limited to non-benzodiazepine agents such as zolpidem. Visual and auditory symptoms should be evaluated by appropriate medical specialists.

The patient is able to return to play or work after at least 24 hours of recovery and when cleared to do so by a neurologist or clinical practitioner experienced in the management of concussion. In many states statutes specify these requirements. In general, the patient is able to return to play when symptoms no longer require treatment. At this point, many practitioners will subject the patient to provocative testing such as performing exertion (e.g., running) followed by cognitive testing. If this does not cause symptoms to recur and the patient performs well cognitively, then he or she is allowed to return to full activity.

For moderate to severe TBI the initial care goals are the "ABCs" of airway, breathing, and circulation. Next is "D" for disability (neurologic). Every patient should undergo a detailed neurologic examination to ascertain the level of neurologic disability. An initial Glasgow Coma Score (GCS) should be assigned to each patient. The GCS (Table 117-2) categorizes patients with TBI and provides a quantifiable measure of impairment.

Patients with severe TBI are those who present with GCS scores of eight or less. To optimize outcome, medical management should adhere to currently accepted clinical guidelines such as the Brain Trauma Foundation "Clinical Guidelines for Severe TBI." An important early intervention is airway protection, usually by endotracheal intubation. If elevated intracranial pressure (ICP) is suspected, elevate the patient's head to 30° and keep it midline, ideally with a rigid neck collar (at least until the cervical spine can be evaluated for stability). Mannitol should be given intravenously at a dose of 0.5-1.0 gm/kg. Hyperventilation may also be used with a goal of pCO<sub>2</sub> 34-36 mm Hg. ICP should be kept less than 20 mm Hg with the cerebral perfusion pressure (CPP) greater than 60 mm Hg. A head CT without contrast

## TABLE 117-2 AMERICAN ACADEMY OF NEUROLOGY CONCUSSION MANAGEMENT

## **GRADE 2 GRADE 1 (MILD)** (MODERATE) **GRADE 3 (SEVERE)** Remove from duty/ Remove from duty for Take to emergency work/play the rest of the day department Examine immediately Examine frequently for Neurologic evaluation, and at 5-minute signs of CNS including appropriate intervals deterioration neuroimaging Consider hospital May return to duty/ Physician's neuro exam work/play if clear ASAP (within 24 admission within 15 minutes Return to duty/work/ play after 1 full asymptomatic week (after being cleared by

physician)	
GRADE OF CONCUSSION	RETURN TO PLAY/WORK
Grade 1 (first)	15 minutes
Grade 1 (second injury)	1 week
Grade 2 (first)	1 week
Grade 2 (second injury)	2 weeks
Grade 3 (first) (brief LOC)	1 week
Grade 3 (first) (long LOC)	2 weeks
Grade 3 (second injury)	1 month
Grade 3 (third injury)	Consult a neurologist

should be done as soon as possible to identify lesions that will require surgery and to determine the extent of injury.

If ICP remains poorly controlled, one can consider administering an intravenous bolus of 23% hypertonic saline (50 mL) followed by continuous infusion of 2% or 3% hypertonic saline (75-125 mL/hr) through a central venous catheter. If these interventions are unsuccessful, pharmacological coma or surgical decompression should be considered. Pharmacological coma can be induced with pentobarbital. This is given as a loading dose of 5 mg/kg intravenously, followed by an infusion of 1-3 mg/kg/hr. Alternatively, propofol can be used, which is administered as a loading dose of 2mg/kg intravenously, followed by an infusion up to 200  $\mu$ g/min. Continuous EEG monitoring is helpful as the limit of drug-induced coma is achieving ICP control or cerebral electrical burst-suppression. Persistently elevated ICP after all these efforts is ominous. Consideration should be given to frontal or temporal lobe decompression and hemicraniectomy.

To meet CPP goals, patients must first be adequately hydrated. The goal of TBI fluid management is to increase the osmolar gradient between systemic vasculature and brain. For this purpose, hyperosmolar intravenous solutions are used, such as normal saline. Other options are hypertonic saline (e.g., 3% sodium solutions). If meeting CPP goals is difficult with intravenous fluids alone, vasoactive pharmacologic agents such as norepinephrine and phenylephrine can be administered. These two agents are preferred because they are considered to have the least effect on cerebral vasomotor tone. Barbiturates and propofol are myocardial depressants, therefore aggressive cardiovascular management will probably be necessary when pharmacological coma is induced.

Agitation can be treated with lorazepam or haloperidol. If inadequate, then infusions of midazolam or propofol may be used. Pain should be controlled: acetaminophen and nonsteroidal anti-inflammatory agents are adequate for mild discomfort; however, for moderate to severe pain, narcotic analgesics such as fentanyl or morphine should be used. A benefit of opioids is that they can be reversed by naloxone to allow reassessment of neurologic status.

Hypoxia, seizures, and fever must be avoided. Maintaining  $pO_2$  at approximately 100 mm Hg is sufficient. Phenytoin is administered for the first 7 days after injury because it will reduce early onset seizures. After 7 days it should be stopped. It can be restarted if seizures recur. Fever should be reduced with antipyretics such as acetaminophen, using a cooling blanket if needed. Other important management considerations include prevention of gastric stress ulcer, deep vein thrombosis (DVT), and decubitus ulcers. Feeding should be instituted as soon as practical to maintain nutrition.

After the initial few hours, efforts should be made to reduce hyperventilation, which is indicated only for initial emergency management. After 12 hours, metabolic compensation negates the ameliorative effects of respiratory alkalosis caused by the hypocapnic state induced by hyperventilation.

Repeated neurologic examination and continuous ICP and CPP measurement are indicated. Generally, the peak period of cerebral edema is from 48 to 96 hours after TBI. Thereafter, the edema resolves spontaneously and clinical improvement should follow.