A complication of TBI is post-concussive syndrome (PCS). Diagnosis can be made using the Post-Concussion Symptom Scale (PCSS) and Graded Symptom Checklist (GSC). The most common symptoms of PCS are headache, difficulty concentrating, appetite changes, sleep abnormalities, and irritability. PCS has a variable presentation and duration depending on the patient and the severity of TBI. In general, PCS lasts for a few weeks post-injury. However, uncommonly, it can persist beyond a year or more. Treatment is symptomatic. For headache, nonsteroidal anti-inflammatory agents, migraine drugs, and biofeedback can be effective. For cognitive dysfunction, neuropsychological testing may be helpful in determining appropriate intervention, which may include cognitive behavior therapy.

Traumatic Spinal Cord Injury (TSCI)

The emergency management of traumatic injury to the spinal cord has greatly improved with adherence to the American Association of Neurological Surgeons "Guidelines for the Management of Cervical Spine and Spinal Cord Injuries." Therapy begins with the "ABC" of airway, breathing, and circulation. A secure airway is absolutely vital. For patients suffering from high cervical lesions, spontaneous ventilation will be lost. Lesions below C5 may also impair ventilatory capability. If the airway or ventilatory efforts are compromised, emergency intubation is required. For a patient in whom cervical spine trauma has not been assessed, the preferred method is nasotracheal intubation using fiberoptic guidance. Other approaches are nasotracheal (blind) or orotracheal intubation, provided that cervical spine alignment is maintained by traction.

Maintaining an intravasacular volume is essential in TSCI. Hypotension may be due to either neurogenic shock or hypovolemia. For neurogenic shock, vasopressive pharmacologic agents, such as phenylephrine, may be needed. If tachycardia is present, then hypovolemia is the more likely etiology and fluid resuscitation with normal saline is the appropriate initial management.

After addressing the "ABC's," a neurologic history and examination should be obtained. An accompanying TBI needs be considered. Up to 50% of TSCI patients have an associated TBI. Neuroimaging is often indicated, but not all patients need radiographic study. A normal neurologic assessment obviates the need for imaging studies; however, a complaint of burning hands or of pain over the spine, numbness, tingling, or weakness indicates possible spinal cord injury. The time of injury should be recorded as accurately as possible. A detailed neurologic examination is needed to identify the level of the injury, the completeness of any deficits, and to document the degree of neurologic dysfunction at the earliest time possible. The level of the injury is the lowest spinal cord segment with intact motor and sensory function. The prognosis for neurologic improvement is better if the lesion is incomplete than complete. Following the acute injury, serial examinations must be made frequently.

If spinal cord injury is suspected, the patient should be immediately and appropriately immobilized with a rigid collar or back board, or both. Radiologic evaluation should begin with plain x-rays of the bony spine. Abnormalities on x-rays should lead to further neuroimaging. Bony vertebrae should be examined with CT and the spinal cord with MRI. Intervertebral and paravertebral soft tissue are best studied with MRI. A chest x-ray should also be obtained in order to visualize the lower cervical and thoracic vertebrae. Presence of a pleural effusion in the setting of a possible thoracic spine injury suggests a hemothorax.

If the C-spine x-rays are normal but the patient complains of neck pain, then ligamentous injury may be present. Ligamentous injury is evaluated by flexion-extension C-spine x-rays. However, in the acute period, pain may prevent an adequate study. These patients should be kept in a rigid cervical collar for a few days until the pain and neck muscle spasm resolves. At that time, imaging may be performed. If abnormal, the patient will need surgical evaluation.

The use of methylprednisolone for TSCI is no longer advocated.

Spinal Cord Syndromes

There are three main spinal cord syndromes: Anterior cord, Brown-Sequard, and Central cord. Anterior cord syndrome is associated with deficits referable to bilateral anterior and lateral spinal cord columns. There is loss of touch sensation, pain, temperature, and motor function below the level of the lesion. The posterior column functions of proprioception and vibratory sensation remain intact. In Brown-Sequard syndrome, the deficits are due to injury to a lateral half of the cord. There is functional loss of ipsilateral motor, touch, proprioception and vibration, and contralateral pain and temperature. Central cord results in a "man in a barrel" syndrome: motor paralysis of both upper extremities with sparing of the lower extremities. Weakness is greater proximally than distally. Pain and temperature sensations are generally reduced, but proprioception and vibration are spared.

Spinal Shock

Spinal shock may occur after acute injury causing a temporary loss of spinal reflexes below the level of injury. Neurologic examination will reveal loss of muscle stretch reflexes, bulbocavernosus reflex, and the anal wink. In high cervical injuries, the lower reflexes (bulbocavernosus and anal wink) may be preserved. There may also be the "Schiff-Sherrington" phenomenon, in which reflexes are affected above the level of injury. Additionally, there may be loss of autonomic reflexes leading to neurogenic shock and ileus and urinary retention.

Acute and Subacute Management

In the intensive care unit, the patient will need continued treatment. Once methylprednisolone therapy has completed, there is no need for further steroid use. TSCI patients require close cardiovascular and respiratory monitoring. Other issues are genitourinary, bowel, infectious disease, nutrition, skin, and prophylaxis against ulcers and deep vein thrombosis formation.

Patients suffering from spinal cord injury are at risk for neurogenic shock and dysautonomia with resulting peripheral vasodilation and hypotension. Lesions at T3 or above compromise sympathetic tone with hypotension accompanied by bradycardia: the classic neurogenic shock triad of bradycardia, hypotension, and peripheral vasodilation.

Dysautonomia is treated by ensuring adequate circulating volume. The goal is to fluid resuscitate to a euvolemic state. Blood can be used if the patient is anemic (i.e., hematocrit less than 30%). If blood is not required, then either colloid (e.g., albumin