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Post-Anesthesia Nausea and Vomiting: A Review of Pathophysiology, Treatment, and Prevention

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Abstract

Despite all advances in the science and art of anesthesia and surgery, Pos-Anesthesia Nausea and Vomiting (PANV) continues to be a common problem with an average incidence rate of 20-30%. [1] Most patients express concern about PANV and are more worried about it than they are about postoperative pain. These patients readily are willing to pay more for their anesthesia care in order to avoid this discomforting experience. PANV has a very low morbidity rate, however, it has been linked to aspiration pneumonitis, suture wound dehiscence, dehydration, electrolyte imbalance, and hematoma formation in the surgical sites. Additionally, patients may experience delayed discharge from the surgical facility, prolonged nursing care, and unanticipated hospital admission leading to significant increase in health care costs.[2-4]The dentist anesthesiologist is at the forefront of ambulatory anesthesia care and must be familiar with the pathophysiology, pharmacological, non-pharmacological management, and preventive measures pertaining to PANV.

Keywords

Anesthesia, Nausea, Vomiting, Post-Operative, Post-Anesthesia Nausea, Emesis,
Postoperative Nausea and Vomiting Management and Prevention

1. Methods

A literature search was conducted using related keywords in the following databases: PubMed, The Cochran Library, Web of Science, OMIN, and MD Consult. This was supplemented by a search for selected authors. Based on keywords, titles and abstracts, 1590 articles were identified. Pertinent data were then extrapolated from those papers to meet the objectives of this paper to include: 55 review articles covering pathophysiology, management and prevention of post-anesthesia and post-operative nausea and vomiting.

2. Pathophysiology

The pathophysiology of PANV is complex and poorly understood due to lack of adequate controlled clinical studies and lack of a good animal study model. Current evidence suggests that the process is centrally coordinated within the brain and a multitude of neurochemical events activate this process (Figure-1). Within the brainstem, there are two anatomically and functionally distinct areas that control vomiting:[1]

1 Medulla: bilateral vomiting centers within the reticular formation of the medulla receive afferent input from multiple sources and their activation ultimately result in emesis. These medullary vomiting centers receive signals from at least four major sources:[1,6]

A Visceral afferents, primarily the vagus nerve from the gastrointestinal tract. Gastric surgery, or gastric distention can directly activate the vomiting center

B Visceral afferents from outside the gastrointestinal tract. These signals include a variety of organs such as the heart, peritoneum, and the bile duct.

C Afferents from extramedullary centers in the brain. Vestibular system can cause nausea and vomiting as a result of surgery or sudden head movement upon emergence from anesthesia, leading to temporary middle ear imbalance. Psychological stimuli such as fear, anxiety, and anticipation of nausea may lead to

PANV.

D Chemoreceptor triggering zone (See below)

2 Chemoreceptor triggering zone (CTZ): is a bilateral set of centers in the brainstem lying under the floor of the fourth ventricle known as the postrema. The CTZ in the postrema is outside of the blood-brain barrier mechanism, rendering it to direct contact with emitogenic chemicals in the bloodstream. The CTZ has five different receptors that can activate the center by binding to neurotransmitters and similarly inactivate the center by binding to anti-emetic drugs. This multi-receptor architecture opens the therapeutic window to multi-drug therapy to manage PANV. Immunochemical studies of the CTZ reveal that these areas contain: histamine, serotonin, cholinergic, D_2 dopamine, and neurokinin-1 receptors. The emesis center in the reticular formation of medulla is connected to the CTZ via neuronal pathways (Figure-1).[1,6]

Additionally, the nucleus tractus solitarus, an area anatomically located within the medulla and pons, receives neuronal input via cranial nerves (IX, X); from a multitude of organs to include the heart, lungs, airway and the gastrointestinal system. The nucleus tractus solitarus is in communication with the CTZ and the vomiting center within the medulla. Pharyngeal stimulation leading to a gag reflex can activate the vomiting center via the nucleus tractus solitarus (Figure-1). [1,6]

The motor component of vomiting reflex is coordinated by both autonomic and somatic senses, regulated by the vomiting center of the brainstem. The vomiting reflex is divided into two physiologic phases. Phase I: Mediated by sympathetic and parasympathetic neuronal activity leading to sensation of nausea, salivation, tachycardia, sweating, and pupil dilation, followed by Phase II: retching and vomiting with repulsion of gastric contents through the oropharynx.[1, 6-7]

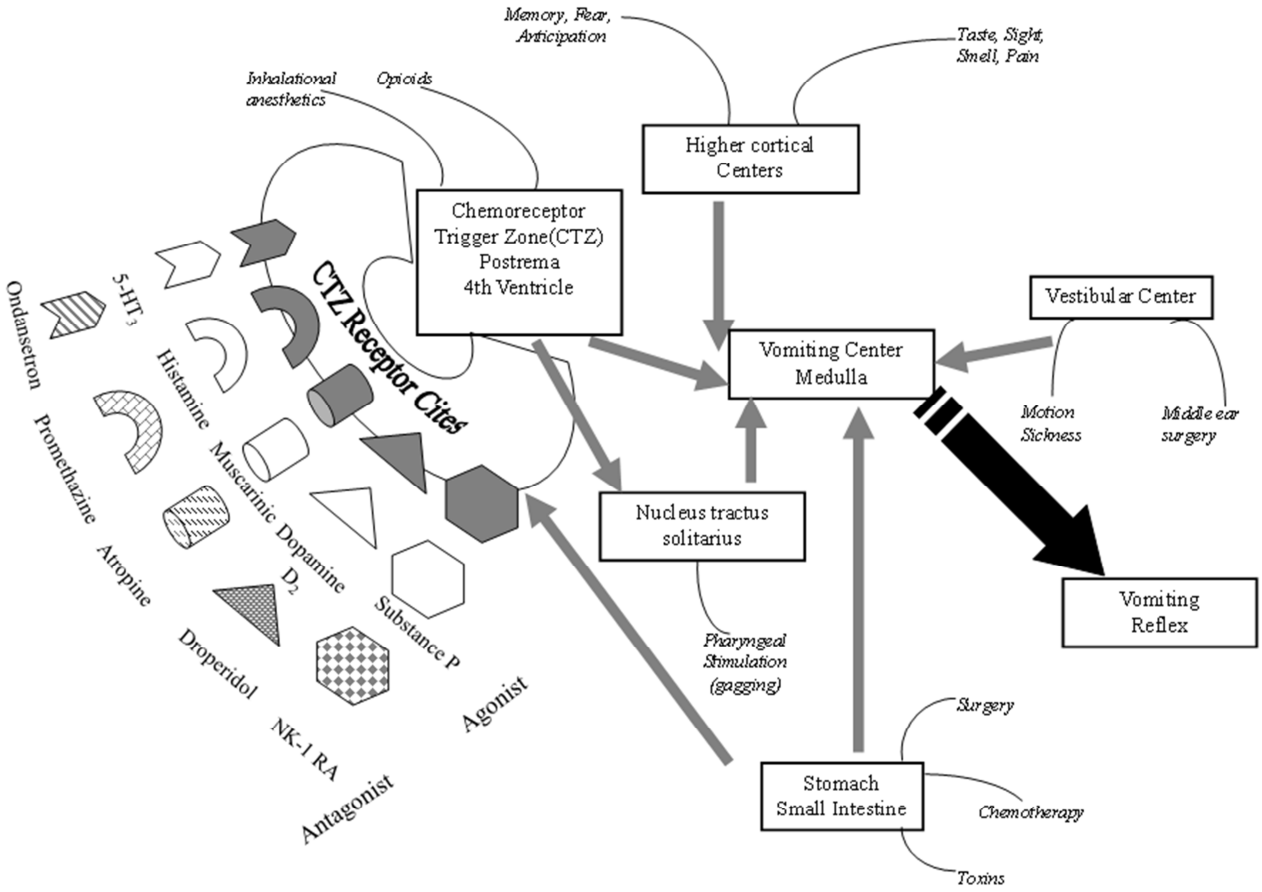

Figure 1. *Pathophysiology of PANV*

2.1. Factors Influencing Post-Anesthetic Nausea and Vomiting

To prevent or minimize PANV, the dentist anesthesiologist must identify patients who may benefit from pharmacological and non-pharmacological antiemetics therapeutics. Multiple predictive study models have identified: patient factors, anesthetic factors, and surgical factors as main contributors to development of PANV. [8]

2.1.1. Patient Factors

In adult patients, female gender, non-smoking, a previous history of PANV or motion sickness, are among the most important patient-specific predictors. Additional studies have pointed out anxiety, history of migraine headaches, obesity, delayed gastric emptying and pre-operative eating to play a significant role. Children are two times more likely to develop PANV than adults. This risk factor is very low in

infants and toddlers, increases up to age 5 and is highest between the ages of 6 and 16 years.[8-9]

2.1.2. Anesthesia Factors

Use of general anesthesia with volatile anesthetics and longer duration of exposure to inhalation agents are anesthesia-dependent risk factors. Halogenated anesthetics such as sevoflurane and desflurane are associated with lower rates of PANV than enflurane and halothane. Nitrous Oxide increases the incidence of PANV by directly stimulating the sympathetic nervous system, possible abdominal distention and further diffusion into spaces in the intestines activating specific mechanoreceptors. Use of nitrous oxide increases middle-ear pressure and directly stimulates the vestibular component of the vomiting center. Nitrous oxide can also directly stimulate central opioid receptors leading to PANV. Opioids are powerful emetogenic agents, which can directly activate the CTZ. Use of opioids in the post-anesthetic phase for surgical pain control can significantly increase the incidence of PANV, but so can post operative pain. This often requires the clinician to achieve a balance between good post-operative pain control and avoidance of PANV. Use of propofol for induction and maintenance of anesthesia as part of a total intravenous anesthetic technique (TIVA) has been linked to reduce the risk of PANV. Ketamine is a popular drug for short outpatient surgical procedures. Retrospective studies of Ketamine use for induction and maintenance of anesthesia reveal an increase in hallucination, vivid dreams and an increase in PANV. This has been linked to release of endogenous catecholamines. Uses of anticholinesterases such as neostigmine for reversal of non-depolarizing neuromuscular blocking drugs can increase the incidence of PANV due to their muscarinic actions on the intestinal tract. Finally, pharyngeal stimulation and gastric distention upon induction phase of anesthesia may stimulate the vomiting center in the medulla.[8-9]

2.1.3. Surgical Factors

Evidence suggests that Patients undergoing ophthalmologic, otolaryngologic, dental, oral and maxillofacial surgery, general orthopedic, plastics, laparoscopic, cranial and gynecological surgery have an associated increase risk for PANV. Post-surgically, sudden head movement upon extubation, ambulation, and transfer on stretchers to post recovery unit may create vestibular imbalance leading to PANV. Post-surgical hypovolemia, leading to hypotension, and dizziness can cause nausea and vomiting. Post-operative pain and use of opioids for control of pain are both emitogenic factors. Post-operative early oral intake can affect PANV as well.[8-9]

2.2. Pharmacological Treatment

There are several major classes of antiemetic drugs used in the management of PANV: antihistamines, anticholinergics, Dopamine D_2 antagonists and $5HT_3$ serotonin antagonists. Antiemetic drugs may have action at more than one receptor, but they tend to exert more prominent action at one or two receptors. Therefore, a combination of antiemetic drugs can have greater clinical benefit than a single drug.

2.2.1. Antihistamines

Antihistamines are competitive inhibitors of both the H_1 and muscarinic receptors of the vomiting center and to a lesser degree at CTZ. These drugs are particularly effective in management of nausea and vomiting due to vestibular activation of emetic pathway (e.g. middle ear surgery). Due to their antimuscarinic action, they can produce anticholinergic side effects such as drowsiness and sedation. Promethazine hydrochloride, a phenothiazine derivative is the most commonly used antihistaminic drug used in this category administered by oral, rectal, intramuscular, or intravenous routes.[6] Promethazine is also available as a transdermal gel prepared by compounding pharmacy centers. The topical gel is applied to the skin of inner wrist and may be an alternative route of administration for patients who do not tolerate oral or rectal medications at home. Promethazine is buffered with acetic acid-sodium acetate and has a pH between 4.0 to 5.5.[10] Multiple case reports in the literature have shown that accidental intra-arterial injection and or perivascular extravasation of promethazine can lead to significant chemical irritation and tissue necrosis resulting in digital or extremity amputation. The signs and symptoms may include pain in the site of injection, paresthesia of the digits, soft tissue edema and erythema followed by late onset signs of tissue necrosis.[11-12]Currently there are no published treatment algorithms for this devastating complication. However, early consultations with vascular and hand surgeons with emphasis on anticoagulation and thrombolysis are crucial in prevention of further damage.[13]Additionally, the Food and Drug Administration (FDA) is requiring the addition of a black box warning, cautioning that the drug should not be used in children under the age of 2 years old because of a potential for fatal respiratory depression.[14]

2.2.2. Anticholinergics

Anticholinergic drugs mainly block the action of acetylcholine at the muscarinic receptors of the vestibular apparatus and nucleus tractus solitarius and to a lesser degree at the CTZ. These drugs are useful in management of nausea and vomiting after middle-ear surgery; which is likely to alter the function of the vestibular apparatus. Transdermal Scopolamine is the most commonly drug used in this class of antiemetics. Its application continues up to twenty-four hours before the onset of surgery and has been shown to reduce the incidence of nausea and vomiting. Side effects include drowsiness, sedation, urinary retention, and dry mouth. Scopolamine can cross the blood-brain barrier and cause central nervous system excitation resulting in hallucination, confusion, and disorientation. [15-16]

2.3. Dopamine D_2 Antagonists

The chemoreceptor-triggering zone has an abundance of dopamine D_2 receptors. Their activation propagates signals to the vomiting center to induce nausea and vomiting.

Antagonists to these receptors at the CTZ include: butyrophenones, benzamides and phenothiazines

2.3.1. Butyrophenones

Droperidol has until recently been one of the most commonly used drugs for management of PANV. It is a heterocyclic neuroleptic, which inhibits dopaminergic receptors in the CTZ. In 2001, the FDA added a Black Box warning to the package insert of droperidol. This warning is related to reports of prolonged QT interval syndrome and cardiac dysrhythmias including Torsades de pointes. The FDA advises prolonged (2-3 hours) EKG monitoring when droperidol is used even at low doses (0.625mg-1.25mg) for the management of nausea and vomiting. Due to its potential arrhythmic effects, the FDA strongly recommends its use only in patients who have failed adequate response to other treatments.[17] However, this warning is highly controversial and multiple editorial reviews have concluded that actual cases of the arrhythmias that occurred in the case reports are confusing and none occurred after small doses of droperidol (1.25mg or less).[18] Therefore, there is little evidence to show cause and effect relationship between therapeutic doses of droperidol for PANV and cardiac arrhythmias.[18] Never the less, following the FDA recommendation, the dentist anesthesiologist must administer droperidol with extreme caution to patients who may be at risk for prolonged QT syndrome (e.g., congestive heart failure, bradycardia, use of diuretics, cardiac hypertrophy, hypokalemia, and hypomagnesemia, age over 65 of years and alcohol abuse). Droperidol should be initiated at a low dose, typically 0.625mg and to a maximum dose of 1.25mg as needed to achieve the desired effect. At higher doses, central nervous system extrapyramidal activity and possible hypotension due to its peripheral alpha-blocking properties have been noted. [18-20]

2.3.2. Benzamides

Metoclopramide and domperiodone are the most commonly used Benzamides. Metoclopramide a derivative of procainamide is capable of blocking central and peripheral dopamine receptors and promotes gastric mobility while enhancing lower esophageal tone. At high doses, metoclopramide also exhibits a weak $5HT_3$ blocking activity. Theoretically these properties make metoclopramide an ideal drug with concurrent opioid administration.[6] However, clinical reviews of randomized placebo trials have found no difference between the placebo group and patients treated with metoclopramide for management of PANV. Additionally, combination of this drug with another antiemetic has not shown to produce enhanced efficacy. As a result, these groups of D2 blockers are no longer used as a first line of treatment for management of PANV.[21] Metoclopramide is capable of crossing blood brain barriers and must be avoided in patients who suffer from nausea and have Parkinson's disease. Receiving large doses of metoclopramide has resulted in high incidence of dystonic reactions, particularly in children. Finally, due to its prokinetic properties, it must be avoided in patients with suspected bowel obstruction. Domperiodone is a central dopamine antagonist without the $5HT_3$ antagonist effects. It does not cross the blood brain barrier and may be an alternative for patients with Parkinson's disease.[6]

2.3.3. Phenothiazines

Prochlorperazine belongs to the phenothiazine group of antipsychotic medications, currently used primarily in the management of nausea and vertigo. The antiemetic actions of phenothiazines are attributed to their central blocking action of dopamine receptors at the CTZ and are active against emetic stimuli arising from the gastrointestinal tract. Prochlorperazine, the commonest phenothiazine used is available as an oral, rectal and an injectable form. An inhaled form of the medication is currently undergoing research trials. Prochlorperazine has been a very popular anti-emetic drug for a number of years. However, it can produce significant sedation, thereby delaying discharge.[1,6] In addition, extrapyramidal side effects, persistent tardive dyskinesia and neuroleptic malignant syndrome have been associated with phenothiazine antipsychotic medications. The dentist anesthesiologist must also monitor the patient for significant hypotension after administration of prochlorperazine especially in the elderly and those with compromised cardiovascular system, and be prepared to provide appropriate treatment. Hypotension from prochlorperazine should be treated with fluids and a vasoconstrictor such as phenylephrine. Other pressures, such as epinephrine is not recommended because it may cause a paradoxical further lowering of blood pressure.[1,6]

2.4. Serotonin 5-HT_3 Antagonists

These groups of antiemetic drugs were introduced in the 1990s for the management of chemotherapy-induced nausea and vomiting. They have since been proven highly effective and currently are the most commonly used antiemetic class of drugs for the management of PANV. There are at least seven different types of serotonin receptors, each with a different function. The 5-HT_3 receptors are present periphery, within the area of CTZ, as well as in the nucleus tractus solitarius. Ondansetron, one of the first 5-HT_3 antagonists to be introduced is still the most commonly used drug in this class. Others include, granisetron, tropisetron, dolasetron and palonosetron. [22-23]

2.4.1. Ondansetron

Ondansetron is a carbazalone derivative that is structurally related to serotonin and has specific 5-HT_3 receptor antagonist properties, without altering dopamine, histamine, and adrenergic or cholinergic receptors. Ondansetron has a relatively short half life (3-5 hours) and is available in oral (tablet, elixir, and orally disintegrating tablets), intravenous, and suppository forms.[24] The dose-response relationship studies of ondansetron reveal that optimal dose for prophylaxis prevention of PANV is 4mg intravenously with 8mg for higher risk patients, without any additional benefit

from increased doses (16mg). [25] Its administration near the end of the surgery may result in significantly less incidence of PANV. Additionally, for the treatment of established PANV, no significant differences are found between 4mg or 8mg when used as a rescue medication in the post anesthetic period. [26]

2.4.2. Granisetron, Tropisetron and Dolasetron

Although multiple studies have compared the efficacy of different 5-HT_3 antagonists, they only vary primarily by their duration of action. The elimination half-life of granisetron and tropisetron is approximately nine hours, 2.5 times longer than ondansetron. Dolasetron is metabolized into hydrodolasetron, which is the antiemetic compound. Hydrodolaseteron has a half-life of seven hours. As a result, Granisetron, Dolasetron, and Tropisetron may require less frequent dosing than their parent drug Ondansetron. [27-29]

2.4.3. Palonosetron

Palonosetron is one of the latest 5-HT_3 receptor antagonists used for the management of chemotherapy-induced nausea and vomiting. In 2008, the FDA approved its use for management of PANV. Palonosetron shows a binding affinity that is 100 times the potency of ondansetron for the 5-HT_3 receptors. The half-life in healthy adult volunteers has been recorded to up to forty hours. Preliminary studies have shown that a dose of 0.075mg given Perioperatively provides effective prophylaxis and treatment of PANV. Current and future research needs to be done towards comparisons of the efficacy of palonosentron and other 5-HT_3 antagonists in establishing appropriate drug dosage and cost effectiveness in management of PANV. [30]

Most common side effects of all 5-HT_3 antagonists include headache, flushing, constipation, and a transient elevation of liver enzymes. All 5-HT_3 antagonists block cardiac sodium ion channels in vitro studies and therefore have a potential in causing cardiac conduction. EKC changes to include a transient increase in PR, QRS, and QT intervals have been recorded after administration of Dolasetron and prolonged QT intervals after administration of ondansetron. However, single dose usage of 5-HT_3 antagonists is considered unlikely to cause any cardiovascular effects. [31-32]

2.4.4. Other Drugs

(i) Steroids

The antiemetic role of glucocorticoids such as dexamethasone has been well established in management of PANV. It is hypothesized that dexamethasone reduces the physiologic levels of tryptophan, the biochemical precursor to 5-hydroxytryptomine, or have an anti-inflammatory action on the gastrointestinal tract, reducing the release of serotonin.

Intravenous administration of dexamethasone at the beginning of surgery has been shown to be most effective in prevention of PANV. Its antiemetic efficacy is also better if it is used in combination with another drug such as the 5-HT_3 antagonists rather than as the sole agent. The immune suppression activity of dexamathasone and its correlation for potential postoperative wound infection at dosages given for management of PANV has not been studied. [33]

(ii) Propofol

Propofol has been found to reduce the incidence of PANV when it is used both as an induction and a maintenance agent. Clinical studies have shown that a 20mg bolus of propofol given in the postoperative period is effective in the treatment of established PANV. The mechanism of action of propofol as an antiemetic agent is still under investigation. There appears to be no specific antiemetic receptor activity, and no gastric prokinetic changes. Animal studies using immunohistochemistry have demonstrated a reduction in brain postrema activity and a lower concentration of serotonin in presence of propofol administration.[34-35]

(iii) Oxygen

The use of supplemental oxygen in prevention of PANV has been the subject of multiple clinical investigations. Oxygen at 80% concentration administered intraoperatively reduced the incidence of PANV from 30% to 17% when compared with 30% oxygen. Additionally, 30% oxygen given intraoperatively and for 2 hours postoperatively has been shown to be as effective as 8mg of ondansetron for prevention of PANV. These studies may explain the potential role of higher arterial blood oxygen saturation in reducing the incidence of PANV.[36]

2.5. Neurokinin-1 (NK_1) Antagonists

NK-1 receptor antagonists represent the newest class of anti-emetic drugs currently under investigation for prevention and management of PANV. Substance P is a neuropeptide, which plays an essential role in transmission of noxious stimuli from peripheral nervous system. Substance P is a ligand for the NK-1 receptors found within the CTZ, and in the medullary areas where emetic inputs converge and the NK-1 receptor antagonists may be useful in blocking this central emetic stimuli.[37]

2.6. Nonpharmacologic Treatments

None pharmacological techniques such as acupuncture, elcetroacupuncture, transcutaneous electrical stimulation, and acupressure have all been examined as alternatives to antiemetic medications. The location of stimulation is at the pericardium P-6 (Neiguan) point located between the tendons of Palmaris longus and flexor carpii radialis muscles 4cm proximal to the wrist crease. It is hypothesized that acupuncture at this point may lead to release of Beta-endorphins in the cerebral spinal fluid, potentiating the endogenous antiemetic mechanisms and altering serotonin transmission. The main findings of meta-analysis examining this method of management of PANV, demonstrate that the precise stimulation of P-6 point is more important than the modality of its stimulation. In the adult population, no pharmacologic techniques are more effective than placebo and at least comparable to Ondansetron in one study in

prevention of PANV. In children, P-6 point stimulation is not effective when compared with either placebo or antiemetic medications. Finally, the duration of P-6 stimulation, the degree of needle penetration, and dominant hand versus non-dominant hand are all variables between different clinical trials, which require further investigation. No serious side effects have been reported with this modality of prevention and treatment.[38]

3. Discussion

Successful prevention and treatment of PANV requires a systemic approach to the patient, surgical and anesthetic risk factors. The first goal of the dentist anesthesiologist should be to minimize base line anesthetic risk factors by assuring adequate hydration of the patient, reducing preoperative anxiety, minimizing opioid use, avoiding high dose reversal of a neuromuscular antagonist, using high concentration of oxygen, minimizing prolonged high concentrations of nitrous oxide and utilizing a total intravenous anesthesia technique with propofol thereby avoiding inhalational anesthetics.

Therapeutic prophylaxis use of antiemetics has been extensively studied. Essentially there is no 100% effectiveness in prevention of PANV. Some patients may receive prophylactic antiemetics without needing it, while others needing it; receive antiemetics and still experience PANV. The science is not perfected at this time. To identify those patients who may benefit from antiemetics, predictive clinical studies have been conducted. Apfel, et al conducted a two-center in-patient study and developed a risk score system using four factors: female gender, history of motion sickness, previous PANV, non-smoker and use of perioperative opioids. If none, 1,2,3 or 4 of these risk factors were present, the incidence of PANV were 10%, 21%, 39%, 61% and 79% respectively. Currently evidence suggests that there is no need for prophylactic treatment in patients with minimal to no risks for development of PANV. For patients who have higher risks factors discussed earlier in this article, base line anesthetic risk factor reduction, a multi-modal pharmacological approach and possible addition of non-pharmacological modalities are recommended for prevention of PANV.[39-41] Table-1

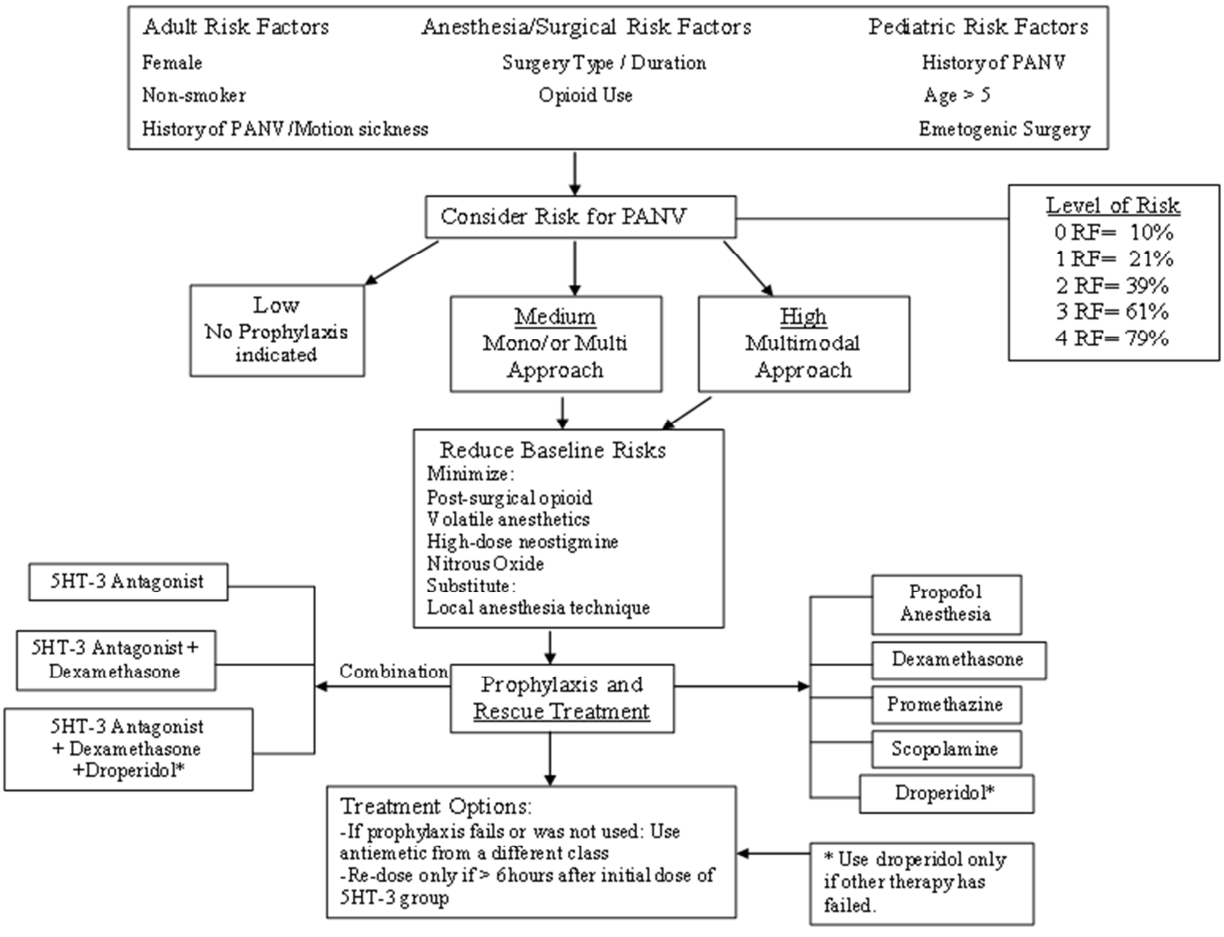

Figure 2. *Algorithm for management of PANV*

In the pediatric population, the risk of PANV can be much higher than the adult population, indicating a greater need for prophylactic coverage. Currently Ondansetron is approved for use in children as young as one-month-of age. Recommended dosage is between 50-100 micrograms/kilogram. Dolasetron is also recommended for prophylaxis but only for children aged 2 years and older. Recommended dose of dolasetron is 350-microgram/

kilogram. PANV evaluation in children is usually limited to the vomiting phase due to the limits of evaluation in nonverbal children. 5HT-3 antagonists provide a higher anti-vomiting coverage than nausea reduction and therefore have been chosen as the first line of treatment in this population.[40-41] Table-2

Patients with PANV who did not receive prophylaxis or in whom prophylactic treatment failed, treatment should be with an antiemetic from a class of drugs that is different from the prophylactic antiemetic. If no prophylaxis was given, the first line therapy should be a 5HT-3 antagonist. Additionally, dexamethasone, droperidol, promethazine and a low dose of propofol (20mg) may be given in the post anesthetic care unit for rescue therapy. It should be noted that antiemetic effects with low doses of propofol are brief.

Table 1. *Recommended dosage of commonly used antiemetics for the prophylaxis of PANV in adults*

Agent	Dosage
Ondasetron	4mg IV 8mg PO 1 hour before induction
Dolasetron	12.5mg IV 100mg PO 1 hour before induction
Granisetron	20-40mcg/kg IV
Promethazine	25mg PO 1 hour before induction 12.5-25mg IV
Prochlorperazine	5-10mg IV 5-15mg PO 1 hour before induction
Metoclopramide	10mg IV (not effective as the sole agent)
Dexamethasone	4mg IV
Droperidol	0.625-1.25mg IV

Table 2. *Recommended dosage of antiemetics commonly used for the prophylaxis and treatment of PANV in children*

Agent	Dosage
Prophylaxis	
Ondansetron	0.05mg/kg IV
Dolasetron	Age>2 years: 1.8mg/kg IV before induction
Droperidol	0.015mg-0.075mg/kg IV
Treatment	
Ondansetron	0.05mg/kg IV
Droperidol	0.1mg/kg IV

Table 3. *Recommended dosage of antiemetics commonly used for treatment of PANV in adults*

Agent	Dosage
Ondasetron	1-4mg IV postoperatively
Dolasetron	12.5mg IV postoperatively
Promethazine	12.5-25mg IV every 4 hours as needed postoperatively 10-25mg PO every 4-6 hours as needed postoperatively
Prochlorperazine	5-10mg IV 5-15mg PO postoperatively
Metoclopramide	10mg IV (not effective as the sole agent)
Droperidol	0.625-1.25mg IV postoperatively
Propofol	20mg IV postoperatively in PACU

In the post anesthetic period, the clinician should start rescue treatment when the patient complains of nausea or is vomiting, simultaneous evaluation of possible etiological factors related to anesthetic and surgical procedure should be initiated. In the oral and maxillofacial surgery or dental patient, blood draining into the oral or nasal pharynx is a common etiological factor. Once rescue treatment is initiated, the provider is left with the question of what to do in case the treatment fails. Repeated dosing of 5HT-3 inhibitors within the first 6 hours after the initial dose whether it was given for treatment or prophylaxis provide no additional benefits in management of established PANV. Increasing the second dose within that same period appears to provide no additional benefit either. Therefore, a second drug from a different class of antiemetic must be chosen as a rescue medication. After the initial 6 hours, the same medication can be repeated to provide additional receptor activity to reduce PANV.[40-41] Table-3

4. Conclusion

PANV is the leading cause of emergency hospital admission. In some high-risk patients, the risk of PANV could be as high as 80%. But according to the International Anesthesia Research Society (IARS) guidelines, prophylactic treatment for all patients is not deemed cost-effective[9]. And the panel suggested not all patients should receive prophylaxis. The prophylactic intervention is reserved only for those who are at "moderate" risk for PANV; however, the committee did not define "moderate" risk – A decision was made, instead, to allow the health care professionals who use the IARS guidelines to determine the level of risk according to their own clinical judgments and institutional requirements.[41]

Nomenclature

Nausea: The sensation of unease and discomfort in the stomach typically associated with decrease gastric motility and an urge to vomit, but without the explosive muscular movement of the aerodigestive tract.[5]

Retching: Presence of spasmodic, explosive aerodigestive muscular movement without the expulsion of stomach contents.[5]

Vomiting: The forceful expulsion of gastrointestinal contents through the mouth and (Emesis) sometimes the nose, caused by contraction of muscles.[5]

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