Bulletin of Environment, Pharmacology and Life Sciences
Bull. Env. Pharmacol. Life Sci., Vol 12 [7] June 2023: 311-315

Online ISSN 2277-1808
Journal's URL:http://www.bepls.com
CODEN: BEPLAD

REVIEW ARTICLE **OPEN ACCESS**

Post-Operative Nausea and Vomiting - A Conceptual Study

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ABSTRACT
The vomiting reflex developed as a protective mechanism and was primarily focused on the digestive system. It can also be a sign of a number of illnesses that affect the digestive system and other organs. Both surgeons and anaesthetists are challenged by the incidence of nausea in the postoperative period. Pre, Intra, and postoperative factors that contribute to postoperative nausea and vomiting should be avoided or managed in order to protect patients from its unpleasant effects. Postoperative nausea and vomiting can be prevented and treated using antiemetics, acupuncture, and other medications. Making postoperative nausea and vomiting as undesirable as postoperative pain should be a goal for those who care for patients during the recovery period.
Keywords: Vomiting, Post Operative, Antiemetics, Nausea

Received 12.04.2023 Revised 22.04.2023 Accepted 23.06.2023

INTRODUCTION

Following recent technical advancements that reduced anaesthetic-related mortality and life-threatening morbidity, postoperative pain management and emetic symptoms have come to the forefront of research. While it's true that postoperative pain management has received a lot of attention, postoperative nausea and vomiting have received less of it (PONV) [1,2]

In the natural world, nausea and vomiting are considered undesirable side effects from a therapeutic standpoint, yet they serve specialised purposes that help the animal survive. Vomiting commonly occurs after nausea. Typically, more people experience nausea than vomiting, however, vomiting itself can sometimes make people feel better. Although general anaesthesia for surgical operations has been linked to nausea [18] and vomiting, not all postoperative nausea and vomiting are brought on by anaesthetics. [1,3,4] Vomiting is a physiological process that can be used either as a therapeutic intervention or as a warning indicator. [1,4,5] The majority of the time, postoperative vomiting is an unintentional reaction to an aberrant stimulus that might be psychological, physiological, or pharmaceutical. However, persistent vomiting after surgery may indicate a significant issue that has to be addressed and not covered up with sedatives or antiemetic medication.

An urge to vomit or the fear of doing so are both symptoms of nausea and an unpleasant sensation in the throat and upper abdomen. It could be momentary or not, come before vomiting, or just happen by itself. Vomiting is the violent passage of the contents of the upper gastrointestinal system into the mouth. Retching and vomiting both entail the same muscle groups being active, but there is no evacuation of gastrointestinal contents. Regurgitation and oesophageal reflux are not the same as vomiting because neither one is as strong or includes the same muscle activity. [3,5]

PONV still happens frequently, which is unpleasant for patients and could be bad for their postoperative recovery [1,6] "The intraoperative or postoperative administration of opioids is the most frequent cause of postoperative nausea and vomiting. [6,8,9] Many patients considered PONV to be a particular anaesthesia problem. After general anaesthesia, PONV is prevalent, and prophylactic antiemetics medication has been advised for individuals at risk [10] "Even though emesis is now only sometimes caused by anaesthetics,[7]

some patients still have a higher risk of experiencing it after anaesthesia. Despite recent developments in contemporary anaesthesia, PONV knowledge and treatment have made only limited progress. [11]

PATHOPHYSIOLOGY

The three key components of the reflexive action of vomiting are emetic detectors, integrative mechanisms, and motor outputs.[3]

EMETIC DETECTORS

Since the primary goal of the vomiting reflex is to protect against unintentional poisoning from food, the gut must possess sensing mechanisms that can trigger the reflex.[5] The main nerve for detecting emetic impulses is the vagus. Chemoreceptors in the mucosa of the upper gut monitor several environmental factors, whereas mechanoreceptors in the muscular wall of the gut are activated by contraction and distension of the gut.[3] They react to irritations, mucosal stroking, acids, alkalis, hypertonic solutions, and temperatures.

AREA POSTREMA

It is one of the circumventricular organs of the brain and is situated outside both the blood-brain barrier and the cerebrospinal fluid-brain barrier in the caudal portion of the fourth ventricle, close to the region of the obex. The vomiting centre is activated when the chemoreceptor trigger zone for emesis cells in this region is stimulated.

VESTIBULAR SYSTEM

The vestibular labyrinthine system is essential for the induction of emesis by motion stimuli; so sudden head movement should be avoided after treatment to minimize any labyrinthine input to the vomiting centre. The movement of patients on trolleys after surgery may also induce vomiting, through this system [12]

HIGHER INFLUENCES

Instead of serving as primary detectors, inputs from the higher centres seem to play a facilitative role in adjusting the sensitivity of the brain stem emetic mechanism.

MISCELLANEOUS INPUTS

Unpleasant taste can induce vomiting, nausea and gagging can be evoked by mechanical stimulation of pharyngeal afferents to the brain stem in the glossopharyngeal nerve. In myocardial infarction, ventricular cardiac afferents may induce nausea and vomiting when activated

THE ORGANISATION OF THE VOMITING REFLEX

The motor elements of the reflex are mediated by both somatic and autonomic neurons.[5] These channels serve purposes other than emesis. Gastric relaxation for food storage is mediated by vagal innervations of the stomach. In a distinct pattern, these pathways are activated. It is possible to think about vomiting as a stereotyped motor programme requiring the coordination of the somatic and autonomic neural systems. [5,12] The term "vomiting centre" is frequently used to refer to the core mechanism that controls emesis. The motor components of the reflex are coordinated in the brain stem. Pre-ejection and ejection are two distinguishable stages of the vomiting reflex.

PRE-EJECTION

The feeling of nausea is a characteristic of the prodromal phase. There are some obvious indicators, such as cold salivation is mediated by parasympathetic nerves, whereas sweating, cutaneous vasoconstriction, and pupillary dilation are handled by sympathetic nerves. Other modifications include tachycardia and a decrease in stomach secretion. Via vagal afferent nerves stimulating postganglionic neurons in the stomach wall and employing vasoactive intestinal peptides or nitric acid as neurotransmitters, the proximal stomach relaxes right before ejection. [3]A retrograde massive contraction also occurs at the same moment, starting in the mid-small gut and moving towards the stomach. Acetylcholine serves as a neurotransmitter and is controlled by the vagal system.

EJECTION

Retching and vomiting are comprised of this, with vomiting being the only time when oral gastrointestinal contents are expelled. The somatic muscles of the belly and the diaphragm contract primarily during both retching and vomiting. The peri-oesophageal diaphragm relaxes during vomiting to promote the transit of

gastric contents into the oesophagus. During retching and vomiting, the abdominal muscles and the entire diaphragm spasm synchronously. The descending diaphragm compressing the stomach and the abdominal muscles contracting under the influence of somatic motor neurons induce the ejection of gastric contents.
Anaesthesia and surgery cause vomiting because some aspects of these procedures can trigger emetic detectors. PONV is probably multifaceted, with varying contributions from each element depending on the clinical condition, and it is unlikely that any one aspect of the surgical environment is the sole cause.[13] Food, psychological stress, and pre-operative surgical indications are some of the contributing factors. Except when ingested in great quantities, food is not an emetic stimulus, therefore the issue here is likely one of stimulus interaction or sensitization to one stimulus by another, in this example, food and the anaesthetic medication.
The forthcoming operation may induce stress in some patients and this can lead to the release of stress hormones like ACTH, growth hormone and prolactin, with possible involvement in PONV. [3,5] Anxious patients may swallow large quantities of air before the operation (aerophagy) producing distention of the upper gastrointestinal tract, thus contributing to the genesis of PONV [3]
PONV may be caused by postoperative events such as premedication and intubation. Analgesics and antiemetics are the two primary categories of precautionary medications. Morphine and pethidine, two regularly used analgesics, have been investigated concerning PONV. [8,9] In analgesic doses, these medicines reduce stomach emptying and make the emetic reflex more sensitive to labyrinthine stimulation. Increased 5-HT release from the small intestine and vasopressin (ADH) release from the posterior pituitary both contribute to PONV. [3,13] During intubation, stimulation of the brain stem-projecting pharyngeal mechanoreceptive afferents can cause the gagging reflex, which, if unchecked, can result in retching and vomiting.[3]
The anaesthetic and the surgery are intraoperative factors that contribute to PONV. The emesis of the anaesthetic drugs is influenced by the state of the anaesthesia itself.[11] The physical effects of the anaesthetics may contribute to PONV because the incidence of PONV is higher with volatile than with intravenous anaesthetics. Emesis can be brought on by abdominal distention and the activation of vagal and splanchnic afferents during manual breathing with a mask. By impairing gastrointestinal motility, anaesthetics may cause nausea and vomiting, which may be exacerbated by the effects of surgery on gut health.
Additionally, haemorrhage and surgical intervention may intensify the effects of anaesthesia on blood pressure. [3,11,13] During spinal anaesthesia, hypotension is more likely to result in nausea and vomiting. Increased intracranial pressure and an additional cause of emesis can result from the vasodilatory actions of halothane, enflurane, and isoflurane on cerebral blood vessels. The effects of surgery on PONV are split into two categories: those that apply to all surgeries generally, and those that apply just to certain kinds of surgeries where PONV is more likely to occur. Surgery, especially abdominal procedures involving considerable manipulation, anaesthetics, and other factors impair the motility of the gastrointestinal tract. Ophthalmic surgery, ear, nose, and throat surgery, and other surgical procedures are known to cause PONV frequently because they activate the oculoemetric reflex and vestibular afferent pathways, which are both related to motion sickness. Even though the intestine is not the target organ for the surgical operation, the intestine and the mesentery are subject to some displacement, manipulation, and traction during abdominal and gynaecological surgery.[12]
The procedures are signalled to the central nervous system via vagal and splanchnic afferents that enter the intestine. PONV usually lasts less than 24 hours,[12] and is most intense during the first 2 hours, although the precise pattern depends upon many factors. The factors already discussed above may occur in the postoperative period, especially if they have a long-time course or may initiate secondary processes with prolonged effects.[11]
Of all the medications used before surgery, morphine and other opioids have the highest risk of causing PONV. [3,9] By sensitising the vestibular system and preventing gastrointestinal motility, they have a direct emetic impact. As sedation naturally suppresses the emetic reflex, the anaesthesia recovery rate has an impact on the prevalence of PONV. PONV incidence has been linked to the usage of neostigmine to assist in the restoration of neuromuscular inhibition. [14] It is challenging to evaluate the role of pain from a mechanistic perspective in the development of PONV. The central nervous system is generally awoken or alerted by pain; thus, the patient may become awake enough to feel nausea produced by various inputs. Thus, pain does not cause nausea but merely facilitates its expression.

CONSEQUENCES OF PONV

PONV is unpleasant and unappealing. Having negative consequences that could raise morbidity and mortality is also connected to being upsetting to the patient and their caretakers. The results can be segmented into physical, metabolic, and psychological effects.[11]

PHYSICAL

Retching and vomiting are fairly violent (active) and intense physical acts, and may place some stress on certain structures particularly if prolonged. These include rib fractures, stomach herniation, muscular strain and exhaustion, and rupture of cutaneous arteries in the upper body. Oesophageal tears leading to mediastinitis and rupture resulting in haemorrhage (Mallory Weiss Syndrome) are also included. Along with bleeding and wound constriction at the surgical site, which may or may not result in the formation of haematomas, postoperative vomiting has been linked to increased intracranial and intraocular pressure as well as bleeding from skin flaps in cosmetic surgery. Vomiting can cause tension and strain on surgical wounds, which can make postoperative pain worse.[4] Aspiration of vomitus during the postoperative period causes cardiorespiratory reflexes to be triggered, which increases the risk of morbidity and mortality. [1,12]

METABOLIC

Dehydration, alkalemia, and fluid and electrolyte imbalances can result from frequent vomiting, especially in children. [1,3,13]

PSYCHOLOGICAL

A patient who occasionally experiences PONV might feel like having the same experience with subsequent operations. All of these effects could lead to a delayed hospital discharge, which would raise the system's cost burden because the patients might need more time in the recovery room, specialized care from nurses and doctors, and extra medications, intravenous fluids, and supplies.

In several large series, the incidence of PONV in the recovery room during the first 24 hours following anaesthesia has stayed fairly stable at between 10% and 30%. [16] Age, body habit, gender, delayed gastric emptying, anaesthetic history, motion sickness, anxiety, premedication, anaesthetic technique, medication, postoperative analgesia and regional blocks, as well as the kind and length of operation, all have an impact on PONV. [6,11,15]

MANAGEMENT OF PONV

Today, antiemetics is the mainstay of postoperative nausea and vomiting treatment (PONV). Antiemetics come in a variety of forms and are employed in the treatment of PONV. Among these are gastrointestinal prokinetic medications with antidopaminergic effects and butyrophenones, which have antiemetic qualities as a result of antidopaminergic effects.[20] Drugs including hyoscine, atropine, and certain antihistamine receptor type 1 antagonists exhibit a central anticholinergic effect, which is linked to antiemetic efficacy (e.g., cyclizine). It's possible that these medications' anticholinergic effects are what give them their antiemetic properties.[17]

Hyoscine and atropine are examples of anticholinergics that act both centrally and peripherally and have antiemetic properties. The production of a transdermal form of hyoscine is an effort to address the issues of its short half-life and dose-dependent negative effects.

Acupuncture has been used to prevent PONV, and in certain cases, it works well and has no negative side effects.

Since they are very efficient and have few adverse effects, 5-Hydroxytryptamine subtype 3 (5HT) receptor antagonists represent a significant advancement in the treatment of emesis. Although in research by Goll and colleagues, supplementing oxygen more effectively decreased the risk of PONV than ondansetron.[20] Despite the fact that oxygen is relatively cheap and risk-free, they advised against using it in developing nations. According to Wang and Colleagues, the steroid dexamethasone was superior to saline in reducing nausea and vomiting that were brought on by epidural morphine for post-caesarean analgesia. [21]

CONCLUSION

Patients suffer from postoperative nausea and vomiting. It is important to address the PONV-causing causes. Those who manage patients in the postoperative phase must make efforts to prevent or treat PONV in order to reduce morbidity and death from it.

Conflicts Of Interest

None declared.

Financial support

None declared.

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CITATION OF THIS ARTICLE

Aashik M Raju, Akashdeep A Meshram, post-operative nausea and vomiting - a conceptual study. Bull. Env. Pharmacol. Life Sci., Vol 12 [7] June 2023: 311-315