Review and recommendations for the prevention, management, and treatment of postoperative and postdischarge nausea and vomiting

Danielle Cruthirds, PhD,[a] Pamela J. Sims, PharmD, PhD,[a] and Patrick J. Louis, DDS, MD[b]
Samford University and University of Alabama, Birmingham, Alabama

Patients have rated severe nausea to be worse than postoperative pain. The overall incidence of postoperative nausea and vomiting (PONV) is 25%-30% and can lead to delayed discharge and unanticipated hospital admission. After outpatient surgery, the overall incidence of postdischarge nausea has been reported to be 17% and of vomiting 8%, higher than nausea and vomiting reported during the procedure or recovery. Patients who experienced postdischarge nausea and vomiting (PDNV) were unable to resume normal daily activities as quickly. This paper addresses the frequency, pathophysiology and patient perception of PONV and PDNV and reviews antiemetics and adjunctive medications used for the prevention, management, and treatment of PONV and PDNV. For each, the indication, mechanism of action, adverse effects, drug interactions, and implications for oral surgery and outpatient sedation are provided. Because many antiemetics are available for prevention, management, and treatment of PONV and PDNV, optimal medication choices are important for each procedure and patient. (Oral Surg Oral Med Oral Pathol Oral Radiol 2013;115:601-611)

Patients have rated severe nausea to be worse than postoperative pain.[1] The overall incidence of postoperative nausea and vomiting (PONV) has been reported to be 25%-30% and can lead to delayed discharge and unanticipated hospital admission.[2] After outpatient surgical procedures, the overall incidence of postdischarge nausea has been reported to be 17% (range 0%-55%) and of postdischarge vomiting 8% (0%-16%), which are higher than nausea and vomiting reported during the procedure or recovery in the office.[1,3,4] Patients who experienced postdischarge nausea and vomiting (PDNV) were not able to resume their normal daily activities as quickly as those who did not.[4] The present paper examines the frequency of PONV and PDNV, the pathophysiology of nausea and vomiting, and how PONV and PDNV are perceived by patients, reviews antiemetics and adjunctive medications, and discusses current therapies in the prevention, management, and treatment of PONV and PDNV.

PATHOPHYSIOLOGY OF POSTOPERATIVE NAUSEA AND VOMITING

A 20%-30% incidence of PONV can be expected with the use of intravenous sedation and general anesthesia for oral and maxillofacial surgery procedures.[5-7] By definition, nausea is described as a subjectively unpleasant sensation associated with awareness of the urge to vomit, and vomiting is described as the forceful expulsion of upper gastrointestinal contents via the mouth brought about by the powerful sustained contraction of the abdominal muscles.[8] Both nausea and vomiting are responses to certain stimuli. These stimuli can include olfactory, visual, vestibular, and psychogenic sources. Factors that trigger PONV include stimuli before, during, and after the operative procedure.[9]

The mechanisms leading to nausea are for the most part unclear. They are thought to be the result of the disruption of the normal contraction-relaxation pattern of the stomach caused by a response to stimuli in the higher brain centers.[10,11] Associated changes include gastrointestinal (GI) motility, GI relaxation, duodenal retroperistalsis, decreased gastric acid secretion, increased salivation, pallor, tachycardia, feelings of hot and cold, and diaphoresis.[10,11]

The vomiting reflex appears to originate in the emetic or vomiting center located in the medulla. There are multiple sensory inputs involved in this reflex,

Statement of Clinical Relevance

Antiemetics have important and potentially serious effects when used in combination with local anesthesia, conscious sedation, or other postoperative medications. This comprehensive review seeks to improve clinicians' knowledge regarding postoperative and postdischarge nausea and vomiting to inform and guide treatment.

[a]Department of Pharmaceutical, Social, and Administrative Sciences, McWhorter School of Pharmacy, Samford University.
[b]Department of Oral and Maxillofacial Surgery, University of Alabama.
Received for publication Apr 23, 2012; returned for revision Aug 26, 2012; accepted for publication Sep 18, 2012.

2212-4403/$ - see front matter
http://dx.doi.org/10.1016/j.oooo.2012.09.088

including the vagus nerve (cranial nerve X), the vestibular nerve (cranial nerve VIII), the limbic system, and the chemoreceptor trigger zone (CRTZ).[9] The vagus nerve relays sensory input, primarily thru serotonin, from the mechanoreceptors and chemoreceptors in the GI tract, respiratory tract, and cardiovascular system. The vestibular nerve relays input from the auditory labyrinth, with the primary neurotransmitters being histamine and acetylcholine. The limbic system appears to play a role in the learned response of anticipatory nausea and vomiting.[12] The CRTZ is exposed to blood and cerebrospinal fluid and, therefore, can react to substances in the blood. Several neurotransmitters and neuromodulators trigger this area, including serotonin, dopamine, histamine, acetylcholine, substance P, and adrenaline.[13] The CRTZ identifies harmful substances and relays this to the vomiting center (VC). The VC then activates the efferent motor pathways initiating the vomiting response.[10,14] Metabolic factors that are mediated by the vomiting center and CRTZ include uremia, diabetes mellitus (hypo- or hyperglycemia), electrolyte disturbances (sodium, potassium), hormonal imbalances (estrogen, progesterone), and pregnancy. Sensory stimulation includes tactile stimulation of the posterior pharynx as well as stretching, inflammation, or injury to the airway. The vomiting cascade is complex and includes multiple steps. The cascade continues until the stimulus is no longer present.[10]

NAUSEA AND VOMITING AFTER OFFICE-BASED ANESTHESIA

In recent years, office-based anesthesia has been used more frequently as well as being applicable to more complex cases. Complications associated with office-based anesthesia include vomiting during induction (0.1%) or in the recovery room (0.3%), laryngospasm or bronchospasm (0.3%), cardiac arrhythmias (0.1%), syncope (0.1%), prolonged recovery (0.2%), and peripheral vascular injury (0.1%).[16]

Although the rates of these complications are low, patients rate severe nausea as worse than postoperative pain.[1] This specific complication can delay discharge and is one of the leading causes of unexpected hospital admission after planned ambulatory surgery.[17] A 2002 review showed an incidence of 17% for nausea (range 0%-55%) and 8% for vomiting (range 0%-16%) after outpatient surgery.[15] Of those that did not experience PONV while in the hospital, almost one-half experienced it on day 1-3 after procedure. This further complicates the management of PONV because its occurrence is usually not witnessed by the care team.[15] Many surgeons continue to view PONV as a minor complication that poses little threat to the patient.[18] In contrast, patients view this complication as being more debilitating than the operation itself.[15]

ANTIEMETICS IN ORAL AND MAXILLOFACIAL SURGERY

Activation of the VC or the sensation of nausea may result from the stimulation of the CRTZ, the vestibular apparatus, visceral afferent inputs, and cortical inputs. At least 3 nerves and 7 neurotransmitters are involved, making prophylaxis and treatment complex.[19] An important stimulus for PONV in oral or maxillofacial surgery is the effect of swallowed blood in the stomach.[19] Blood in the stomach is one of the strongest peripheral-acting emetogenic stimuli and is difficult to treat by antiemetic medication alone.[20,21] Perioperative suctioning of the oral cavity to remove secretions and blood before the patient swallows is important. Two procedures commonly performed by Oral and Maxillofacial Surgeons during a surgery conducted under general anesthesia that help decrease blood from the oral cavity entering the stomach include placement of an oropharyngeal pack and placement of a nasogastric tube to suction the stomach contents at the completion of surgery. Accumulation of blood in the stomach often results in nausea and vomiting and must be removed to obtain complete relief.[20,21]

There are currently 2 schools of thought regarding the management of PONV: prophylactic antiemetic treatment and symptomatic treatment.[18] Complications such as aspiration of vomitus and asphyxia have often been cited to justify use of prophylaxis. In general, universal prophylaxis is not warranted.[22,23] Nonpharmacologic strategies to reduce the baseline risk should first be considered.[19] Reducing or avoiding opioids in patients that are at high risk for nausea and vomiting is an important strategy.[19] Opioids can increase the risk of PONV through several mechanisms: direct stimulation of the area postrema, decrease in GI motility with prolongation of gastric emptying time, and sensitization of the otic and vestibular areas to motion.[2,9] Patient movement after surgery with stimulation of endolymph in the inner ear appears to increase the frequency of opioid-induced emesis.[2] Patients receiving very large doses of opioids during general anesthesia have a lower incidence of PONV compared with patients undergoing outpatient surgery who receive significantly lower doses of opioids.[24] More frequent changes in body position that occur in the ambulatory patient increase the frequency of opioid-induced emesis.[2,24] PONV in outpatients often occurs after movement from chair to standing, after ambulation, or during the car ride home.[25] These dose-related effects may last for up to 6 hours after opioid administration.[25] Nitrous oxide (N_2O) can cause PONV by direct central nervous sys-