Mononeuropathies

Lumbosacral or brachial plexopathies may be secondary to direct trauma, usually from motor vehicle accidents or surgery. Insertion of catheters into the iliac arteries or aorta may dislodge thrombi, and the resulting embolization impairs vascular supply to nerves and, in this manner, induces focal ischemic plexopathy (60). Direct surgical trauma to vessels may also induce vascular insufficiency. Motorcycle accidents commonly traumatize the brachial plexus. Proximal lesions are suggested by Horner's syndrome, winging of the scapular, and diaphragm paralysis. Electrophysiologic studies ideally performed after 3 weeks will further help localize the lesion. Myelography, CT myelography, or MRI may provide more positive evidence of root avulsion, which would preclude attempts at operative nerve repair. Fractures of the pelvis may cause varying patterns of damage to the lumbosacral plexus. Observations of focal weakness on reflex or voluntarily induced movement, plus abnormalities of the deep tendon reflexes, may provide an initial clue to the presence of such damage. Thus, weakness of hip abduction and flexion, knee extension, and an absent ankle jerk suggest damage to L2-4 roots of the lumbosacral plexus. Electrophysiologic studies should successfully demonstrate abnormalities on motor and sensory NCS and needle EMG that localize the lesion to the brachial or lumbosacral plexus.

There are several types of mononeuropathies. If the primary reason for admission to the ICU was the postoperative state, the initial surgery may have induced a mononeuropathy when operating room equipment or perhaps the surgery itself directly damaged a peripheral nerve. For example, weakness of dorsiflexion of the wrist and digits and an absent brachioradialis reflex suggests radial nerve damage in the spinal groove of the humerus by fracture or direct compression. Phrenic nerves, either bilaterally or unilaterally, may be damaged at the time of surgery by direct trauma or by the application of cold, as occurs in the hypothermia associated with cardiac surgery (61). Distal nerves may be damaged as a result of impairment of nutrient blood supply and distal embolization, as for example in the occurrence of femoral or sciatic neuropathy after cardiac or vascular surgery. Electrophysiologic studies show a relatively pure axonopathy affecting motor and sensory fibers.

Patients who are being anticoagulated run the risk of hemorrhage because a sudden rise in tissue pressure produces a compartment syndrome and severe compression results in nerve ischemia. The compartments most commonly involved are the iliofascias and gluteal, producing acute femoral or sciatic neuropathies (62). Fractures and soft tissue trauma may also induce compartment syndromes. An immediate CT should be ordered, which will show the location of the hemorrhage. Then, surgical decompression may successfully decompress the nerve. The situation is so acute and emergent that electrophysiologic studies are of little value.

PATHOPHYSIOLOGY OF CRITICAL ILLNESS NEUROPATHY AND MYOPATHY

The microcirculation is disturbed in sepsis (Fig. 3). Blood vessels supplying the peripheral nerves lack autoregulation (63), rendering these vessels susceptible to such disturbances. Moreover, cytokines that are secreted in sepsis have histamine-like properties that may increase microvascular permeability (28). The resulting endoneurial edema induces hypoxia by an increase in intercapillary distance and other mechanisms. Severe energy deficits can induce a primarily distal axonal degeneration if highly energy-dependant systems involve axonal transport of structural proteins. The predominantly distal involvement may explain why recovery in some patients can be surprisingly short, conforming to the short length of nerve through which axonal regeneration takes place. It is possible that cytokines themselves may have a direct toxic effect on peripheral nerves. Tumor necrosis factor decreases the resting transmembrane potential of skeletal muscle fibers in vitro (64) and induces muscle proteolysis in animals (65). Hund et al. (25) used a bioassay method to extract a low-molecular-weight fraction of the serum of patients with CIP that proved to be toxic to rat spinal motor neurons.

Disturbances of the microcirculation to nerve and muscle may also explain the effects of neuromuscular blocking agents and corticosteroids. Through increased capillary permeability induced by sepsis, neuromuscular blocking agents, notably vecuronium or its metabolite, 3 desacetyl-vecuronium (30), could have a direct toxic effect on peripheral nerve axons. These neuromuscular blocking agents may also cause functional denervation through their prolonged neuromuscular blocking action (66). The result would be denervation atrophy of muscle and a relatively pure motor neuropathy.

We have always been concerned that antibiotics, particularly aminoglycosides, with their known neural toxicity, might cause CIP. These drugs might gain access to the peripheral nerve as a result of increased capillary permeability. However, there has been no statistical proof that antibiotics cause peripheral nerve dysfunction in sepsis (14). Nonetheless, this possibility should be explored by basic science experiments.

The schema shown in Fig. 4 explains the acute quadriplegic myopathy that develops when critically ill patients are treated with neuromuscular blocking agents and corticosteroids. Animal experiments by Karpati et al. (67) showed that if muscle was first denervated by nerve transection and then corticosteroids given, a thick filament myopathy similar to that myopathy seen in humans could
be induced. In humans, CIP and neuromuscular blocking agents combine to denervate muscle, and steroids in turn induce myopathic changes. The rapidly evolving myopathy reported recently by Al-Lozi et al. (68) is characterized by destruction of thick filaments throughout the muscle fibers and acute necrotizing myopathy (56,57) and may simply represent a further stage of this process. Two postulated mechanisms of protein degradation in critically ill patients prompted by corticosteroids include the activation of ubiquitin (49) and alterations of calpain with abnormal calcium homeostasis (53).

REFERENCES


