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Postoperative Nausea and Vomiting: a Review of Current Literature

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ABSTRACT

Introduction: Postoperative nausea and vomiting (PONV) is one of the commonest complaints following anaesthesia, and can result in morbidity like wound dehiscence, bleeding, pulmonary aspiration of gastric contents, fluid and electrolyte disturbances, delayed hospital discharge, unexpected hospital admission, and decreased patient satisfaction.

Method: A literature search was done on the Medline and relevant articles chosen.

Results: Despite the vast amount of research done in this field and the variety of antiemetic drugs available, PONV still has a high incidence. Many factors are associated with PONV. Quantifying the relative impact of risk factors on PONV has resulted in the development of risk models, which can stratify risk categories and hence allow the anaesthetist to identify those patients at higher risk for PONV. The management of PONV requires a multi-modal approach which can include the use of less emetogenic anaesthetic techniques, balanced analgesia, appropriate intravenous hydration, the use of pharmacotherapy and possibly non-pharmacologic methods.

Conclusions: The use of risk models facilitates the judicious use of pharmacotherapy to ameliorate PONV especially in the high-risk patient and may lead to a more cost effective and efficient means of managing PONV.

Keywords: incidence, risk factors, antiemetics, risk models, management

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PHYSIOLOGY OF NAUSEA AND VOMITING

Nausea is the conscious recognition of excitation of an area in the medulla that is associated with the vomiting (emetic) centre, which mediates the vomiting response. The medullary vomiting centre is located in the lateral reticular formation of the medulla[1], close to the fourth cerebral ventricle. It receives afferents from the chemoreceptor trigger zone (CTZ), vestibular apparatus, cerebellum, higher cortical and brainstem centres, and solitary tract nucleus. These structures are rich in dopaminergic, muscarinic, serotoninergic, histaminic and opioid receptors. Block of these receptors may be the mechanism of the antiemetic action of drugs. Efferents are transmitted via cranial nerves V, VII, IX, X and XII to the gastrointestinal tract and through the spinal nerves to the diaphragm and abdominal muscles to cause the mechanical act of vomiting.

The chemoreceptor trigger zone is in or near the area postrema, on the lateral walls of the fourth ventricle near the obex. It includes serotonin, dopamine, histamine, muscarinic and opioid receptors. The CTZ is not protected by the blood-brain barrier. Hence, it can be activated by chemical stimuli received through the systemic circulation as well as the cerebrospinal fluid. The cerebral cortex is stimulated by smell and physiologic stresses. Motion can stimulate the vestibular apparatus, which may also stimulate the CTZ. The neurovegetative system is sensitive principally to gastrointestinal stimulation. Blocking of impulses from the CTZ does not prevent vomiting due to irritative stimuli arising from the gastrointestinal tract.

INCIDENCE AND RISK FACTORS

General anaesthesia using volatile anaesthetics is associated with an average incidence of postoperative nausea and vomiting (PONV) ranging between 20% and 30%[1]. PONV is thought to be multifactorial in origin, involving anaesthetic, surgical, and individual risk factors[1-4]. Only some of these factors can be influenced by the anaesthetist (Table I).

Factors not under the control of the anaesthetist

Some of these factors which affect the incidence of PONV include age, sex, history of previous PONV or motion sickness, smoking, surgical procedure, duration of surgery and anaesthesia, and patient and parental anxiety.

Sinclair et al reported that the incidence of PONV decreased after age 50 years. Age decreased the likelihood of PONV by 13% for each 10-year increase[5].

However, Koivuranta et al did not find age to be a predictive factor for nausea, except for patients older than 50 years who were undergoing joint replacement and spinal surgery, in whom there was an increased risk for postoperative vomiting[6].

Women have three times the risk for PONV compared to men[6,7]. This gender difference has been attributed to variations in serum gonadotropin or other hormone levels[1,8-10].

History of previous PONV or motion sickness increases the risk for PONV by two to three times. This factor has been reported as a strong predictor of PONV[5-7].

Smoking is associated with a decreased risk for PONV[5-7,11]. The relative risk for PONV in smokers is 0.6[11]. Sinclair et al reported that smoking decreased the likelihood of PONV by 34%[5].

Some operations are reported to be associated with a higher incidence of PONV than others. These include plastic (breast augmentation), ophthalmologic (strabismus repair), ENT-dental, gynaecologic, laparascopic (sterilisation), genitourinary, orthopaedic surgery (shoulder procedures), mastectomies and lumpectomies[5]. However, there are conflicting reports on whether the type of operation is a predictor of PONV[5-7,12]. Its causal impact on PONV remains questionable because a high incidence of PONV after certain operations might be caused by the involvement of high-risk patients[7]. It is unclear if the association is caused by the different anaesthetic agents[8], the different lengths of operation[2], or the operation itself[13].

With increasing duration of surgery and anaesthesia, the risk of PONV increases possibly because of greater accumulation of emetogenic anaesthetic agents[1,5,8]. The incidence of PONV increases from 2.8% in patients with a surgical duration of less than 30 minutes to 27.7% in patients with a surgical duration of between 151 to 180 minutes. The duration of anaesthesia increases the risk for PONV by 59% for each 30-minute increase[5].

Factors under the control of the anaesthetist

These are factors related to the anaesthetic. Factors such as premedication, type of anaesthesia, intraoperative anaesthetic drugs, postoperative management and antiemetic drugs can affect the incidence of PONV.

Premedication

Premedication is used for anxiolysis, sedation, analgesia, and to reduce airway secretions and cardiovascular responses during induction. In children, it facilitates separation of the child from the parents and acceptance of the face mask during induction. The α2 agonist clonidine, can reduce

Table I. Factors affecting the incidence of postoperative nausea and vomiting.

Factors not under the control of the anaesthetist

1) Age
2) Sex
3) History of previous PONV or motion sickness
4) Smoking
5) Surgical procedure
6) Duration of surgery and anaesthesia
7) Patient and parental anxiety

Factors under the control of the anaesthetist

1) Premedication
2) Type of anaesthesia
3) Intraoperative anaesthetic drugs
   (a) Nitrous oxide
   (b) Intravenous agents
   (c) Inhalation agents
   (d) Antagonists of non-depolarising neuromuscular blocking drugs
4) Postoperative management
   (a) Pain management
      (i) Local anaesthetics
      (ii) NSAIDs
      (iii) Opioids
   (b) Movement
   (c) Oral intake
   (d) Non-pharmacological – acupressure/acupuncture
5) Antiemetic drugs
6) Other factors – hypovolemia, gastric distension

Table II. Summary guidelines for the prophylaxis and rescue of postoperative nausea and vomiting[7,49,85]. Incidence of PONV in each risk group is indicated in brackets.

Risk factors	Prophylaxis	Rescue
• Female gender • Non-smoking status • History of PONV or motion sickness • Postoperative use of opioids		
None (10%)	None recommended	Ondansetron 1 or 4 mg
One (21%)	None recommended	Ondansetron 1 or 4 mg
Two (39%)	Droperidol 0.625 or 1.25 mg	Ondansetron 1 or 4 mg
Three (61%)	Droperidol 0.625 or 1.25 mg plus dexamethasone 8 mg ± metoclopramide 10 mg	Ondansetron 1 or 4 mg
Four (79%)	Droperidol 0.625 or 1.25 mg plus dexamethasone 8 mg plus ondansetron 4 or 8 mg	Metoclopramide 10 mg or any other group of antiemetic (e.g. phenothiazine, 5-HT$_3$ receptor antagonist like granisetron)

PONV in children after strabismus repair[14]. It is postulated that clonidine may reduce PONV by decreasing anxiety[14]. Premedication with opioid analgesics, on the other hand, increases the risk of PONV[15].

Type of anaesthesia

It has been reported that patients receiving general anaesthesia were 11 times more likely to experience PONV than those who received monitored anaesthetic care, regional anaesthesia or a chronic pain block[5].

Intraoperative anaesthetic drugs

There have been conflicting reports regarding the effect of nitrous oxide on PONV. It has been reported that nitrous oxide produces a greater incidence of vomiting[16], that omission of nitrous oxide reduces the incidence of vomiting[17], but only if the baseline risk of vomiting is high in the patient population[18]. However, there was no reduction in the incidence of nausea when nitrous oxide was omitted[17,18]. A study in rats suggested that nitrous oxide causes stimulation of the medullary periventricular dopaminergic system, which includes the CTZ, and this could be responsible for the nausea and vomiting observed after nitrous oxide anaesthesia in humans[19]. Caution must be made when omitting nitrous oxide to reduce PONV because the risk of intraoperative awareness would increase[18].

Modern potent inhalation anaesthetics are associated with a lower incidence of PONV than ether and cyclopropane[1]. The differences in the incidence of PONV with isoflurane, desflurane, sevoflurane and enflurane are not well documented.

Propofol, an intravenous hypnotic agent, is associated with a lower incidence of postoperative nausea and vomiting when it is used for induction of anaesthesia, compared with thiopentone[20]. In fact, sub-hypnotic doses of propofol were effective in reducing nausea and vomiting associated with general anaesthesia[21-23]. The mechanism of propofol for reducing PONV is unknown. Thiopentone, etomidate, and ketamine are more emetogenic than propofol[20,24]. There is insufficient evidence, however, that total intravenous anaesthesia with propofol is an anaesthetic technique with a low emetogenic potential that is clinically relevant[17].

It is commonly thought that the use of antagonists of neuromuscular block (anticholinesterases) such as neostigmine for the reversal of non-depolarising neuromuscular blocking drugs can increase the incidence of PONV[25] due to the muscarinic actions on the gastrointestinal tract. It is interesting then that some authors reported no significant difference in PONV between those who received a reversal agent and those who did not[5,26]. Atropine given concomitantly with neostigmine may decrease PONV during the early postoperative period.

Postoperative factors

Pain can increase the incidence of PONV[27] by prolonging gastric emptying time and hence resulting in nausea and vomiting. Opioids are often used to treat postoperative pain. However, the use of postoperative opioids can increase PONV. Apfel and colleagues have derived a risk score for predicting PONV, which includes the use of postoperative opioids as a significant predictor[7]. Opioids' mechanisms of action are direct stimulation of the CTZ, increased vestibular sensitivity, and decreased motility of the stomach, and small and large intestines[28]. However, as the emetogenic profile of opioids varies in different individuals, it is possible to reduce severity of opioid-induced PONV by selecting a different opioid. Balanced analgesia using combinations of systemic opioids, regional nerve blocks, local anaesthetics, and other forms of analgesia like non-steroidal anti-inflammatory drugs (NSAIDS) can be used to manage pain and reduce the incidence of opioid-related PONV[29,30].

Regional anaesthesia can be used as the sole anaesthetic or as a supplement to general anaesthesia. It can reduce PONV[5,6,13] possibly by reducing the requirement of general anaesthetics and opioids during an operation and by serving as residual analgesia in the early postoperative period with the subsequent decreased use of postoperative opioids for analgesia.

Postoperative hypovolemia can result in orthostatic hypotension, dehydration and dizziness, all of which can increase PONV. Appropriate intraoperative fluid administration has been reported to reduce postoperative nausea and vomiting following ambulatory surgery[31].

Gastric distension has been associated with increased PONV[1]. However, routine aspiration of gastric contents via orogastric suctioning has either no effect or increases the risk for PONV[13,32,33].

Early motion postoperatively[6,8] including nursing procedures, ambulation, and transfer on stretcher, wheelchair or vehicle can increase PONV, especially in patients who have received opioids.

Postoperative oral intake can affect PONV as well. Van den Berg et al have shown that many patients who vomit in the postoperative period do so after taking their first drink[34]. Patients should choose when they want to start oral intake and the diet can be advanced accordingly when they are ready.

ANTIEMETIC DRUGS

There are several classes of drugs that constitute the mainstay of antiemetic therapy. These range from older drugs like droperidol, metoclopramide to 5-HT_3

antagonists, which were the focus of many studies and clinical trials in the 90s. Despite extensive research and the introduction of newer classes of antiemetic drugs with better efficacy and safety profiles, there seems to be little progress in reducing the incidence of PONV.

Butyrophenones

Droperidol is the only commonly used butyrophenone for its antiemetic action. It is a heterocyclic neuroleptic which inhibits dopaminergic receptors in the chemoreceptor trigger zone of the medulla. Side effects include sedation, drowsiness (dose-dependent), dysphoria, restlessness and rarely extrapyramidal reactions. Children may be more vulnerable to droperidol-related extrapyramidal symptoms. The likelihood of droperidol-related sedation or drowsiness increases with increasing doses above 0.625 mg, from a risk of one in 24 with 1.25 mg, to one in 8 with 2.5 mg. Its anti-nausea effect is not dose-dependent, is more pronounced than the anti-vomiting effect, and is short-lived. Its anti-vomiting efficacy improves considerably with increasing doses not beyond 2.5 mg[35]. Droperidol, in doses as low as 0.625 or 1.25 mg has been shown to be as effective as ondansetron 4 mg without increasing sedation, agitation, anxiety or delaying discharge[36]. Droperidol and ondansetron were similarly effective in preventing PONV in adults. Droperidol in small doses (e.g. 0.625 mg) is highly effective in adults and has minimal side-effects[37].

Benzamides

Metoclopramide is the most effective antiemetic of this class and has been used for almost 40 years. It is a dopamine antagonist that is structurally similar to procainamide. Its antiemetic effect results from antagonism of dopamine's effects in the chemoreceptor trigger zone. At high doses, it also antagonises 5-HT_3 receptors. Additional antiemetic effects are due to its dopaminergic and cholinergic actions on the gastrointestinal tract with increases in lower esophageal sphincter tone and facilitation of gastric emptying into the small intestine. These latter effects reverse the gastric immobility and cephaled peristalsis that accompany the vomiting reflex. Opioid-induced PONV can be treated with metoclopramide because it reverses the gastric stasis induced by morphine. There was no evidence of dose-responsiveness, with the best documented regimen in adults being intravenous (i.v.) 10 mg and in children i.v. 0.25 mg/kg[38]. Side effects include abdominal cramping, sedation, dizziness, and rarely dystonic extrapyramidal reactions (oculogyric crises, opisthotonus, trismus, torticollis), and cardiac dysrhythmias. Metoclopramide has been shown not to be as effective as ondansetron and droperidol in preventing postoperative vomiting in a meta-analysis[37]. However, a systematic review showed that metoclopramide has no clinically relevant antiemetic effect and does not show an increased risk of adverse effects in the doses currently used in anaesthesia. It is likely that the doses used in daily clinical practice are too low. Hence, the continued use of metoclopramide in the dose ranges tested in these studies is inadequate[38].

Histamine Receptor Antagonists

Those for use in PONV are the H_1 receptor antagonists, with the most commonly used being dimenhydrinate. H_1 receptor antagonists are competitive antagonists of histamine by occupying H_1 receptors on effector cell membranes, thus preventing histamine binding and activity. They have sedative effects, especially first-generation drugs. Dimenhydrinate's efficacy in motion sickness and inner ear diseases results from inhibition of the integrative functioning of the vestibular nuclei by decreasing vestibular and visual input. Intravenous dimenhydrinate 20 mg decreases vomiting after outpatient surgery in adults[39]. In children, i.v. dimenhydrinate 0.5 mg/kg significantly decreases the incidence of vomiting after strabismus surgery and is not associated with prolonged sedation[40].

Muscarinic Receptor Antagonists

Morphine and synthetic opioids increase vestibular sensitivity[28]. The vestibular apparatus of the inner ear and the nucleus of the tractus solitarius are rich in muscarinic and histamine receptors. It is postulated that scopolamine blocks transmission to the medulla of impulses arising from overstimulation of the vestibular apparatus. Application of a scopolamine patch before the induction of anesthesia protects against PONV after middle ear surgery that is likely to alter the function of the vestibular apparatus[41]. Transdermal scopolamine patches can reduce PONV in patients receiving epidural morphine[42,43]. Side effects include sedation, dry mouth and visual disturbances.

5-HT_3 Receptor Antagonists

These drugs produce pure antagonism of the 5-HT_3 receptor. The introduction of this class of drugs in the 90s represents a major improvement in the pharmacotherapy of chemotherapy and radiation therapy-induced nausea and vomiting. They have since proven to be highly effective in the prevention and treatment of postoperative nausea and vomiting. They are not effective in the treatment of motion-induced nausea and vomiting. Ondansetron, the first 5-HT_3 receptor antagonist to be introduced, is the most commonly used drug of this class. Others include granisetron, tropisetron and dolasetron.

Ondansetron

Ondansetron is a carbazalone derivative that is structurally related to serotonin and possesses specific 5-HT_3 subtype receptor antagonist properties, without altering dopamine, histamine, adrenergic, or cholinergic receptor activity[32]. The most serious side effects of ondansetron are rare hypersensitivity reactions[44]. Other more commonly reported side effects are headache, light-headedness, dizziness, flushing at the i.v. site, transient increases in the plasma concentrations of liver transaminase enzymes, a warm epigastric sensation, and constipation[44-46]. Cardiac dysrhythmias have been reported after i.v. administration of ondansetron and metoclopramide[47]. Of 100 patients receiving prophylactic ondansetron, three will have transiently elevated liver enzymes, and three will have a headache who would not have had these adverse effects without the drug. Ondansetron-induced headache may be dose-dependent but for the other adverse effects, no such dose-dependence could be established[48]. The usual clinical doses of ondansetron (4 to 8 mg), droperidol (0.625 - 1.25 mg) and metoclopramide (10 mg) do not differ in the overall incidence of adverse effects[37].

There are numerous studies on the efficacy of ondansetron for preventing PONV. Tramèr et al reported that for every 100 patients at high risk for PONV who receive ondansetron for the prevention of PONV, 20 (number-needed-to-treat: 5) patients will not vomit who would have vomited without treatment. The optimal prophylactic intravenous dose of ondansetron was likely to be 8 mg for long-term efficacy. The antiemetic efficacy of ondansetron was consistently better than its anti-nausea efficacy[48]. Watcha and White re-analysed data used by Tramèr et al[48] and found that the absolute success rates for propyhlaxis with ondansetron 4 and 8 mg i.v. did not significantly differ for the separate incidences of nausea and vomiting[49]. Ondansetron administered near the end of the surgery, rather than before surgery, may result in higher efficacy and better patient satisfaction[50]. In their meta-analysis, Domino et al concluded that ondansetron and droperidol were more effective than metoclopramide in preventing PONV. Ondansetron was more effective than droperidol in preventing PONV in children but they were equally effective in adults[37]. Some studies, however, reported that ondansetron was not effective for the prevention of PONV[51,52] or that ondansetron was no more effective than supplemental intraoperative oxygen[53].

As for the treatment of established PONV, Tramèr et al concluded that there were no differences in the effectiveness of 1, 4, or 8 mg ondansetron when used for rescue from PONV in the PACU[54]. They also concluded that ondansetron did not differ significantly in its antiemetic effects from droperidol or metoclopramide when given in the PACU for established emesis. However, other studies comparing ondansetron and metoclopramide have shown that ondansetron has greater efficacy in controlling established PONV[55,56].

Granisetron

Granisetron is a more selective 5-IV_3 receptor antagonist than ondansetron. An i.v. dose as low as 0.04 mg/kg is effective in the prevention of PONV[57,58]. The elimination half-life of granisetron (nine hours) is 2.5 times longer than that of ondansetron and thus may require less frequent dosing. The high cost of granisetron may limit its clinical application[57].

Dolasetron

Dolasetron is a highly potent and selective 5-IVT_3 receptor antagonist. The optimal dose for prophylaxis is 50 mg if given at induction of anaesthesia[59]. Established PONV is effectively ameliorated by IV dolasetron 12.5 mg[60]. After its administration, dolasetron is rapidly metabolised to hydrodolasetron, which is responsible for the antiemetic effect. Hydrodolasetron has an elimination half-life of approximately eight hours and is 100 times more potent as a serotonin antagonist than the parent compound.

Tropisetron

Tropisetron is an indoleacetic acid ester of tropine that possesses 5-HT_3 receptor antagonist activity. Intravenous tropisetron 2 mg in adults or 0.1 mg/kg in children may be effective against PONV[61-63]. It has a longer half-life than ondansetron but whether this translates to a clinical advantage remains unclear.

Other Drugs

The antiemetic mechanism of glucocorticoids (dexamethasone and methylprednisolone) is unknown. Besides dexamethasone's traditional use in chemotherapy-related emesis, it has also been used more recently as prophylaxis for PONV. When there is a high risk of postoperative nausea and vomiting, a single prophylactic dose of IV dexamethasone 8 or 10 mg, is antiemetic compared with placebo, without evidence of any clinically relevant toxicity in otherwise healthy patients, with late efficacy most pronounced[64]. Dexamethasone has antiemetic effects that are reportedly comparable with conventional antiemetic agents[65]. Antiemetic efficacy is better when it is used in combination with another antiemetic drug than when it is used as the sole agent[65].

NK_1 receptor antagonists represent another new class of antiemetics that are under study at the moment.

NK_1 receptors are abundant in the medullary areas where emetic inputs converge. Animal studies suggest that NK_1 receptor antagonists have a wide spectrum of antiemetic activity. It has been reported to be more effective than ondansetron for prophylaxis against PONV after gynaecologic surgery[66] and superior to placebo in the treatment of established PONV[67].

Combination Drug Therapy

Despite the many drugs available for PONV, there is no single drug that can claim to be the miracle cure for this deceptively simple problem. Combination drug therapy could be the answer since it is reasonable to postulate that different pharmacological classes of drugs, with different mechanisms of action, in combination should be more effective than single drugs alone in inhibiting the complex emetic reflex. Moreover, any enhanced antiemetic efficacy of combination drug therapy could result in the reduction of the dosing of the respective drugs, hence improving the side effect profile. Many combinations of antiemetic drugs have been tested with varying efficacy. The combination of dexamethasone with a serotonin receptor antagonist is superior to a serotonin receptor antagonist alone in preventing PONV[64,65]. The combination of droperidol with ondansetron has been reported to be more effective than either drug alone in preventing PONV[68-70] but some authors believe there is a lack of evidence to support this[71]. Other combinations like ondansetron and cyclizine[72], ondansetron and promethazine[73], droperidol and metoclopramide[74], dimenhydrinate and metoclopramide[75], dimenhydrinate and droperidol[76], have been tried with varying efficacy in preventing PONV.

NON-PHARMACOLOGIC METHODS

Non-pharmacologic methods have also been studied for their efficacy in PONV prevention. These include acupuncture, electroacupuncture, transcutaneous electrical nerve stimulation, acupoint stimulation, and acupressure. Lee and Done, in their meta-analysis, showed that nonpharmacologic techniques were equivalent to commonly used antiemetic drugs in preventing PONV in adults but not in children[77]. Supplemental oxygen has also been shown to have a protective effect against PONV[53,78]. The cost of newer antiemetic drugs and their possible side effects may warrant renewed interest and research in this area.

COST-EFFECTIVE MANAGEMENT OF PONV

With escalating health care costs and faced with a myriad of antiemetic drugs in use today, the anaesthetist's choice of antiemetic drug depends not only on its efficacy and safety profile, but also on its cost-effectiveness[79].

The cost-effectiveness of antiemetics depends on the effectiveness and cost of the drug, incidence of PONV in the hospital's population and whether the antiemetic is used for prophylaxis or treatment of established PONV. Some authors advocate the use of prophylactic antiemetic while others report that it does not improve outcome or patient satisfaction[46,49,68,80-85]. As the frequency of PONV decreases, it becomes less cost-effective to use prophylactic antiemetics. Prophylaxis with ondansetron has been reported to be cost-effective if the incidence of PONV exceeds 30 to 33%. Prophylactic droperidol is cost-effective if the incidence of PONV exceeds 10 to 13%[84,86]. Prophylaxis versus treatment with antiemetics remains controversial at present.

STRATEGY FOR EFFECTIVE MANAGEMENT OF PONV

Several authors have attempted to quantify the relative impact of risk factors on PONV[26,87,88] and set up risk models for its prediction[5-7,12,88]. Recently, risk scores for predicting PONV have been developed[6,7,12] and attempts made at cross-validation between centres to test their general applicability[7]. Apfel and Koivuranta each independently developed risk scores based mainly upon patient-related risk factors as the strongest predictors[6,12]. They then collaborated in a study of their risk scores by cross-validations between two centres and reported that risk scores derived from one centre were valid in the other, and could be simplified without significant loss of discriminating power. The four most important predictors of PONV included in their final simple risk score were female gender, prior history of PONV or motion sickness, non-smoking, and the use of postoperative opioids. If no or only one risk factor is present, the incidence of PONV may vary between 10% and 21%. If at least two risk factors are present, the incidence may rise to between 39% and 78%. They suggested that prophylactic antiemetic therapy be considered for patients with at least two out of four risk factors[7]. In their risk model, which included patient-, anaesthesia-, and surgery-related factors, Sinclair et al reported that patients' risk for PONV could be predicted according to their gender, age, smoking status, previous history of PONV or motion sickness, duration of anaesthesia, anaesthetic technique, and type of surgery[5].

Watcha proposed the following guidelines for the prophylaxis and therapy of PONV[85]. A low, mild, moderate, high, and extremely high risk for PONV is determined by the presence of none, one, two, three, or four of the following factors respectively: female gender, nonsmoker status, previous PONV or motion sickness, and opioid use[7]. For patients with a low risk for

PONV, no prophylaxis is recommended and if PONV occurs, ondansetron 1 mg or dolasetron 12.5 mg can be administered. For patients with a mild to moderate risk for PONV, he proposed prophylaxis with droperidol 1.25 mg, and rescue with ondansetron 1 mg or dolasetron 12.5 mg for breakthrough PONV. For those with a high risk for PONV, prophylaxis with droperidol 1.25 mg and a steroid plus an optional metoclopramide is suggested. Those with breakthrough PONV can be given ondansetron 1 mg or dolasetron 2.5 mg. Finally for those with an extremely high risk for PONV, he proposed prophylaxis with droperidol 1.25 mg with a steroid in combination with either ondansetron 8 mg or dolasetron 12.5 mg. Those with breakthrough PONV can be administered metoclopramide, phenothiazine, an additional dose of 5-HT_3 antagonist or another antiemetic. The choice and dosage of antiemetic drugs for prophylaxis and rescue are based on their efficacy, safety, and cost-effectiveness[85]. It may be reasonable to use a drug from a class other than the one used for prophylaxis for treating breakthrough PONV[49,89]. Additional doses of the same antiemetic may not be effective[82].

SUMMARY

PONV is one of the commonest complaints following anaesthesia, and can result in morbidity like wound dehiscence, bleeding, pulmonary aspiration of gastric contents, fluid and electrolyte disturbances, delayed hospital discharge, unexpected hospital admission, and decreased patient satisfaction. Despite the vast amount of research done in this field and the variety of antiemetic drugs available, PONV still has a high incidence. Knowledge of the risk factors of PONV can assist the anaesthetist in the judicious use of pharmacotherapy to ameliorate this problem, especially in the high-risk patient. The management of PONV requires a multi-modal approach which can include the use of less emetogenic anaesthetic techniques, balanced analgesia, appropriate intravenous hydration, the use of pharmacotherapy and possibly non-pharmacologic methods. Some suggested clinical guidelines for the prophylaxis and rescue of postoperative nausea and vomiting are given in Table II.

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