Postoperative nausea and vomiting

Yolande Squire

Ruth Spencer

Abstract

Postoperative nausea and vomiting (PONV) remains a common clinical problem that increases patient morbidity, healthcare costs and affects patient satisfaction. This article outlines the physiology, reviews the available drugs and suggests a structure using risk stratification that helps to plan sensible clinical management.

Keywords Antiemetics; nausea; risk stratification; vomiting

Royal College of Anaesthetists CPD Matrix: 1A02; 2A07

Learning objectives

After reading this article, you should be able to:

- understand the risk factors for PONV
- understand the efficacy of antiemetic drugs
- plan and deliver an anaesthetic that will reduce the risk of PONV
- manage persistent nausea and vomiting postoperatively

Postoperative nausea and vomiting (PONV) occurs in at least 30%[1] of operations. It is often one of the most feared side effects of surgery, even above pain. Although in most cases it is self-limiting, each episode of vomiting delays discharge from the recovery room and increases the risk of unplanned admission. It may also be associated with more severe complications including pulmonary aspiration, dehydration, electrolyte abnormalities, raised intracranial and intraocular pressures, wound dehiscence and oesophageal rupture.

The management of PONV involves risk stratification, prevention and treatment. Both pharmacological and non-pharmacological interventions may be used.

Definitions

Nausea is the sensation of needing to vomit, which may include activation of central, sympathetic and parasympathetic responses.

Vomiting is the involuntary oral expulsion of gastric contents via coordinated autonomic, gastrointestinal and respiratory system activity. It can be considered in two phases. In the pre-ejection phase, sympathetic activation causes tachypnoea, tachycardia, hypertension, sweating, pallor and hypersalivation. During the ejection phase, the epiglottis closes and forceful coordination of diaphragm, abdominal musculature and oesophagogastric constrictors leads to forceful expulsion of gastric and upper duodenal contents.

Pathophysiology of nausea and vomiting

The physiology of nausea and vomiting is complex. A good understanding of current knowledge helps explain the pharmacological targets and therapies detailed below. Two key areas of the brain are important in the action of vomiting: the vomiting centre and the chemoreceptor trigger zone (Figure 1).

The vomiting center (VC)

This lies in the lateral reticular formation of the medulla and receives afferent impulses via cranial nerves (CN) from the vestibulocochlear apparatus of the middle ear (CN VIII), carotid baroreceptor impulses (CN IX), gastrointestinal chemo and stretch receptors (CN X) and aortic baroreceptors (CN X). It also receives afferents from higher cortical centres involved in pain, anticipation, memory, sight and fear as well as spinal cord afferents from peripheral pain pathways.

The VC coordinates the smooth and striated muscles involved in vomiting via the 'special visceral efferent nerves', CN V, VII, IX, X and XI. These innervate the muscles of the face, neck and oropharynx in a coordinated fashion. Motor, sympathetic and parasympathetic outflow to the gastrointestinal tract and secretory organs are carried by the autonomic general visceral efferents of CN II, VII, IX and X. Finally, efferent branches from the VC travel via spinal nerves to the diaphragm and abdominal muscles.

The chemoreceptor trigger zone (CTZ)

This lies in the area postrema in the floor of the IVth ventricle. It is functionally outside the blood brain barrier and is sensitive to chemical stimulation via drugs and toxins present in the bloodstream. Its efferents act directly on the vomiting centre.

Neurotransmitters

A multitude of neurotransmitters are involved in the vomiting pathways. The important ones are shown in Table 1. Other transmitters such as neurokinin-1 (substance P) also play a role in the emetic reflex.

Risk factors for PONV

While multiple factors combine to increase the risk of PONV, the reason some patients suffer more than others remains difficult to predict. Risks are traditionally grouped into patient, surgical and anaesthetic factors (Table 2).

Patient factors

Although no single factor can predict PONV, strong predictors include female gender (which carries a threefold increased risk) and previous history. PONV demonstrates familial inheritance[2] possibly due to inherited genetic traits. Studies have found associations between PONV and specific genetic polymorphisms,

Yolande Squire *MBChB, BSc is a Clinical fellow at North Bristol NHS Trust, UK. Conflicts of interest: none declared.*

Ruth Spencer *MRCP FRCA is a Consultant Anaesthetist at North Bristol NHS Trust, UK. Conflicts of interest: none declared.*

Please cite this article in press as: Squire Y, Spencer R, Postoperative nausea and vomiting, Anaesthesia and intensive care medicine (2018), https://doi.org/10.1016/j.mpaic.2018.06.009

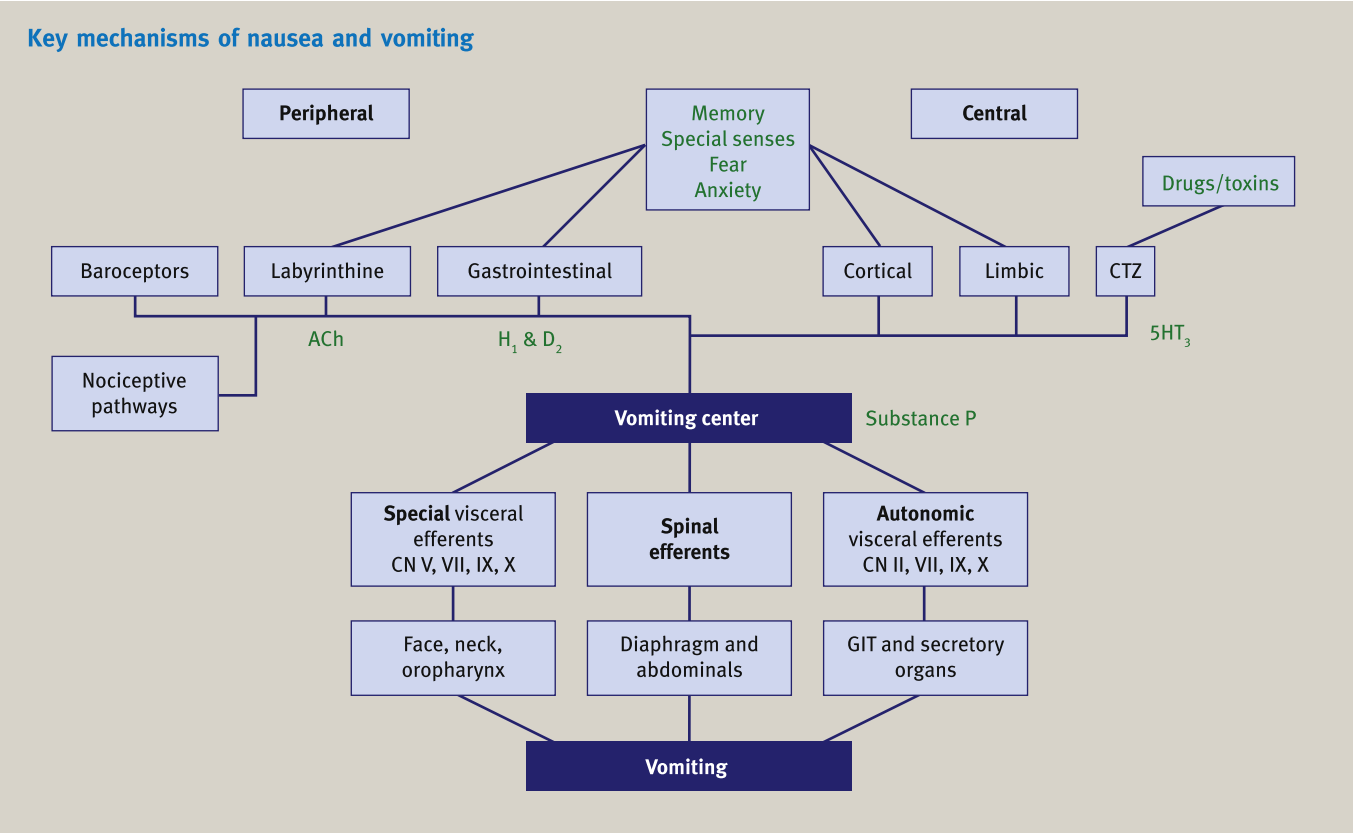

Figure 1

Neurotransmitters and receptors

Neurotransmitter	Receptor	Location
Histamine	H_1 receptors	Peripherally
Dopamine	D_2 receptors	Peripherally in GI tract & centrally
Serotonin	5-HT_3 receptors	Centrally
Acetylcholine	Muscarinic receptors	Vestibular apparatus

Table 1

such as certain single nucleotide traits of acetylcholine M3 subtype receptors (linked to motion sickness) as well cytochrome P450 genotypes responsible for rapid metabolism of many drugs and toxins.[3,4] These could act as valuable targets in the future for patient specific pharmacogenetic targets.

Smoking is strongly linked to a reduced risk of PONV. Although mechanisms are not fully understood this could be through a variety of effects including hepatic cytochrome p450 enzyme induction,[5] down regulation of the CTZ by recurrent exposure to emetogenic substances, or the presence of an antiemetic in cigarette smoke. Several studies investigating the role of nicotine patches in preventing PONV have not shown any benefit to this strategy and have even suggested the potential of nicotine therapy to increase the risk of vomiting.[6]

Patient age, although not included in any risk scoring systems, probably has an effect at least in statistical terms. The evidence for this is conflicting. Some studies report no impact of age on incidence of PONV, whereas others support the commonly held belief that incidence rises from birth to a peak around the time of puberty.[7] Risk then falls approximately 10% with each decade of life.[8]

Surgical factors

In adult and paediatric cohorts, the risk of PONV is directly related to length of surgery.

There is ongoing debate whether gynaecological surgery itself is a risk factor or the increased risk is related to the female patient group. In either event, these patients usually require a multimodal approach to symptoms.

Abdominal and laparoscopic procedures may be associated with PONV due to length of procedure rather than specific additional risk.

In ENT procedures the risk could be related to vestibulocochlear involvement, permissive hypotension and ingestion of blood and debris from oropharyngeal soiling. Ophthalmic surgery, in particular strabismus correction, is associated with increased PONV. This particularly affects the paediatric population.[9]

Anaesthetic factors

Gastric insufflation as a result of bag-mask ventilation leads to increased PONV. Overall there is a lower risk from spontaneous ventilation via a supraglottic airway than tracheal intubation.

The emetogenic capacity of volatile anaesthetics appears to be dose related, with longer procedures more likely to cause problems. The use of nitrous oxide further increases the risk. Total

Please cite this article in press as: Squire Y, Spencer R, Postoperative nausea and vomiting, Anaesthesia and intensive care medicine (2018), https://doi.org/10.1016/j.mpaic.2018.06.009