Peripheral neuropathy (PN) often is a side effect for patients with cancer treated with neurotoxic chemotherapeutic agents and is defined as inflammation, injury, or degeneration of the peripheral nerve fiber(s) (Wilkes, 1999). Although PN has received less attention than other symptoms and the actual incidence is unknown, researchers estimated that it occurs in 10%–20% of patients with cancer (Armstrong, Rust, & Kohtz, 1997). Having a pre-existing neuropathy or pre-existing condition known to cause neuropathy, such as HIV, diabetes mellitus, alcoholism, or vitamin deficiency (especially vitamin B), puts patients at risk for developing PN. Early symptoms of PN include tingling and numbness in the fingers and toes (stocking-glove distribution). As the condition worsens, patients may experience the loss of deep tendon reflexes, pain, impaired temperature sensation, diminished touch and position sense, and reduced muscle tone (Almadrones). The frequency of PN is increasing partly because of the wider use of cranial nerves. The nerves of the PNS peripherally to the CNS (afferent), and the motor nerve fibers transmit impulses from the CNS to the muscles or organs (effenter). The large fiber sensory nerves control vibration and position sense, and the small fiber sensory nerves control touch, pain, and temperature. Motor nerves control movement and maintain muscle tone. Symptoms are related directly to the nerve fibers that are affected. For example, large fiber damage is associated with vibration and position sense changes whereas small fiber damage is associated with pain and temperature sensation changes. Neurotoxic chemotherapeutic agents can cause injury to the sensory and motor axons and Schwann cells and, if involved, can breakdown the myelin sheath (Wilkes, 1999).

The autonomic nervous system (ANS) is part of the PNS and controls involuntary body functions, such as control of internal organs. Damage to the nerves of the ANS causes changes in the functioning of the bowel, bladder, or blood pressure, resulting in constipation, incontinence, or orthostatic hypotension (Almadrones). The cranial nerves transmit sensory and motor impulses to and from the CNS from the head and neck area. The most common cranial nerve damage caused by chemotherapy is ototoxicity. Other effects may include ocular toxicity (e.g., blurred vision, ptosis) or taste changes (Almadrones). Patients experience symptoms directly related to the portion of the PNS that is affected.

What factors place an individual at risk for developing PN? Known risk factors include having high-dose chemotherapy or cumulative doses of neurotoxic drugs; being over 60 years old; concurrently using neurotoxic drugs; having had previous radiotherapy to the spinal fields that resulted in neurologic damage; having a pre-existing neuropathy as a result of diabetes mellitus or infection with HIV or Charcot-Marie Tooth disease (a hereditary neuropathic joint disease); and being malnourished with a vitamin deficiency (e.g., B complex) (Boyle, 2000). IV chemotherapy administration or a combination of chemotherapy regimens that may increase synergistic effects (e.g., paclitaxel + carboplatin) also can create a PN risk (Boyle).

PN affects both sensory and motor nerve fibers. Early symptoms include tingling, numbness, and burning in the fingers and toes; symptoms usually are experienced bilaterally and are worse in the lower extremities. This is described as the sensation of wearing a sock or glove, referred to as “stocking-glove” distribution (see Figure 1). Later, symptoms may progress to pain and the loss of deep tendon reflexes, two-point.