solutions) or crystalloid (e.g., normal saline) may be used. Central venous pressure (CVP) should be maintained at 4-6 mm Hg. Hypervolemia should be avoided because it will exacerbate peripheral edema. Once an adequate circulating volume has been achieved, vasopressive agents can be used, (e.g., phenylephrine, norepinephine, or dopamine). The mean arterial pressure (MAP) should be 85 mm Hg or greater. Symptomatic bradycardia can be treated with atropine.

Patients with TSCI are at risk for ventilatory compromise. Patients whose injuries are at C5 or higher typically require mechanical ventilation with an appropriate tidal volume (6-10 mL/kg), FiO_2 and mandatory machine driven rate. The FiO_2 inspired oxygen concentration should give a pO_2 between 80-100 mm Hg. The rate should be set to give a pCO_2 of 40 mm Hg. Positive end-expiratory pressure (PEEP) should also be used to minimize atelectasis. If the patient does not show signs of ventilatory recovery within 2 weeks of intubation, a tracheostomy should be considered. Lesions below C5 may also be associated with inadequate spontaneous ventilation. Mid-cervical lesions may be associated with intact but compromised diaphragm function. If suspected, a "sniff" test under fluoroscopy can be performed to determine if both hemidiaphragms are functioning properly. If not, intubation/tracheostomy with volume-controlled ventilation may be needed. If intact, then pressure support (PS) ventilation sufficient to maintain an appropriate tidal volume with oxygenation and PEEP should be set as described above.

Patients with cervical lesions at C6 and below, including the thoracic cord, do not require mechanical ventilation. However, their ventilatory effort may be inadequate because the thoracic cord innervates intercostal muscles, which are accessory muscles of respiration. Such patients have decreased cough and inability to increase ventilation when needed, leading to atelectasis and inability to clear secretions, which can cause pneumonia. Such patients need assistance with clearing their airway: chest percussion, suctioning, and encouragement in coughing.

Thromboembolic disease is a leading cause of morbidity and mortality in patients with TSCI: up to 80% will develop DVT without prophylaxis. All patients with TSCI should receive both anticoagulation and have mechanical compression devices applied to their legs. Sequential compression devices (SCD) or compression stockings should be placed as soon as possible. When hemostasis is assured, low molecular weight heparin (LMWH) should be initiated. Unfractionated heparin may also be used in conjunction with SCD but LMWH is preferred. An inferior vena cava filter may be placed in those patients in whom anticoagulation therapy is contraindicated, but it should not be the primary means of preventing DVT.

Mid-low thoracic spinal cord injury can lead to ileus. A nasogastric tube should be placed to decompress the stomach. Parental nutrition should be started as soon as possible. Enteral feeding should be delayed until gastrointestinal motility returns, usually between 2 to 3 weeks. Pharmacologic agents that promote motility are metoclopromide, erythromycin, and cisapride. Gastric ulcer should be prevented with medication: H2 receptor antagonists, proton pump inhibitors, antacids, or sucralfate. Pancreatitis and trauma-related bowel perforation occur: loss of abdominal muscle tone and visceral sensation may mask usual clinical findings of pain, guarding, or rigidity.

Bladder tone may be lost due to spinal shock. A Foley catheter should be placed and maintained for a minimum of 5 to 7 days to drain the bladder and to evaluate circulatory volume and renal status. Once spinal shock resolves, autonomic dysreflexia may occur from bladder distention: skin flushing and hypertension. Clinical examination by palpation and percussion will reveal a distended bladder, which can be treated with intermittent catheterization or bladder training. Phenoxybenzamine may be helpful in this condition.

Nutrition should be given. Until enteral feeding can begin, parenteral nutrition should be used. A caloric level of 80% of the Harris-Benedict prediction should be used for quadriplegic patients. The full Harris-Benedict predicted amount should be used for patients with thoracic spine injuries and below. Skin care is essential to prevent decubitus ulcers. Mechanical kinetic beds, regular log rolling (every 2 hours), and padded orthocics are all useful in minimize this complication.

Orthotics, physical therapy, and occupational therapy (for cervical cord injury) are useful. Therapy should begin as soon as the spine is stabilized with the goal of minimizing contractures and beginning the rehabilitation. Once therapy begins, energy expenditure will increase requiring additional nutrition. If intermittent compression devices need to be removed during therapy, heparin dose may need to be increased.

PROGNOSIS

Tramatic Brain Injury (TBI)

The most useful prognostic indicator following TBI is the neurologic examination at presentation. Clearly, the better the neurologic examination, the higher the likelihood of improved recovery. The initial GCS score is a very reliable prognostic indicator. The lower the initial GCS score, the less likely a patient will have meaningful neurologic or functional recovery.

Tramatic Spinal Cord Injury (TSCI)

For TSCI, the completeness of the injury is the most useful prognosticator. The American Spine Injury Association Impairment Scale grades spinal cord injury on the basis of completeness (Table 117-3). A grade "A" or complete motor and sensory deficit below the lesion is the most ominous prognosis. If such a lesion persists longer than 24 hours, there is little reasonable likelihood of meaningful recovery. On the other hand, partial injuries, even severe, have substantial probability of recovery.

FUTURE

TBI and TSCI are serious neurologic conditions with significant implications on society. Prevention remains the most effective way of reducing the incidences of these diseases. Introduction of practice guidelines have contributed to improved outcome from TBI and TSCI. Sadly, morbidity remains a serious problem. Medical management is largely confined to supportive efforts primarily directed towards minimizing secondary injury, optimizing perfusion and oxygenation, and preventing nonneurologic morbidity. Surgical intervention helps restore structural stability, minimize further injury, and reduce the lesion. However