The management of chemotherapy-induced nausea and vomiting (CINV) has improved significantly with the use of selective serotonin receptor antagonists, which are effective for managing acute nausea (Hesketh, 2008); the more recent addition of neurokinin-1-receptor (NK1) antagonists, which are more effective for the treatment of delayed nausea (Dando & Perry, 2004; Sanger & Andrews, 2006); and the publication of evidence-based standards of practice (Kris et al., 2006). Even with these combination drugs and the use of the American Society of Clinical Oncology’s guidelines (Kris et al., 2006), uncontrolled nausea still is reported in 36% (Waqar et al., 2008) to 59% (Cohen, de Moor, Eisenberg, Ming, & Hu, 2007) of treated patients and in 75% of patients not receiving prophylactic NK1 antagonist (aprepitant) therapy (Erazo Valle, Wisniewski, Figueroa Vadillo, Burke, & Martinez Corona, 2006).

The mechanisms for CINV are not fully understood, although some theories focus on the relationship between the small intestine’s endocrine and enterochromaffin cells, which release serotonin (5-hydroxytryptamin [5-HT]) in response to chemotherapy-related damage to the duodenal mucosa (Saito, Takano, & Kamiya, 2003). The serotonin released from the duodenum binds to vagal afferent 5-HT3 receptors, which then transmit the afferent impulses to the emetic center in the brain (Hogan & Grant, 1997). This process is responsible for early nausea and vomiting during the first 24 hours of drug administration. Delayed nausea and vomiting result from more complex responses and the combined effect of serotonin release and disrupted gut motility (Carelle et al., 2002). Cellular damage and breakdown in the stomach and small intestine also contribute to delayed nausea and vomiting (Baker, Morzorati, & Ellett, 2005).

Absorbed toxic materials circulating in the blood, including those associated with chemotherapeutic agents, also can act directly on the area postrema of the brain where the blood-brain barrier is relatively permeable.