

REVIEW

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The neural mechanism and pathways underlying postoperative nausea and vomiting: a comprehensive review

Zhu Zhang^{1,2,3} and Xin Wang^{1,2,3*}

Abstract

This scoping review aims to synthesize evidence regarding the neural mechanisms and pathways underlying postoperative nausea and vomiting (PONV). We systematically searched the PubMed, Embase and Cochrane Library databases up to 2024. The findings indicate that PONV is a common complications after surgeries involving anaesthesia and can also lead to severe postoperative discomfort. Various surgical procedures and anaesthesia methods can trigger PONV, and the underlying neural mechanisms and signalling pathways are complex. It is currently assumed that these pathways do not exist independently of each other but that a central mode generator encompasses all relevant neurons and conducting nerve fibres, including the nucleus of the solitary tract, the dorsal nucleus of the tenth nerve, the area of the nerve, etc. After the information is integrated, it is transferred to the digestive tract, thereby causing PONV. In this article, we discuss the progress of research on the factors that influence PONV and identify the underlying pathways.

Keywords Anaesthesia, Postoperative nausea and vomiting, Inducement, Neural pathways

Background

Postoperative nausea and vomiting (PONV), which is one of the most common complications and side effects after surgeries involving anaesthesia, refers to nausea and vomiting symptoms that occur within 24–48 h after surgeries involving anaesthesia. PONV has a high incidence between 2 and 6 h postoperation. The prevalence of nausea and vomiting is ~ 20–30%, and in patients with high-risk factors, the prevalence is as high

as 56–92% and the incidence is as high as 30–56%. PONV can cause physical and mental discomfort, delayed recovery and prolonged hospital stays; thus, nausea and vomiting are among the focuses of research on rapid rehabilitation surgery [1]. Nausea and vomiting are also major causes of rapid rehabilitation surgery (RRS) [2]; they also increase healthcare costs and lead to a decrease in patient satisfaction. The study revealed that annual medical expenditures for the treatment of postoperative nausea and vomiting in the United States increased by \$253,270 to \$519,617 [1]. Currently, most studies generally assume that the independent risk factors for PONV are as follows: female sex, laparoscopic surgery, a history of PONV, a history of motion sickness, inhalation anaesthetics, and the use of opioids [3]. However, there are many other risk factors for PONV, including the neural mechanisms underlying its occurrence. Therefore, there is no consensus regarding its prevention.

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The physiological process of nausea and vomiting

Overview of nausea and vomiting

Nausea is a common clinical symptom that is a common precursor symptom of vomiting. However, in some cases, vomiting is not preceded by nausea. The onset of nausea is mediated by a complex reflex that, owing to the coordination of various muscles, causes discomfort and tightness in the epigastric region, which then leads to dry heaving or vomiting [4]. When the body responds to vomiting but has not yet excreted the stomach contents, the diaphragm and abdominal muscles contract and relax at the same time, the bottom of the stomach relaxes, and the intestines undergo strong retrograde contraction, which is also a form of routine gastric contraction. Intestinal reactions occur before vomiting. Sometimes, the final event is not expelling stomach contents, which is called dry heaving.

The vomiting response is commonly understood to involve expelling the contents of the stomach through the oesophagus and mouth. Vomiting occurs after nausea and is actually a protective reflex of the body. The vomiting process depends on the coordinated and continuous contraction of many smooth and somatic muscles. For example, the contraction of the abdominal muscles during vomiting is significantly longer than that of the diaphragm. The contraction time is greatly increased so that the stomach contents can pass through the diaphragm segment more easily. Vomiting can be divided into reflex vomiting (in which the body expels harmful substances from the body through vomiting), central vomiting (associated with certain diseases, such as meningitis), and vestibular disorder vomiting (caused by dysfunction of the vestibular system and sensory organ that influence the body's perception of spatial orientation and balance).

The factors influencing postoperative nausea and vomiting

Anaesthetic drug effects

Many different types of anaesthetics are commonly used in modern anaesthesia, such as inhalational anaesthetics (e.g., sevoflurane and isoflurane), sedatives, and opioid analgesics (e.g., sufentanil, fentanyl, and pethidine). Anaesthesia is considered one of the main triggers of PONV [5]. Many studies have shown that anaesthetics can cause nausea and vomiting when combined with certain protein targets. In addition, the mechanism of PONV may be related to the stimulation of the vomiting centre in the brainstem, which receives certain vomiting signals from the body. In this context, the use of anti-nausea and vomiting medications, such as ondansetron, granisetron (5-HT₃ receptor antagonist), and colestipol, represents a breakthrough [6]. The use of ERAS also

represents a breakthrough in this field. Owing to the implementation of ERAS at both the international and national levels, PONV has currently reached the same level as postoperative pain and requires special attention from anaesthesiologists. Therefore, new drugs and anaesthesia aids will be available now, thereby reducing the incidence of PONV, promoting the use of ERAS in surgical patients [7], increasing the turnover rate of patients in wards, reducing the hospital costs for each patient and enhancing patient comfort. Remimazolam, which is a new anaesthetic that is similar to midazolam, can bind to benzodiazepine receptors to form GABA–benzodiazepine complexes, thereby reducing dopamine activity and the release of 5-HT₃. These sequelae lead to a reduction in dopamine intake or adenosine intake in the medullary vomiting centre, in turn causing a reduction in adenosine-mediated synthesis as well as the release and synthesis of adenosine in the CTZ, which is located in the last region of the fourth ventricle. Adenosine-mediated synthesis, release and postsynaptic dopamine receptor activity in the terminal zone of the fourth ventricle are reduced [8, 9]. Therefore, increased use of Rima during the induction of anaesthesia may reduce the incidence of PONV. Rima reduces the use of sevoflurane, an inhalation anaesthetic; furthermore, Rima also reduced the nonhepatic and renal metabolism and half-life of sevoflurane [10]. Studies have shown that the use of 0.20–0.25 mg/kg remimazolam during anaesthesia induction can significantly reduce the incidence of PONV in surgical patients within 2–6 h after surgery.

Gender and age

Most research shows that women and young patients are more susceptible to PONV. Some studies have shown that patients older than 50 years have a much lower risk of PONV than younger patients [11]. Experimental data have shown that women have a much greater risk of developing PONV than men, even when the same type of surgery, the same method of anaesthesia induction, and the same anaesthesia management method are used; this sex difference is partially due to variance in estrin values [12]. Therefore, it is recommended that male patients can usually be examined without PONV prophylaxis, whereas female patients should generally be routinely administered prophylaxis before and during surgery, taking blood volume into account [13]. For example, severe hypotension usually occurs during a caesarean section, resulting in a significant reduction in visceral blood flow and the release of vomiting factors in the gastrointestinal tract, resulting in PONV [14]. Therefore, attention must be devoted to the patient's blood volume, and crystalloid or colloid fluids must be supplemented accordingly before and during surgery. In addition,

Stefanie Klenke demonstrated that genetic factors influence this risk of PONV. The polymorphism of the CHRM3 rs2165870 and KCNB2 rs349358 SNPs has a significant impact on the incidence of PONV; however, this result lacks relevant evidence in the flying white population [15].

Type of surgery

Some types of surgical procedures (e.g., laparoscopic surgery, especially weight loss surgery and breast surgery) are more likely to trigger PONV. This association may be related to the surgical site and size of the surgery. For example, the incidence of PONV is much greater in laparoscopic gynaecological procedures than in laparoscopic gastrointestinal procedures [16]. In particular, since gastrointestinal surgeries themselves stimulate the gastrointestinal tract, most gastrointestinal surgeries affect the tenth abdominal nerve, which is one of the most important peripheral afferent pathways for triggering PONV. Such surgeries can affect PONV through peripheral pathways and affect gastrointestinal motility [17]. When nausea occurs, the normal function of the stomach changes. Therefore, we speculate that altered gastrointestinal motility after stomach and small intestine surgical procedures could be the cause of nausea in these patients.

History of preoperative vomiting and movement disorders

Patients with a history of preoperative vomiting or movement disorders are more likely to experience PONV. This is relatively easy to understand, similar to genetic history, family history, etc. Patients who are more sensitive to nausea and vomiting are more likely to experience PONV.

Bowel obstruction and gastrointestinal dysfunction

Intestinal obstruction or premature feeding and drinking can easily cause food or liquid to accumulate in the delicate intestine, thus preventing the intestine from moving normally and increasing the risk of PONV. Especially after various types of operations, patients experience insufficient intake of potassium, sodium, chlorine, etc. Due to long-term abstinence from alcohol and fasting, electrolyte imbalances occur, which lead to PONV.

Postoperative pain

Postoperative pain can cause hypotension and hypoxia, which can lead to the onset of nausea and vomiting. Similarly, patients with supine hypotension syndrome after anaesthesia induction during a caesarean section may experience severe nausea and vomiting. In particular, if the surgical method is open abdominal

surgery, the patient will experience more pain as the size of the wound increases. A higher postoperative pain score also leads to a greater risk of developing PONV [18]. Therefore, patients receive more opioids for pain relief after surgery. However, the opioid dose is strongly correlated with the incidence of PONV [12]. The risk of PONV decreases when the dose of opioids is reduced by half.

Nausea- and vomiting-related scores

Since the risk of PONV is different for each patient, we can use PONV risk assessment to evaluate patients before and after surgery, thus applying individual anaesthesia methods and surgical methods to reduce postoperative complications. One commonly used evaluation method is the simplified Apfel scoring system [1, 19], which has four evaluation indicators, as shown in Table 1. This evaluation method varies greatly in actual application, so it is not a very accurate tool for stratifying PONV risk. However, this tool is based on patient data from outpatient surgeries and is, therefore, not suitable for inpatients.

The neural pathway mechanism of postoperative nausea and vomiting

It is generally accepted that all types of nausea and vomiting centres are located in the medulla oblongata of the brain and are composed of a series of interconnected exodus neurons. Furthermore, there is no isolated vomiting centre; rather, the various neurons are the neurons that are associated with the vomiting centre and that are loosely distributed in the medulla oblongata. When nausea and vomiting occur, the corresponding neurons are activated one after the other to be integrated into a central pattern generator (CPG). The medulla oblongata of the brain has two distinct functional areas in response to vomiting: the nerve reflex centre and the chemical receptor trigger zone. Impulses from the chemical receptor trigger zone are also transmitted to

Table 1 Apfel scoring system

Adult PONV brief risk score	
Risk factors	Score
Females	1 point
Nonsmokers	1 point
History of PONV	1 point
Postoperative use of opioids	1 point
aggregate	0–4 points

Description: The probability of PONV when the Apfel score is 0 is 10%; when the score is 1 point, the probability increases by 20%; if the Apfel score is 2, 3, and 4 points, the probability of PONV is 50%, 70% and 90%

the former for reception and processing [20]. Therefore, the vomiting reflex can consist of three parts: the receptor, central integration and motor efference, which are generally similar to the reflexive arc. The reflex arc consists of receptors (e.g., gastrointestinal chemoreceptors), afferent nerves, central nuclei (e.g., the nucleus tractus solitarius and parabrachial nucleus), efferent nerves, and effectors (e.g., the diaphragm and abdominal muscles). Vomiting requires coordinated activation of this pathway [21]. To put it simply, nausea and vomiting are also part of a special reflex arc.

Most nausea and vomiting symptoms are transmitted via the abovementioned route to the vestibular system and to the solitary nucleus before the route of transmission is reached.

Receptors and afferent nerves

As shown in Fig. 1, factors such as emetic agents in the blood and intestinal lumen contents and the gastric tone act can stimulate the corresponding receptor to complete the first step in the onset of nausea and vomiting. However, different stimulating factors are also uploaded via different routes. The afferent vagus nerve of the abdomen is a mechanical stimulation receptor that primarily senses the vomiting stimulus [22]. If there is a pulling stimulus in the gastrointestinal tract, the tenth nerve will capture it sharply and forwards it upwards. As chemical stimulus receptors, chemically sensitive receptors primarily perceive various irritating factors in the blood and in the digestive tract as well as some substances that are harmful to the organism.

The vestibular system consists of the brain centre (in the brainstem) and the external vestibular organs (in

the ear). The peripheral vestibular apparatus consists of the external, superior and posterior semicircular canals; the ellipsoid capsule; and the bulb capsule. As a functional organ for maintaining balance, the peripheral vestibular receptor can receive motion-related sensory inputs from the vestibule of the inner ear and sense the body as well as body movements and head position to maintain body balance [23]. Studies have shown that changes in head and body alignment can impact the autonomic nervous system via the vestibular system. For example, head rotation can stimulate the nucleus solitarius and the cephalic ventral medullary reticular system, which is caused by the downwards action of the autonomic brainstem nerves. In addition, nausea and vomiting caused by motion sickness are strongly related to the vestibular system [24]. In other words, the vestibular organs are generally considered receptors for vomiting caused by exercise.

Gastrointestinal vagal afferent fibres: gastrointestinal vagal afferent fibres can dominate the stomach and intestines, such as the release of 5-hydroxytryptamine from intestinal endocrine cells that sense circulating drugs or local toxins in the gastrointestinal tract. When the stomach lining is irritated or damaged, enterochromaffin cells in the intestine can be stimulated to release substances, such as 5-HT or neurotransmitters. Most of them are projected onto a single core. The relevant receptors of the tenth gastrointestinal nerve in the fibres are combined with neurotransmitters to transmit the signal to the dorsal brainstem, with some signals projecting to the posterior zone, causing nausea and vomiting [25].

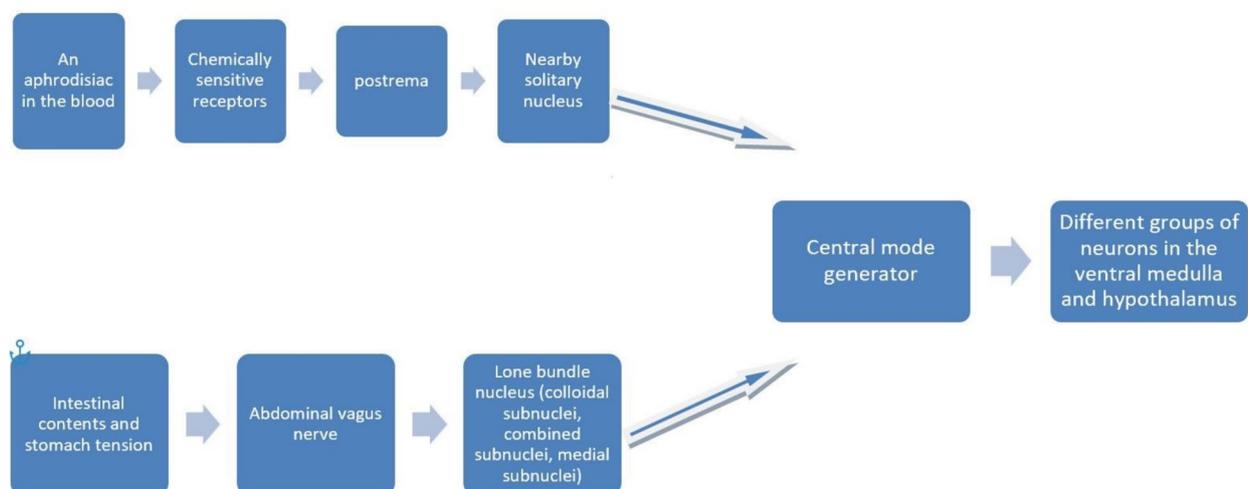


Fig. 1 Neurological reflex pathways of nausea and vomiting

The last zone (postrema) is located at the floor of the fourth ventricle, on the back of the medulla oblongata and above the solitary bundle nucleus. The final area includes the vomiting reflex centre and the chemoreceptor trigger zone (CTZ); together with the medulla oblongata and vomiting centre, these regions are involved in the control of nausea and vomiting, thus causing central vomiting. Since this area does not have good protection from the blood–brain barrier, some scientists even consider that it lacks a blood–brain barrier, thus increasing the vulnerability of local neurons and glial cells to various chemical stimuli, such as gastrin and angiotensin. At this point, intractable belching, nausea, vomiting and other digestive symptoms may occur [26]. Therefore, this phenomenon is called medulla oblongata syndrome by some, in which the above clinical manifestations usually appear as the first symptom.

The cerebral cortex, thalamus and hypothalamus: activation of the cerebral cortex and the thalamic and hypothalamic regions triggers mental vomiting as well as vomiting induced by visual or olfactory stimulation [27]. When some patients are in a state of stress, fear or anxiety, or suffering from tremendous psychological and mental stimulation, the sensory organs transmit this information to the cerebral cortex, and through stimulation of the cerebral cortex, the information is transmitted to the medulla vomiting centre, which triggers vomiting.

Nerve centre

The nerve centre, which is also known as the vomiting centre in the reflex arc, includes the nucleus of the solitary tract, the dorsal nucleus of the tenth nerve, the area of the nerve, etc. [27]. The various signals uploaded to the centre are output to the effector after integration and adaptation, causing the body to trigger the corresponding activities.

Efferent nerves and effectors

When the nerve centre has finished integrating various sensory stimuli, the vomiting signal is transmitted downwards. The effector, i.e., our digestive system, receives the signal from the transmitted nerves and produces a neuromuscular response that results in extreme relaxation of the stomach. The intestines then experience a tremendous retrograde contraction that produces rhythmic movement in the gastrointestinal tract, while the posterior abdominal muscles continue to contract strongly. The cardia expands, and the involuntary inhalation of the body close the anorexia. At this point, the vomiting precursors have occurred, thus leading to vomiting. However, some stimuli continue

to act only in the vomiting centre, leading to nausea or increased oral secretions.

Hierarchical integration of PONV pathways

Peripheral triggers: vagal afferents, gut enterochromaffin cells, and 5-HT₃ receptor signalling.

Central integration: nucleus tractus solitarius (NTS) → parabrachial nucleus (PBN) → insular cortex connectivity.

Efferent output: dorsal motor nucleus of the vagus (DMV) and retrograde giant contraction (RGC) coordination.

Treatment of postoperative nausea and vomiting

Use of anti-nausea and vomiting medications

When identifying appropriate anti-nausea and vomiting medications, it is important to consider a patient's sufficient blood volume [28]. However, antiemetics may cause dizziness, headache, constipation, sedation, sleepiness, and additional severe reactions. When PONV occurs, targeted medications, such as 5-HT receptor antagonists and dopamine D₂ receptor antagonists, can be used for treatment. Ondansetron is the most commonly studied 5-HT receptor antagonist, and experimental data have shown that ondansetron is more effective than metoclopramide and dexmedetomidine. Furthermore, the incidence of PONV is lower among patients who take granisetron than among patients who take ondansetron. However, its use of granisetron requires comprehensive consideration of various factors, such as the patient's economic situation. In addition, the use of hormonal drugs has gradually increased. For example, intravenous injection of 8 to 10 mg dexamethasone during the induction of general anaesthesia can reduce PONV within 24 h. Some studies have reported that high-dose corticosteroids (> 15 mg) are more effective than low-dose corticosteroids [29]. Dexamethasone can reduce the need for rescue antiemetics for up to 72 h. However, it is also important to be aware of the side effects of these medications, the vast majority of which are negligible at PONV doses. There are many complications associated with dexamethasone, such as increased risks of infection, delayed wound healing and hyperglycaemia and related complications. Therefore, the side effects of dexamethasone should not be underestimated. 5-HT receptor blockers can prolong the QT interval and exert negative effects on the cardiovascular system, thus increasing the risk of malignant cardiac arrhythmias and sudden cardiac death in patients; furthermore, repeated administration of these agents may lead to receptor tachyphylaxis, thus diminishing efficacy in chronic settings [30]. At present, many new drugs and compound preparations are being

examined in clinical trials. Among them, the natorpitam-palonostron compound preparation (NK1/5-HT3 double antagonism) has exhibited a 24-h PONV control rate of 85%. As a 5-HT3/D2 receptor double antagonist, olanzapine can be used as an anti-PONV agent [31]. In summary, when anti-vomiting medications are used, it is best to use different medications depending on a patient's individual risk factors. However, there is no research on which combination of prevention measures can achieve the best results, and further research is needed [32].

Nonpharmacological strategies

Aromatherapy

Aromatherapy is a complementary therapeutic approach that uses volatile plant extracts (essential oils) to promote physiological and psychological well-being. Postoperative nausea and vomiting (PONV) management primarily involves the inhalation of essential oils (e.g., peppermint, ginger [33], or isopropyl alcohol) [34] to modulate the olfactory system and gastrointestinal neural pathways. Aromatherapy has emerged as a nonpharmacological adjunct for PONV. The inhalation of essential oils (e.g., peppermint or ginger) may modulate nausea through two pathways: (1) direct olfactory stimulation, which suppresses the vomiting centre in the medulla and (2) local absorption of active compounds (e.g., menthol), which act on gastric 5-HT3 receptors [33]. While Cochrane reviews have reported only modest evidence regarding the efficacy of aromatherapy [31], its low cost and safety profile justify its clinical consideration.

Hydration

Hydration in the perioperative context refers to the strategic administration of intravenous or oral fluids to maintain physiological fluid balance [35], optimize tissue perfusion, and prevent hypovolemia. Adequate hydration has been shown to be associated with a reduced risk of PONV, and the underlying mechanism likely involves stabilizing haemodynamics and minimizing gastrointestinal hypoperfusion-induced nausea [1].

Perioperative hydration plays a critical role in preventing PONV. Hypovolemia-induced splanchnic vasoconstriction may trigger nausea via gut ischaemia and serotonin release. Current guidelines recommend balanced crystalloid administration (20–30 mL/kg) to maintain adequate perfusion, although excessive fluids should be avoided due to pulmonary complications [1]. Meta-analyses suggest that this approach reduces the incidence of PONV by 20–30% [35].

Acupressure and acupuncture

Noninvasive acupressure at the PC6 (Neiguan) point has been shown to effectively reduce the incidence of PONV

by ~ 30% [36], comparable to first-line antiemetics, such as ondansetron. The mechanism underlying this effect may involve vagal modulation and endogenous opioid release, which suppress the chemoreceptor trigger zone in the area postrema. In contrast, traditional acupuncture requires trained practitioners to insert needles at specific sites (e.g., PC6 and ST36), potentially enhancing effects through sustained neurohumoural regulation [37]. Despite heterogeneity in trial protocols, recent meta-analyses support the adjunctive roles of acupressure and acupuncture, particularly in high-risk patients [1].

Lifestyle and dietary factors

Emerging evidence has revealed the role of preoperative behavioural interventions in preventing PONV. Anxiety reduction via guided imagery or medication (e.g., benzodiazepines) may suppress HPA axis hyperactivity, thereby decreasing stress-induced nausea [37]. Smokers exhibit a lower incidence of PONV, possibly because of the antidopaminergic effects of nicotine; however, smoking should not be encouraged [5]. Dietary strategies for preventing PONV include oral intake of 1–2 g of ginger preoperatively, which inhibits prostaglandin synthesis and gut serotonin release [38, 39].

Carbohydrate-rich clear liquids can also be administered up to 2 h before surgery to maintain gastric motility and reduce fasting-induced discomfort [40].

Postoperative intake of irritants such as fatty/spicy foods may exacerbate nausea via vagal irritation [37].

Comparative efficacy of PONV prevention strategies

Effective prevention of postoperative nausea and vomiting (PONV) relies on risk stratification and multimodal approaches. Key strategies include:

Pharmacologic agents

5-HT3 antagonists (e.g., ondansetron): first-line for early PONV, minimal sedation. Dexamethasone: enhances efficacy when combined with 5-HT3 antagonists, effective for delayed PONV. NK1 antagonists (e.g., aprepitant): superior for delayed symptoms but higher cost. Dopamine antagonists (e.g., droperidol): effective but limited by sedation/extrapyramidal risks.

Non-pharmacologic methods

Acupuncture/acupressure (e.g., P6 stimulation): mild efficacy, suitable as adjunct therapy. Combination therapy: multimodal regimens (e.g., 5-HT3 antagonist + dexamethasone) demonstrate synergistic effects, reducing PONV incidence by 50–80% in high-risk patients versus monotherapy. Risk-based tailoring: guidelines recommend ≥ 2 interventions for moderate/

Table 2 Comparative efficacy of PONV prevention strategies [1, 33, 41]

Intervention strategy	RRR (%)	NNT	Common adverse reactions	Level of evidence
5-HT3 antagonist	25–30	4–5	Headache, constipation	Ia (Cochrane)
Dexamethasone	20–25	5–6	High blood sugar and insomnia	Ia
Acupressure (PC6)	15–20	6–8	Local skin irritation	Ib(RCT)
Isopropanol infusion	30–35	3–4	Hypotension, injection pain	Ia

high-risk patients. Emerging evidence supports aprepitant or low-dose naloxone for refractory cases.

Overall, a patient-specific approach balancing efficacy, safety, and cost optimizes outcomes (Table 2).

Future directions

While current therapies mitigate the risk of PONV in many patients, critical gaps in knowledge remain. First, GWAS cohorts are urgently needed to map genetic variants (e.g., 5-HT3R SNPs) linked to PONV susceptibility and drug resistance [42]. Second, AI models that integrate real-time EEG entropy and surgical stress biomarkers could enable dynamic risk prediction [43].

Summary

PONV is a common complication that occurs after surgeries involving anaesthesia. PONV affects postoperative recovery and quality of life. This in-depth study of the underlying aetiology, neural mechanisms and pathways of PONV has important reference value for clinicians and can help to reduce the incidence of PONV. Although existing research results and guidelines offer numerous prevention and treatment methods, their practical application is often limited by individual differences and differences in the types of surgery. There have been no significant advances in knowledge on the neurological mechanisms and pathways underlying PONV, thus limiting the development of preventive strategies. Therefore, patients may still experience varying degrees of nausea and vomiting. Future research should further clarify the neural pathways involved in PONV to formulate more effective preventive measures, thereby reducing the occurrence of PONV in the clinic and improving patients' treatment experience.

Abbreviations

PONV	Postoperative nausea and vomiting
ERAS	Rapid rehabilitation surgery
5-HT3	5-Hydroxytryptamine type 3 receptor
CTZ	Chemoreceptor Trigger Zone
NTS	Nucleus tractus solitarius
PBN	Parabrachial nucleus
DMV	Dorsal motor nucleus of the vagus
RGC	Retrograde giant contraction

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Author contributions

ZZ collects data and writes the first draft. It is the main contributor to the manuscript. XW is responsible for researching concept generation, review and revision and fund acquisition. All authors have read and approved the final manuscript.

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Consent for publication

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Competing interests

The authors declare no competing interests.

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