Advances in cancer chemotherapy include several new, highly active agents that are potentially neurotoxic. Aggressive treatment with one or more of the agents has increased the likelihood of cure or long-term survival along with possible development of dose-limiting neurologic effects, particularly in the peripheral nervous system (PNS), which is more sensitive than the central nervous system (CNS) to neurotoxic effects of chemotherapy. This article will discuss chemotherapy-induced peripheral neuropathy (CIPN) in terms of incidence, PNS anatomy and physiology, pathogenesis of CIPN, and current and potential management strategies.

Several challenges regarding CIPN include incomplete understanding of pathogenesis, a lack of evidence-based therapies to alleviate it, and inconsistent assessment for CIPN by nurses and physicians who may view it as less important than other side effects of chemotherapy (Smith, Whedon, & Bookbinder, 2002). As a result, patients do not receive correct information about what to anticipate, how CIPN might progress, and to what degree it will resolve (Nail, 2001). This is particularly problematic because research is beginning to document that CIPN is among the most distressing symptoms experienced by patients undergoing chemotherapy with neurotoxic drugs and that it affects important activities and quality of life (QOL) (Boehmke & Dickerson, 2005). Furthermore, little data exist regarding the duration of post-therapy CIPN and which manifestations lessen or resolve.

**The Peripheral Nervous System**

A brief review of PNS structures and functions is useful to understand the pathophysiologic mechanisms of CIPN. The PNS and CNS transmit, integrate, interpret, and respond to information from the external and internal environments. The CNS (brain and spinal cord) is protected by the blood-brain vascular barrier that inhibits diffusion of large molecules, highly charged ions, and many drugs from the bloodstream into CNS tissues (Willis, 2000). A similar vascular barrier does not protect the...