 = Abnormal extension**
- 1 = None**

Figure 6-7. Introduced by Teasdale and Jannett in 1974, the Glasgow Coma Scale provides an objective, rapidly performed assessment of the level of consciousness. The postresuscitation GCS is one of the strongest predictors of long-term outcome following craniocerebral trauma.

NEUROLOGICAL EXAMINATION

In the acutely head injured patient, the neurological examination is necessarily abbreviated and should focus on the level of consciousness, the pupillary light reflexes, extraocular eye movements, the motor examination, and lower brainstem reflexes for patients in deep coma. As part of this initial survey, the head should be carefully palpated to detect bony step-offs, and all scalp lacerations should be gently probed to assess for depressed fractures and foreign bodies. Signs of basal skull fractures also should be sought, including hemotympanum, cerebrospinal fluid otorrhea or rhinorrhea, and retromastoid or periorbital ecchymosis and tenderness. This initial assessment, along with inspection of the neck and thoracolumbar spine, should take no longer than 5 to 10 min. Before leaving the emergency department for CT, the cervical spine should be evaluated with a lateral view x-ray, which includes the cervicothoracic junction. If time permits, anteroposterior and open mouth views should also be obtained before leaving the emergency department.

The triad of a deteriorating level of consciousness, pupillary dilation, and an associated hemiparesis has long been recognized as highly suggestive of a hemispheric mass lesion causing transtentorial herniation. However, any one of these findings in a patient after head injury may be a manifestation of a traumatic hematoma and when noted should heighten the urgency of evaluation. Such signs also may be absent, falsely localizing, or may occur in patients with diffuse brain injuries. Other etiologies of diminished consciousness also should be considered in the patient who appears to have sustained a head injury, including seizure activity, the postictal state, alcohol intoxication, drug overdoses, and severe hypoglycemia.

Because the neurological examination is frequently unreliable in accurately predicting intracranial pathology, radiological evaluation, preferably with CT, is always indicated in the acutely head injured patient.

A CT is indicated in all head-injured patients with a depressed level of consciousness, including those who are heavily inebriated. Too frequently, alcohol-intoxicated patients are initially observed in the emergency room and CT is prompted by neurologic deterioration. A timely CT is also indicated in all patients with a GCS of 15 who sustained a loss of consciousness, are amnesic to the injury, have a focal neurological deficit, or have signs of a basilar or calvarial skull fracture. An axial CT without contrast rapidly defines intracranial lesions and determines whether urgent neurosurgical intervention is required. Obtaining both brain and bone "windows" will help determine the etiology and the significance of focal neurological findings and whether a skull fracture is present. When CT is available, skull x-rays are obviated. When CT is of limited availability or nonexistent, skull x-rays still have a role, as demonstrated by the study of Miller and coworkers. If a skull fracture is seen, and if the patient is stable, further close observation is indicated; or, preferably, the patient can be transferred to a facility where CT is available. In patients who are comatose or deteriorating, if a neurosurgeon is present, further diagnostic evaluation with cerebral angiography, contrast or air ventriculography, or exploratory burr hole is indicated.

Scanning Criteria Following Craniocerebral Trauma

GCS 14 or less

GCS 15 with:

- **documented loss of consciousness**
- **amnesia for injury**
- **focal neurological deficit**
- **signs of basal or calvarial skull fracture**

Figure 68. Given the significant risk of rapid deterioration in the mildly head injured patient, the threshold for obtaining a timely CT scan should be low.

THE DETERIORATING PATIENT

Despite the strong predictive value of the GCS a significant portion of patients with seemingly minor injury, based on their initial level of consciousness, will have sustained a serious intracranial insult. Klauber and associates analyzing over 7900 head trauma victims from 41 hospitals, concluded that the excessive mortality noted in some of the hospital was likely due to inadequate observation of the less severely injured patients. Stein and Ross specifically addressed individuals with a postresuscitation GCS of 13. In 658 patients with an initial GCS of 13 to 15, 18 percent had abnormalities on CT scan and 5 percent required surgery. Of the 62 patients with a GCS of 13, 40 percent had abnormalities and 10 percent required surgery. They concluded that such patients should be considered to have sustained a moderate head injury and that their initial management should be more rigorous and vigilant than is customary for individuals with a GCS of 14 or 15.

Severe reports have addressed the problem of patients who initially are conscious after injury but then worsen to a GCS of 8 or less. Such patients who "talk and deteriorate"

have comprised 10 to 32 percent severe of head injury victims. Lobato and colleagues noted that 25 percent of 838 severely head injured patients talked at some point prior to lapsing into coma. Almost 36 percent of these patients were fully oriented during their lucid interval. Most notably, 80 percent had a focal mass lesion, requiring evacuation in the majority of cases. Over a 10- years period Miller and coworkers documented 183 patients with closed head injury, an initial GCS of 15, and who subsequently required evacuation of a traumatic hematoma. This cohort represented 17 percent of all patients requiring craniotomy for acute traumatic hematoma during the study period. Approximately 40 percent of patients reported no loss of consciousness, posttraumatic amnesia, headache, or vomiting when first evaluated. However, 33 percent had a focal neurological deficit despite being fully conscious, 19 percent had signs of basal skull fracture, and 60 percent had a skull fracture on skull x-ray. These studies provide strong rationale for close monitoring the conscious head-injury patient, including frequent neurological assessment and an early CT.

EARLY MANAGEMENT OF INTRACRANIAL HYPERTENSION

Measure to control intracranial hypertension should begin prior to obtaining a CT in individuals who arrive in coma, have a precipitous decline in level in consciousness, or develop pupillary asymmetry or hemiparesis, given the likelihood that a traumatic mass lesion is responsible for the patient's deterioration. Such interventions include intubation and assisted ventilation, sedation bolus mannitol therapy, and administration of prophylactic phenytoin. Prevention of hypotension by establishing euvolemia and in some cases vasopressor therapy also is critical to maintain adequate CPP and to help control ICP. This aggressive approach will result in overtreatment of some patients, but may be a deciding factor in other whose precarious neurological state is greatly improved by such interventions. Rapid implementation of these maneuvers will generally "buy time" and allow for a precise CT diagnosis to be made.

Once a secure airway is established, narcotic sedation with morphine to fentanyl should be achieved; a neuro muscular blocking agent such as vecuronium or pancuronium can be added when agitation or abnormal posturing persist. Mild hyperventilation to achieve pa_{CO_2} of 35 mmHg should be instituted and a 0.5 to 1.0 g/kg intravenous bolus of mannitol administered. The risk of inducing hypotension in the hypovolemic patient by mannitol administration is low, provided adequate fluid replacement with normal saline and/or colloid administration is occurring simultaneously. Mannitol used in this manner is highly effective in treating both intracranial hypertension and hypovolemia and will improve CPP. Prophylactic anticonvulsant therapy with phenytoin (18 mg/kg intravenous loading dose) should be administered as an additional preemptive measure against intracranial hypertension given that generalized seizures in a patient with an intracranial mass lesion can have devastating consequences.

Emergent Management of Intracranial Hypertension

- **Intubation**
- **Controlled ventilation to PaCo₂ 35 mmHg**
- **Volume resuscitation**
- **Establishment of normotension**
- **Narcotic sedation/ neuromuscular blockade**
- **Bolus mannitol 1 gram/kg**
- **Phenytoin 18 mg/kg**

Figure 6-9. When head injured patients arrive in coma (GCS 8 or less), precipitously deteriorate in level of consciousness, or have pupillary dilation or obvious hemiparesis, such interventions are urgently indicated prior to obtaining a head CT.

SPECIAL CONCERN IN THE MULTIPLE TRAUMA PATIENT

Extracranial trauma complicates 30 to 70 percent of head injuries, approximately 5 percent of head injury victim sustain a cervicel spine injury. In the TCDB cohort multiple trauma accrued in 70 percent of patients but was not a major determinant of mortality in any age group. Only when severe extracranial injuries accompany mild or moderate head injury is mortality significantly influenced by associated injuries. Because of these aggressive prehospital and emergency care common today, many critically ill trauma patients are intubated, sedated, and pharmacologically paralyzed before being seen by a neurosurgeon. Close cooperation and communication with the trauma of emergency physicians is essential in order to obtain information on the patient's initial neurological status and to plan a coherent sequence of diagnostic and therapeutic procedures. If the neurosurgeon is not present during the initial moments of evaluation, the emergency or trauma physicians must be able to perform an adequate neurological examination and be adept at making appropriate early management decisions regarding the patient's neurological status. For example, patients should not be detained in the emergency room to undergo diagnostic peritoneal lavages or other lengthy investigation studies prior to head CT if they are hemodynamically stable and have no findings to suggest a serious intraabdominal or thoracic injury. Adherence to this principle is especially important in all moderate and severe head injury patients and in those who have a focal neurological deficit or evidence of a calvarial or skull base fracture. On occasion, however, a patient will be so hemodynamically unstable as to require emergency thoracotomy or laparotomy prior to obtaining a head CT. In this situation, the initial neurological assessment should determine whether ICP monitoring or exploratory burr holes are required while the patient is under general anesthesia. In patients with localizing signs such as significant pupillary asymmetry or marked hemiparesis, or in those with abnormal posturing, exploratory burr holes are warranted. In patients with a GCS of 13 or less without localizing signs, intraoperative ICP monitoring is recommended, given that most of these patients will have significant intracranial injuries. Any patient with a GCS of 14 or 15 who

has extensive external craniocervical trauma also should be considered for monitoring. For patients with a GCS of 8 or less, a ventriculostomy with the ability to drain cerebrospinal fluid is indicated, given the high likelihood of raised ICP in such patients. For those with GCS of 9 or greater, a less invasive monitoring technique such as parenchymal fiber-optic probe may be more prudent and fraught with fewer complications than “blind” ventriculostomy placement.

Because hypotension can significantly depress the level of consciousness independent of head injury, a significant number of patients who undergo intraoperative pressure monitoring as outlined by this algorithm ultimately will not have needed it. The consequences, however, or undiagnosed and untreated intracranial hypertension argue against a less invasive approach. When these patients leave the operating room after their emergency procedure, they should be taken directly for head CT. The noninvasive technique of transcranial nearinfrared spectroscopy has recently been demonstrated by Gopinath and coworkers to be highly accurate in diagnosing traumatic intracranial hematomas. This technique may have its most useful application in hemodynamically unstable multiple trauma patients who must be taken emergency to the operating room prior to CT.

MONITORING AFTER INITIAL RESUSCITATION

Less than half of severe head injury victims and significantly fewer sustaining a moderate closed head injury will require evacuation of a traumatic intracranial hematoma. Whether surgery is required or not, the intensity of monitoring and medical intervention will be determined largely by the patient’s level of consciousness and CT findings. Given the significant risk of neurological deterioration following closed or penetrating injuries of even moderate severity, all such individuals warrant careful and frequent observation, preferably in an intensive care unit. In nonintubated moderate head injury patients, minimal monitoring should include continuous electrocardiogram recording and pulse oximetry; neurological assessments should be performed every 30 min for at least the first 6 h after injury and then on an hourly basis until the patient’s condition has clearly stabilized or is improving. A loss of two points or more on the GCS, new pupillary asymmetry, or hemiparesis warrant an urgent repeat CT. Worsening headache, persistent vomiting, or seizure activity also should prompt an urgent reevaluation by CT. Monitoring of severe head injury patients is deferred to the discussion of critical care in traumatic brain injury.

EARLY REPEAT CT AND COAGULOPATHY

When potential surgical lesions are seen on the initial CT, a repeat scan within 4 to 8 h of the first scan should be part of routine monitoring. Based on a review of 154 consecutive closed head injury patients requiring surgical intervention, McBride and colleagues found that 47.5 percent of patients who had initial nonoperative lesions on CT required surgical intervention based on the findings of the follow-up CT. In a recent study by Stein and associates, 44.5 percent of 337 consecutive patients sustaining a closed head injury developed delayed or progressive lesions seen on a follow-up CT scan. Factors that were significantly associated with the development of delayed cerebral insults included increasing severity of the initial head injury as defined by GCS, the need for cardiopulmonary resuscitation at on

the first CT scan, and the presence of coagulopathy upon admission. Further addressing coagulopathy after closed head injury, the same investigators found elevated prothrombin time, partial thromboplastin time, or low platelet count in 37.5 percent of 253 patients upon admission. In those patients who developed delayed brain injury as defined above, 55 percent had an abnormal prothrombin time, partial thromboplastin time, or platelet count ; in those whose follow-up CT scans did not worsen. Only 9 percent had abnormal coagulation studies. The risk of developing a delayed insult on CT was 85 percent in those with a least one abnormal clotting study and only 31 percent for those without such abnormalities. When coagulopathy is present upon admission. Stein and associates advocate early repeat CT scanning. This concept of timely follow-up evaluation is particularly relevant in many of today's trauma centers, where patients are routinely scanned within 1 to 2 h of injury, when the hemorrhagic process may still be evolving.

INDICATIONS FOR SURGERY

The most critical factors in deciding whether to proceed with surgical evacuation of an intracranial hematoma are the patient's neurological status, the imaging findings, and the extent of extracranial injury. There is little debate that surgical intervention is indicated in a rapidly deteriorating patient who harbors an expanding intracranial hematoma causing significant mass effect. In general, all acute traumatic extraaxial hematoma 1 cm or greater in thickness warrant urgent evacuation. For less obvious situations controversy persists, but severe reasonable statements can be made. Generally, a subdural hematoma or epidural hematoma of over 5 mm in thickness with an equivalent midline shift in a comatose patient (GCS 8 or less) should be evacuated urgently. On the other hand, surgical decompression of a thin-rim subdural hematoma of 3 mm or less, associated with marked hemispheric swelling and a large midline shift, is unlikely to improve the patient's condition or reduce ICP. Such patients are best managed medically. A CT should be repeated, especially if the first scan is obtained within a few hours of injury or if there are other associated lesions, such as contusions that may have progressed in the interim.

Another somewhat controversial group of patients are those with an acute epidural or subdural hematoma 5 to 10 mm thick who have a GCS of 9 to 13. An urgent operative course is warranted in all patients with a deteriorating level of consciousness, pupillary abnormalities, hemiparesis, or when CT reveals effacement of the basilar cisterns or a hematoma in the middle fossa causing mass effect. Patients that can be managed in an intensive care setting with frequent neurological examinations include those with a stable or improving level of consciousness, no focal deficits, and normal basilar cisterns on CT with no appreciable midline shift. Often such patients are older and have some degree of cortical atrophy. Any deterioration warrants a repeat CT or in some cases a direct trip to the operating room. Another group of patients who warrant initial conservative management are those with an interhemispheric subdural hematoma without neurological deficit, given the risks of operating along the sagittal sinus.

Perhaps the most controversy exists over when to evacuate intracerebral hematomas and hemorrhagic contusions, and whether such removal is helpful in controlling ICP and improving outcome. Miller and coworkers in their experience with over 200 severely head

injured patients found that despite removing such lesions, patients with contusions or intracerebral hematomas had the highest incidence of intracranial hypertension of all subgroups in the study. Consequently, they recommend an initial nonoperative course in most cases, resorting to surgery if medical management of intracranial hypertension has failed. Others, such as Cooper, believe that early surgical removal of larger intracerebral hematomas and cerebral contusions provides early control of ICP and helps prevent the cascade of secondary events leading to later intracranial hypertension.

Despite such debate, there is general agreement that the decision to remove an intracerebral hematoma or cerebral contusion should be based on several key factors, including the size of the lesion, its depth and exact location from the cortical surface, the presence of associated lesions, and the patient's neurological status and ICP. Cortical contusion over 2 cm in diameter generally should be removed if there is significant mass effect and ICP is difficult to control medically. Frontal pole and temporal lobe hematomas or contusion 2 cm in diameter or more with significant mass effect, basilar cistern impingement, and midline shift of over 5 mm also should be removed if ICP management is problematic. An initial conservative approach is warranted, however, when eloquent cortex is involved, such as dominant temporal lobe lesions or lesions near the central sulcus. An expectant course is also indicated in comatose but neurologically stable patients with small lesions associated with less than 5 mm midline shift and open basilar cisterns on CT. Similarly, lesions confined to the deep white matter or basal ganglia are best managed without surgery. ICP should be carefully monitored and controlled by medical means. If such measures fail, the patient can be reconsidered for surgery. In patients with a GCS of 9 to 13, a conservative course in an intensive care setting is reasonable in individuals with cerebral contusions or an intracerebral hematoma, provided they have a stable or improving level of consciousness, midline shift of 3 mm or less, and no significant basilar cistern effacement is seen on CT.

Finally, there are situations where the dismal clinical status of the patient warrants a nonoperative course despite the presence of a radiographic surgical lesion. Such cases include the adult patient who after resuscitation remains flaccid with a GCS of 3, nonreactive and dilated pupils, and without spontaneous respirations. Similarly, in patients over the age of 75 with a GCS of 5 or less, a nonoperative course generally should be taken given their almost uniformly poor outcome with or without surgery. All other patients with surgical mass lesions, even those with a postresuscitation GCS of 3, but at least one reactive pupil, warrant emergent operative intervention. A significant, albeit small, portion of such patients will „make a satisfactory recovery. Additionally, in some patients a low GCS may in part be due to intoxication or the postictal state.

CRITICAL CARE IN TRAUMATIC BRAIN INJURY

Intensive care of the patient with traumatic brain injury has two primary and interrelated goals: maintenance or reestablishment of neurological and systemic homeostasis and early detection of neurological deterioration. Of principal importance is achieving adequate CPP and normalizing ICP as well as minimizing or preventing additional secondary insults resulting from hypotension, hypoxia, seizures, hyperthermia, electrolyte disturbance, coagulopathy, and infection. Although the following guidelines pertain largely to patients sustaining severe head injuries, the discussion also is highly relevant to those with moderate

injuries. In individuals with less severe injuries secondary insults may have the greatest impact.

ESSENTIAL MONITORING

Monitoring equipment for the severe head injury patient should include an ICP monitor, central venous pressure line, an arterial pressure line, and continuous pulse oximetry. An end-tidal CO₂ monitor is also useful to allow timely adjustment of the ventilatory rate when spontaneous pa_{co2} changes occur.

HEMODYNAMIC MONITORING

Hemodynamic monitoring and support is a critical component in the overall management strategy of severe head injury patients. The goals of monitoring include optimizing volume status and cardiac function, maximizing tissue perfusion, and averting complications of fluid management and pharmacological hemodynamic therapy. A central venous pressure line or a pulmonary artery (Swan-Ganz) catheter is recommended for management of all severely injured patients. In most young healthy adults, central venous or right atrial pressures tend to reflect volume status and left ventricular function, especially when used in conjunction with other clinical parameters such as blood pressure, pulse rate, and urine output. However, in order or critically ill patients, this correlation often fails. Consequently, a pulmonary artery catheter is recommended for patients over the age of 50 to 60 years, for individuals with preexisting hypertension, cardiac or pulmonary disease, and for those with major extracranial trauma, particularly spinal cord, chest, or abdominal injuries, or when vasopressor or high-dose barbiturates are used. Hypovolemic or cardiogenic shock, respiratory failure, sepsis, or multisystem organ failure also are indication for Swan-Ganz catheterization. In critically ill patients, intracardiac monitoring with measurement of the pulmonary artery wedge pressure gives a more accurate estimation of left ventricular function and left ventricular preload than does the central venous pressure. Other parameters such as mixed venous oxygen saturation and systemic vascular resistance also can be determined to further guide hemodynamic therapy.

MONITORING CEREBRAL PHYSIOLOGY

INTRACRANIAL PRESSURE

Numerous reports demonstrate a significant correlation between intracranial hypertension and poor outcome following traumatic brain injury. All patients with an initial postresuscitation GCS of 8 or less warrant ICP monitoring. Intracranial pressure monitoring also should be considered in patients with a postresuscitation GCS of 9 to 13 who have intracranial hematomas or cerebral contusions causing mass effect that initially do not warrant surgical evacuation. This statement is not meant to advocate an indiscriminate use of ICP monitoring, instead, it accepts the reality that a significant number of patients may subsequently deteriorate as their intracranial injuries evolve. Monitoring of ICP in these patients provides an early warning system for herniation or decreased cerebral perfusion.

The preferred method of monitoring is an intraventricular catheter, given the therapeutic benefit of cerebrospinal fluid drainage for ICP control. Even with diffuse cerebral swelling, most lateral ventricles can be cannulated after one or two attempts. If, however, a ventriculostomy is not placed within three passes, a parenchymal fiber-optic probe

is an acceptable alternative. Repeated passes of a ventricular catheter have been associated with increased risk of intracerebral hematoma, especially in patients with coagulopathy.

The criteria for how long to monitor ICP are not firmly established. Posttraumatic swelling, edema, and progression of hemorrhagic lesions typically are maxima within 48 to 96 h of injury. However, delayed rises in ICP are not uncommon. In a recent series of 53 severe head injury patients, 15 (31 percent) develop a secondary rise in ICP occurring 3 to 10 days after injury. In 6 of the 15 patients, the delayed rise in ICP was uncontrollable, resulting in death; only 2 patients had a good outcome. The most frequent initial diagnoses in these 15 patients were multiple contusions and acute subdural hematoma.

Discontinuation of ICP monitoring is reasonable in patients who maintain normal ICP without specific therapy or with only minimal sedation for at least 24 h. Such patients should also show significant and steady clinical improvement to a GCS of 9 or greater (unless a primary brainstem or diffuse axonal injury is evident), and demonstrate resolving lesions on follow-up CT scans, including visible cisterns. Even if these criteria are met, a longer period of observation may be warranted when initial diagnosis is acute subdural hematoma or multiple contusions, in patients with significant vasospasm, and in those patients with major systemic derangements. Intraoperative ICP monitoring is also generally recommended in moderate or severe head injury patients undergoing general anesthesia within 7 to 10 days of injury for an extracranial operation that cannot otherwise be delayed.

CEREBRAL BLOOD FLOW

As the detrimental effects of cerebral ischemia have become more apparent, greater emphasis has been placed on assessing regional and global cerebral blood flow and metabolism. Measurements of cerebral blood flow with xenon-133, stable xenon CT, or the nitrous oxide saturation method are established techniques and are becoming frequently utilized in the neurosurgical critical care setting. Of the three methods cited, the largest experience has been with the xenon-133 method, starting in the mid-1970s with investigations by Obrist and colleagues. The major advantage of this technique is that it can be performed at the patient's bedside and repeated as needed. Its major drawback is that it essentially provides a hemispheric cortical blood flow measurement, predominantly of the middle cerebral artery distribution. Local areas of critically low blood flow may not be detected. Stable xenon CT does have significantly higher resolution and can provide regional cortical, basal ganglia, and brainstem blood flow measure along with a simultaneous CT anatomic image. The major problem with this technique is that it requires patient transport.

Regardless of the method used to measure cerebral blood flow, such assessments are ideally started within the first 24 h after severe head injury when ischemia is most likely to occur and then on a serial basis over the next several days. The patients most likely to benefit from regular blood flow measurements are those with problematic intracranial hypertension and marginal CPP. In such patients, blood flow assessment can help guide therapeutic maneuvers such as CPP management and use of hyperventilation.

JUGULAR VENOUS OXYGEN SATURATION

Indirect assessment of global cerebral blood flow and metabolism by measurement of jugular venous oxygen saturation is becoming more commonly employed in severely head injured patients. Placement of a fiberoptic catheter into the jugular bulb allows continuous recording of jugular venous oxygen saturation (Sjv_{O_2}), determination of arteriojugular venous oxygen difference (AVD_{O_2}) and in conjunction with cerebral blood flow measurement,

determination of the cerebral metabolic rate for oxygen (CMR_{O_2}). Jugular venous oxygen saturation monitoring appears to be an effective means of detecting episodes of global cerebral ischemia. Normal Sjv_{O_2} is approximately 65 percent; an Sjv_{O_2} below 50 to 55 percent is considered indicative of global ischemia warranting treatment. Because over one-half of recorded desaturations may be due to poor catheter position or calibration, an algorithm for diagnosing the cause of desaturation is necessary. Once this process has been performed and a true episode of desaturation has been documented, appropriate intervention is warranted. In the majority of cases, reduction of ICP, elevation of the Pa_{CO_2} to normocarbic levels, or elevation of the systemic blood pressure will be required to correct the desaturation.

Gopinath and colleagues have recently documented their experience with 166 severe head-injured patients undergoing continuous jugular venous oxygen saturation monitoring during the first five days after injury. One of more confirmed episodes of desaturation to an Sjv_{O_2} of less than 50 percent occurred in 40 percent of patients and was significantly associated with poor outcome; 90 percent of patients sustaining multiple episodes of desaturation had a poor outcome compared to 55 percent of patients without episodes of desaturation. The most frequent causes of oxygen desaturation included intracranial hypertension (44 percent), hypocarbia to a pa_{CO_2} of less than 28 mmHg (27 percent), systemic hypotension (10 percent), and hypoxia (8 percent).

TRANSCRANIAL DOPPLER AND TRAUMATIC VASOSPASM

Traumatic arterial spasm has been variably defined as a middle cerebral artery flow greater than 100 or 120 cm/sec or middle cerebral artery/internal carotid artery ratio (Lindgaard ratio) or greater than 3. In patients with a GCS of 3 to 12, the incidence of traumatic vasospasm has ranged from 7 to 40 percent, with higher incidence in patients with severe head injury. Onset of increased flow velocity occurs between days 1 to 6 after injury, typically on days 2 or 3, with maximal velocities seen between days 5 to 13. The development of spasm has been correlated with CT evidence of subarachnoid blood in several studies. In a study by Martin and coworkers, all three patients with severe vasospasms (middle cerebral flow velocity > 200 cm/s) had subarachnoid blood on CT.

The incidence of cerebral infarction attributable to traumatic vasospasm appears to be relatively low. In four transcranial Doppler investigations, the incidence of spasm-related cerebral infarction ranged from 0 to 5 percent. Combining these four studies, 6 of 226 patients (2.7 percent) developed infarction; all 6 had sustained a severe head injury and had subarachnoid blood on CT. In four patients who developed infarction in a study by Chan and associates, all had episodes of CPP less than 60 mmHg requiring treatment. Martin and coworkers demonstrated a significant correlation between the highest middle cerebral artery flow velocity and the lowest hemispheric cerebral blood flow as measured by the xenon-133 method.

In the TCDB cohort of 753 patients a 39 percent incidence of CT-diagnosed subarachnoid hemorrhage was reported, which correlated with a twofold increase in mortality. It is likely that this increased risk of dying in patients with traumatic subarachnoid hemorrhage was at least partially attributable to vasospasm. Although most studies have demonstrated an association between the presence of traumatic subarachnoid hemorrhage and transcranial Doppler diagnosed vasospasm, the presence of spasm has not been shown to be an independent predictor of outcome. The favorable results associated with maintenance of an adequate CPP may be due in part to an amelioration of the hemodynamic effects of arterial narrowing.

Because transcranial Doppler is a noninvasive study that can be done at the patient's bedside, it is an attractive monitoring technique for the head-injured patient. Given that patients sustaining severe head injury with subarachnoid hemorrhage on CT are the most prone to develop clinically significant vasospasm, such patients are especially appropriate for frequent Doppler studies.

HEMODYNAMIC SUPPORT AND CPP MANAGEMENT

The first step in establishing adequate CPP is vascular volume expansion. If perfusion pressure remains insufficient after volume therapy and after treatment to reduce ICP, the use of induced hypertension or inotropic support is indicated. In patients with precipitous drops in blood pressure or perfusion pressure, urgent and simultaneous use of volume expansion and vasopressor therapy is often required. A search for the underlying cause of hemodynamic instability should also be undertaken.

VOLUME EXPANSION

To establish euvoolemia, administration of full maintenance intravenous fluids should be given at a rate of 1.5 ml/kg/h of half-normal saline with 5% dextrose and potassium supplementation. If serum sodium is less than 140 mmol/L, the use of normal saline (D5 0.9% NaCl with KCl 20 mEq/L) as the maintenance fluids is indicated to prevent the development of a hyposmolar state. If the hematocrit falls below 30 percent transfusion of packed red blood cells is generally indicated to optimize blood rheology and cerebral oxygen delivery, particularly in individuals with problematic ICP or marginal perfusion pressure.

If a central venous pressure line is being used, the central venous pressure should be maintained from 5 to 10 mmHg. If a pulmonary artery catheter is in place, the wedge pressure should be maintained between 10 and 14 mmHg. This range of wedge pressure is associated with maximal cardiac performance in most adults; raising wedge pressure above 14 mmHg typically will not improve cardiac or stroke volume indices but will increase the risk of pulmonary edema. The need for higher intracardiac pressure can be determined by administering further volume and plotting a Starling curve with serial determinations of cardiac output and wedge pressure.

If central venous pressure falls below 5 mmHg, additional fluids should be given as needed in the form of crystalloid or colloid. Volume expansion with either crystalloid or colloid preparations in the brain-injured patient is acceptable, with care being taken not to induce a hyposmolar state with hypo-osmolar fluids or to incite coagulopathy with large doses of hetastarch. When the CPP goal of 70 mmHg is not being met while the central venous pressure or the wedge pressure are in the desired range, additional volume expansion is indicated. Once central venous pressure is over 10 mmHg or wedge pressure is over 14 mmHg and perfusion pressure or blood pressure are still inadequate, induced hypertension or inotropic support generally is indicated.

PHARMACOLOGICAL THERAPY

Use of vasopressor or inotropic support should be dictated by hemodynamic parameters, most important of which are the cardiac output (or cardiac index) and the systemic vascular resistance. Renal function is also an important consideration, given that vasopressors at higher doses can result in extracranial end-organ ischemia. These vasopressors, dopamine (Intropin), norepinephrine (Levophed) and phenylephrine (Neo-Synephrine), and the inotropic agent, dobutamine (Dobutrex) are employed most commonly in the critical care setting. All are sympathomimetic amines with varying degrees of alpha and beta adrenergic activity. All should be administered with continuous arterial pressure monitoring and preferably with a pulmonary artery catheter in place.

Agent	Low	Medium	High
Dopamine	2.00	10.00	20.00
Norepinephrine	0.02	0.10	0.20
Phenylephrine	1.00	2.50	5.00
Dobutamine	5.00	10.00	10.00

TABLE 6-4 Recommended Dosage Range of Sympathomimetics (ug/kg/min)

CHOICE OF AGENT

For most young, previously healthy trauma patients, norepinephrine is an excellent first choice to achieve a significant increase in arterial blood pressure. Because it is a peripheral vasoconstrictor and an inotropic agent, both blood pressure and cardiac output usually are improved. Since it does not increase urine output like low- or moderate-dose dopamine, fluid replacement is typically simplified. The norepinephrine infusion generally should not exceed 0.20 mg/kg/min. If urine output falls below 0.5 mg/kg/h, or if a metabolic acidosis develops, renal doses of dopamine should be started and the hemodynamic indices reassessed when the cardiac index is less than 3.0 L/min/m². The cardiac inotropic agent dobutamine is an effective first agent in improving perfusion pressure. Some patients occasionally will require more than one agent to achieve adequate cerebral perfusion. However, in most cases after reassessing the hemodynamic profile, it is apparent that further volume expansion is indicated.

Following traumatic brain injury with acute intraparenchymal blood on CT, an upper limit for systolic blood pressure of 180 to 200 mmHg is prudent, given the risk of worsening such hemorrhage with excessive hypertension. Chronic hypertensive patients, however, are likely to tolerate- and in fact require- higher arterial pressure to maintain adequate cerebral perfusion.

COMPLICATIONS OF HEMODYNAMIC THERAPY

Complications associated with central venous access, including pneumothorax, arterial injury, or local hematoma formation, occur at a rate of 1 to 2 percent. With the use of strict sterile technique, catheter-related sepsis occurs in less than 1 percent of cases, but the risk does increase if lines are left in place over 72 to 96 h. In general, central venous lines and pulmonary artery catheters should be removed as soon as their clinical utility is exhausted. The benefits from intracardiac monitoring with a Swan-Ganz catheter probably

are negligible in patients who have pulmonary artery wedge or pulmonary artery diastolic pressures that correlate well with central venous pressure. After 48 to 72 h of monitoring such patients, the Swan-Ganz catheter often can be removed and hemodynamic therapy can be based on central venous pressure, systemic blood pressure, and other clinical parameters. With aggressive volume expansion, however, the risk of pulmonary edema is significant. Central venous or wedge pressures should be carefully maintained within reasonable physiological ranges as described earlier. Regular physical examinations and daily chest x-ray are mandatory, especially in elderly patients and in those with multiple injuries. Strict and frequent monitoring of total fluids intake and output is essential in maintaining a euvoletic state. Negative fluid balance should be corrected by replacement of the fluid deficit at frequent intervals.

With the use of high-dose vasopressors, the risk of extracranial end-organ ischemia is significant. Arterial blood gases should be checked at least twice daily and serum lactate determinations obtained if a metabolic acidosis develops. The upper dosage limits of pressor agents should be strictly followed. In patients receiving other potentially nephrotoxic drugs such as aminoglycoside antibiotic and mannitol, pressor therapy carries an even higher risk. Renal doses of dopamine are helpful in preserving kidney function in critically ill patients who require high dose vasopressor. Close monitoring of urine output, blood urea nitrogen, and creatinine also is important. If renal function does deteriorate, pharmacological therapy should be modified accordingly. Daily mannitol doses are ideally kept at 200 g per day or less, and significantly lower doses are recommended if renal insufficiency develops.

TREATMENT OF INTRACRANIAL HYPERTENSION

Intracranial hypertension is variably defined as an ICP of over 10 to 15 mmHg; an ICP of over 20 mmHg sustained for more than 5 min warrants treatment. In patients with temporal lobe or deep frontal lobe lesion where the risk of uncal herniation is greater, an ICP treatment threshold of 15 mmHg may be indicated. With progressive intracranial hypertension or increasing intensity of ICP treatment, the possibility of a new or a reaccumulating intracranial hematoma should be investigated by a repeat CT scan.

Treatment of intracranial hypertension should proceed in a stepwise manner. Routine preemptive measures exercised in all severe head injury patients include maintenance of normothermia, head elevation to 30 degrees, and mild hyperventilation to a p_{aCO_2} of approximately 35 mmHg. Prophylactic anticonvulsants should also be utilized for at least the first week after injury. When acute and sustained rises in ICP occur, rapid manual hyperventilation of the patient should be avoided because the p_{aCO_2} may drop dramatically, resulting in critical cerebral vasoconstriction and ischemia. Instead the maneuvers outlined below should be followed in an expedient and, if necessary, simultaneous manner.

In patients whose ICP is easily controlled over the first 24 to 48 h after injury, suspension of sedation and neuromuscular blocking agents can be done early to permit clinical evaluation. However, in patients with problematic ICP, little is to be gained by early reversal of such therapy. In fact, a significant spike in ICP may occur. This during period, neurologic assessment may necessarily be limited to evaluation of pupillary responses.

Medical Management of Intracranial hypertension

Preemptive Measure

- head elevation to 30°, neutral alignment
- mild hyperventilation ($p_{a_{CO_2}}$ 30-35 mmHg)
- maintenance of euvolemia
- maintenance of CPP 70 mmHg or higher
- maintenance of normothermia ($< 37.5^{\circ}C$)
- seizure prophylaxis (phenytoin)

Primary therapy

- Ventricular CSF drainage
- Sedation (narcotics, benzodiazepines)
- neuromuscular blockade

Secondary Therapy

- bolus mannitol administration
- elevation of cerebral perfusion pressure

Tertiary Therapy

- metabolic suppressive therapy with high-dose barbiturate or propofol

Figure 6-10. medical management of intracranial hypertension. (CSF = cerebrospinal fluid)

SEDATION AND NEUROMUSCULAR BLOCKING AGENTS

Sedation can be initiated with a 5 to 10 mg bolus of IV morphine, which may be repeated once. If it is effective in lowering ICP, a continuous morphine infusion starting at 5 mg/h, should be instituted. The rate can be increased up to 20 mg/h, as needed for agitation or excess motor activity. When intracranial hypertension persists despite narcotic sedation, and if the patient is agitated, has increased motor tone, is shivering or resisting the ventilator, neuromuscular blockade should be added to help control ICP. These agents are effective in maintaining relaxation and can be given as needed or by a continuous infusion. Agitation secondary to alcohol withdrawal also may contribute to intracranial hypertension and is suspected, a benzodiazepine such as lorazepam (Ativan) 2 to 5 mg IV every 6 h, can be administered empirically for 48 to 72 h. Alternatively, the shorter acting benzodiazepine, midazolam (versed) can be used as a continuous infusion.

VENTRICULAR DRAINAGE

When initial measures are inadequate to control ICP, ventricular drainage should be utilized. In patients who exhibit no signs of agitation or increased motor tone, ventricular drainage should be used as the initial treatment to reduce ICP. The ventriculostomy chamber is to be placed at 5 cm above the level of the lateral ventricle with the pressure monitored continuously. The ventriculostomy is open when ICP exceeds the treatment threshold. Continuous ventricular drainage is not recommended because significant ICP spikes may be missed when the ICP is monitored only intermittently. Additionally, catheter occlusion secondary to ventricular collapse around the catheter is more likely to occur when continuous drainage is utilized.

MANNITOL

Mannitol is an excellent volume expander, thereby, improving perfusion pressure. As an osmotic diuretic, it also removes extravascular water from the brain. Additionally, blood viscosity is reduced with mannitol administration, and this improves cerebral blood flow. Because boluses of 0.25 g/kg have been shown to be equally effective in lowering ICP as have large doses, it is recommended that lower doses be used initially. For most adults, a 25-g bolus is effective for lowering ICP and improving CPP and can be repeated as necessary. Serum osmolality should not be allowed to rise above 310 mOsm/kg. If total input and output are regularly matched, a hyperosmolar state will rarely develop. Given that excessive mannitol used can result in acute oliguric renal failure, total daily doses of mannitol ideally should not exceed 200g; in patients with renal insufficiency, lower daily doses are recommended.

PERFUSION PRESSURE MANIPULATION

When ICP remains elevated and CPP is 70 mmHg or less, despite the above maneuvers, an attempt to raise perfusion pressure further to 80 to 100 mmHg can be made before resorting to barbiturate therapy. As discussed earlier in this chapter, many patients may require a perfusion pressure considerably above 70 mmHg before adequate ICP control is achieved.

HIGH DOSES BARBITURATE THERAPY

The mechanism of ICP control with high-dose barbiturate is thought to be largely through a reduction in cerebral metabolism with a coupled decrease in cerebral blood flow and blood volume. The response rate (i.e. control of ICP) has ranged from 30 to 80 percent in five different series reported from 1978 to 1988. Of those patients who responded, mortality was 21 to 33 percent; in nonresponders, mortality ranged from 64 to 89 percent. In a prospective randomized study by Eisenberg and coworkers, only 30 percent of patients responded to high-dose pentobarbital therapy. However, barbiturate treatment conferred a 2:1 benefit over conventional therapy, and this advantage rose to a 4:1 benefit if patients with pre-randomization cardiac complications were excluded. These investigations indicate that high-dose barbiturate therapy for intractable intracranial hypertension is efficacious in some patients, also predicting which patients are likely to respond is not easy. Indications for initiating pentobarbital treatment have not been consistently defined. When all previously outlined measures fail to control ICP, barbiturate therapy can be effective in improving outcome; it should be instituted before irreversible brainstem injury has occurred. If

barbiturates are started too late, ICP may be controlled but the patient is likely to remain neurologically devastated. Reasonable indications to begin high-dose pentobarbital therapy include 30 min of ICP over 30 mmHg despite a CPP of 70 mmHg.

Given the high rate of complications with barbiturate therapy, extreme vigilance must be practised. The patients should be in a normovolemic state prior to onset of therapy and a pulmonary artery catheter inserted. Electroencephalography is essential for monitoring the depth of barbiturate therapy. It is desirable to maintain a burst suppression pattern of isoelectric intervals with bursts of activity at 8 to 12 Hz. However, if electroencephalography is not immediately available, instituting pentobarbital therapy should not be delayed. Pentobarbital administration should begin with a 10 mg/kg loading dose over 30 min followed by 5 mg/kg/h over the next 3 h. If systolic blood pressure drops, the loading dose infusion should be slowed. A maintenance infusion of 1 to 3 mg/kg/h generally will maintain burst suppression and control ICP. Serum pentobarbital levels should be followed; ICP control typically is achieved with serum levels of 30 to 50 mg/100ml. The lowest effective dose to control ICP should be utilized. An adverse hemodynamic response is most likely to occur in older patients and in those with preexisting cardiac instability. In such patients, initiating therapy with lower doses is prudent. Ideally, CPP should still be maintained above 70 mmHg. Loss of pupillary reactivity is common with high-dose pentobarbital therapy and should not be interpreted as treatment failure with irreversible loss of brainstem function. Gradual withdrawal of barbiturate therapy over several days can begin after ICP control has been achieved for 24 to 48 h. Patients in barbiturate coma are at increased risk for pneumonia and sepsis and should be carefully monitored for such infections.

TEMPERATURE REGULATION

Hyperthermia has been associated with worse outcome following severe head injury. Conversely, the use of mild systemic hypothermia to 30 to 34°C in the treatment of severe head injury has yielded promising results in five preliminary clinical studies, and a randomized multicenter study is underway. Therefore, maintenance of systemic normothermia (core temperature less than 37.5°C appears warranted in moderate and severely head injured patients. A regimen utilizing a combination of acetaminophen, cooling blankets and ice-water lavage is generally effective. If shivering is noted, a neuromuscular blocking agent should be added. Given that brain temperatures are consistently higher than body temperatures, optimal temperature management in head-injured patients may best be served by monitoring brain temperature directly.

STEROID

Failure of high-dose corticosteroid therapy to control ICP or improve outcome in patients with traumatic brain injury has been shown in numerous clinical trials. Additionally, associated complications, including blunted immune response, making of infection, gastrointestinal hemorrhage, poor wound healing and elevation in serum glucose levels, make high-dose glucocorticoids unsuitable for the head-injured patient.