

Current Controversies in PONV

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The overall incidence of PONV has not changed over the years, and it is still a problem. PONV causes increased intraocular and intracranial pressures and increased blood pressure and heart rate. Serious medical consequences do occur although rarely, and they include wound dehiscence, bleeding, dehydration and electrolyte imbalance, and aspiration pneumonitis can also occur. PONV also generates increased cost, from increased use of personnel, supplies, and drugs, as well as unplanned admissions. The most important medical consequences of PONV are patient discomfort (mild to severe) and dissatisfaction. Patients can attach a monetary value to avoiding PONV; they would pay more to avoid vomiting than recall without pain (*Macario A et al. Anesth Analg. 1999;89:652-658*).

Anatomy of Emesis: Activating stimuli for PONV include vagal nerve afferents from the viscera (mucosa and muscle), ear and heart as well as cranial nerve input from VIII and IX. Chemical PONV triggers are sensed at the chemoreceptor trigger zone (CTZ), in the area postrema on the floor of the fourth ventricle. It is outside the blood-brain barrier and has many types of drug receptors, including opioid, dopamine, muscarinic cholinergic, histamine and serotonin (*Andrews PLR. Br J Anaesth 1992;69 (Suppl 1)25-19S.*) It is important to remember that PONV can be triggered via all of these pathways. Coordination of the vomiting reflex occurs through the vomiting center, located in the medulla at the lateral reticular formation and tractus solitarius. Efferents for vomiting includes multiple cranial nerves (V, VII, IX, X, XII), spinal nerves (diaphragm, abdominal muscles), and sympathetic and parasympathetic motor responses.

The etiologies of PONV encompass the entire anesthetic and surgical process. There are patient-related factors, surgery-related factors, anesthetic agent factors, anesthetic process factors and postoperative factors (*Philip BK. Pharmacol Ther. 1997;22:18S-25S*). Among the patient-related factors, several appear to produce independent risk for PONV. They are: younger age, female gender, history of PONV (or motion sickness), and non-smoker. Other factors that are commonly believed but not supported in the literature are anxiety and obesity. Disease states associated with decreased gastric motility or emptying are additional risk for emesis. Type of surgery is associated with differential PONV risk. Gynecologic surgery is associated with 40%-77% incidence of PONV and hernia repair/orchiopexy with a 54%-58% incidence. Other high-risk procedures are strabismus repair, breast procedures, and otolaryngologic surgery.

We may be more familiar with some of the anesthesia agent factors. Benzodiazepines decrease PONV, via sedation. Volatile agents cause more PONV than propofol infusion, for ~2 hours postop. Propofol reduces PONV but does not eliminate it, and the protection is dose related and therefore relatively brief (*Tramer M et al. Brit J Anaesth*

1997;78:247-55). Neostigmine increases PONV with a dose response > 2.5 mg, and atropine is more protective than glycopyrrolate. N₂O increases vomiting; this effect is significant only when the baseline incidence is high. (Tramèr M. *Acta Anaesth Scand* 2001;45:4-13, Salmenperä M et al. *Acta Anaesth Scand* 1992;36:445-8, Tramèr M et al. *Br J Anaesth* 1996;76:186-93.)

Opioids stimulate CTZ via multiple mechanisms. Basing the analgesic regimen on NSAIDs reduces opioid need, improves pain relief and decreases the incidence and severity of PONV. There is a dose response between opioids and PONV, so judicious amounts should be given. Giving a higher opioid dose may also lower the effectiveness of antiemetic drugs. (Polati E et al. *Anesth Analg* 1997;85:395-9).

Anesthesia process factors affect PONV. General anesthesia has a higher incidence than major regional than peripheral regional. The duration of anesthetic exposure {surgery duration} increases the incidence by 60% each 30 minutes. Experience of anesthesia provider is also a factor.

Risk factors for PONV in children are similar to adults with the following differences. Pediatric studies often limited to vomiting only, and the vomiting incidence is 2x in children vs. adults. Risk increases with age, then decrease in the adult years. Gender differences are not seen before puberty. In children as well, the PONV risk is procedure specific. (Gan TJ et al. *Anesth Analg* 2007; 105:1615-28).

Postoperative factors affect PONV. These include uncontrolled pain (especially visceral/pelvic pain), opioid administration, and early ambulation and movement. Early oral intake can also be problematic, triggering symptoms when the patients start to move about. Dehydration is an important problem, and adequate IV fluid hydration can decrease PONV. Therefore it is important to have an appropriate duration of preop fast. Per the ASA, this interval is 2 hr for clear liquids (MN/8hr for solids).

There are several classes of antiemetic drugs that can be used for prophylaxis or treatment. Each drug has advantages and disadvantages. We give prophylaxis for higher risk patients and treat other patients if needed. An antiemetic protocol could include droperidol, dexamethasone and a serotonin antagonist. Other useful drugs are transdermal scopolamine, intramuscular ephedrine, and promethazine. The core message for PONV treatment is that if antiemetic drug is given and patient still is symptomatic - Don't Repeat It. Instead, add a drug from a different therapeutic class.

PONV is a multi-factorial problem, and there is no single 'magic bullet' that can address the problem. We are however making progress.