Clinical Manifestations

It is often difficult to determine the extent and severity of damage at first. Immediate loss of function is caused by both anatomic and impaired physiologic function, and improved function may not be evident for weeks or even months. Manifestation of the initial response to acute SCI is flaccid paralysis below the level of the damage. This stage is often referred to as **spinal shock syndrome** and is caused by the sudden disruption of central and autonomic pathways. Local effects of cord edema and ischemia produce a physiologic transection with or without an anatomic severance. Most children with an SCI experience some spinal shock. Manifestations include the absence of reflexes at or below the cord lesion, with flaccidity or limpness of the involved muscles, loss of sensation and motor function, and autonomic dysfunction (symptoms of hypotension, low or high body temperature, loss of bladder and bowel control, and autonomic dysreflexia).

Autonomic paralysis also affects thermoregulatory functions. Afferent impulses from temperature receptors in the skin are not integrated; therefore, the patient is subject to temperature increases or decreases in response to alterations in environmental temperature. Hyperthermia can result from excessive ambient temperature, such as too many covers.

Except in the situations previously mentioned, flaccid paralysis is replaced by spinal reflex activity and increasing spasticity or, in incomplete lesions, greater or lesser degree of neurologic recovery.

The paralytic nature of autonomic function is replaced by **autonomic dysreflexia**, especially when the lesions are above the mid-thoracic level. This autonomic phenomenon is caused by visceral distention or irritation, particularly of the bowel or bladder. Sensory impulses are triggered and travel to the cord lesion, where they are blocked, which causes activation of sympathetic reflex action with disturbed central inhibitory control. Excessive sympathetic activity is manifested by a flushing face, sweating forehead, pupillary constriction, marked hypertension, headache, and bradycardia. The precipitating stimulus may be merely a full bladder or rectum or other internal or external sensory input. It can be a catastrophic event unless the irritation is relieved.

Additional clinical findings of SCI may include numbness, tingling, or burning; priapism; weakness; and loss of bowel and