

Postoperative Nausea and Vomiting: A Review of updated Guidelines

AN EVIDENCE-BASED GUIDE FOR ANESTHESIA PROVIDERS

INCORPORATING THE FIFTH CONSENSUS GUIDELINES (GAN ET AL., 2026), AANA, AND ASA STATEMENTS

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Presenter Disclosures

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Off-Label Content: Any discussion of unapproved or off-label medication dosages will be explicitly highlighted as such during this presentation.

Independent Delivery: All recommendations are derived strictly from independent, peer-reviewed clinical data and consensus statements.

objectives

Discuss

Discuss the etiology and effects of postoperative nausea and vomiting



Define

Define the common causes of PONV



Discuss

Discuss current screening and treatment measures



Review

Review current updates and position statements

The True Burden of PONV



General Incidence: Affects roughly 20% to 30% of all surgical patients globally.



High-Risk Incidence: Reaches up to 70% to 80% in highly susceptible patient cohorts without active prophylaxis.



Patient Satisfaction: Consistently ranked by patients as more distressing than acute postoperative surgical pain.

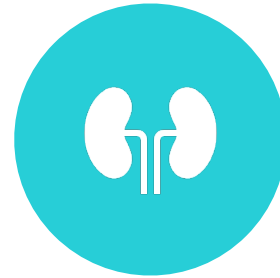


Economic Impact: Leading cause of delayed discharge from the PACU and a top cause of unanticipated ambulatory hospital admissions.

Mechanical & Surgical Complications of Emesis



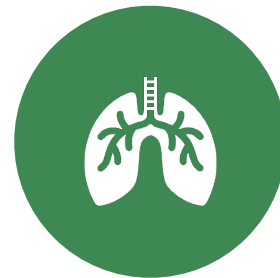
Wound Dehiscence: Forceful contraction of abdominal walls puts high mechanical stress on delicate suture lines.



Surgical Bleeding: Transient spikes in venous pressure during retching can destroy vascular bridges and cause hematomas.



Esophageal Tear: Violent emesis risks causing Mallory-Weiss tears or catastrophic esophageal rupture (Boerhaave's Syndrome).



Airway Risks: Severe threats of aspiration pneumonia, especially in patients emerging with compromised airway protective reflexes.

Associated risks of vomiting

aspiration of gastric
content

increased morbidity
after ocular, tympanic,
and intracranial
procedures.

Can cause elevated hr
and bp; or bradycardia
and hypotension

Risk for mi and
dysrhythmias

Gagging and retching

Parasympathetic
response

Macroeconomic & Throughput Impacts

PACU Stalls: A single emetic episode delays patient discharge from recovery units by an average of 25 to 60 minutes.

Staffing Utilization:
Demands higher 1:1 nursing consumption and increases clinical overhead metrics.

Ambulatory Overheads:
Triggers preventable overnight admissions, driving up facility operational overhead expenditures.

Initiation of the vomiting cascade

Complex interaction of receptors, chemicals and organ systems

Mediated via vomiting center in the dorsal area of the reticular formation of the medulla

Info is transmitted to the VC via several different areas through a variety of pathways

PONV: A Problem

Emesis is the postoperative outcome least preferred by patients

Postoperative Outcomes Least Preferred by Patients

Rank	Postoperative Outcomes
1	Vomiting
2	Gagging on endotracheal tube
3	Incisional pain
4	Nausea
5	Recall without pain
6	Residual weakness
7	Shivering
8	Sore throat
9	Somnolence

a survey of adult patients (N=101) conducted at Stanford University Medical Center. Patients were eligible if they were scheduled to undergo surgery at the center. Patients were asked to rank-order 10 possible postoperative outcomes from most to least desirable. F -test <0.01 .

W. A. et al. *Anesth Analg*. 1999;89:652-658. © 1999. With permission from Lippincott Williams & Wilkins.

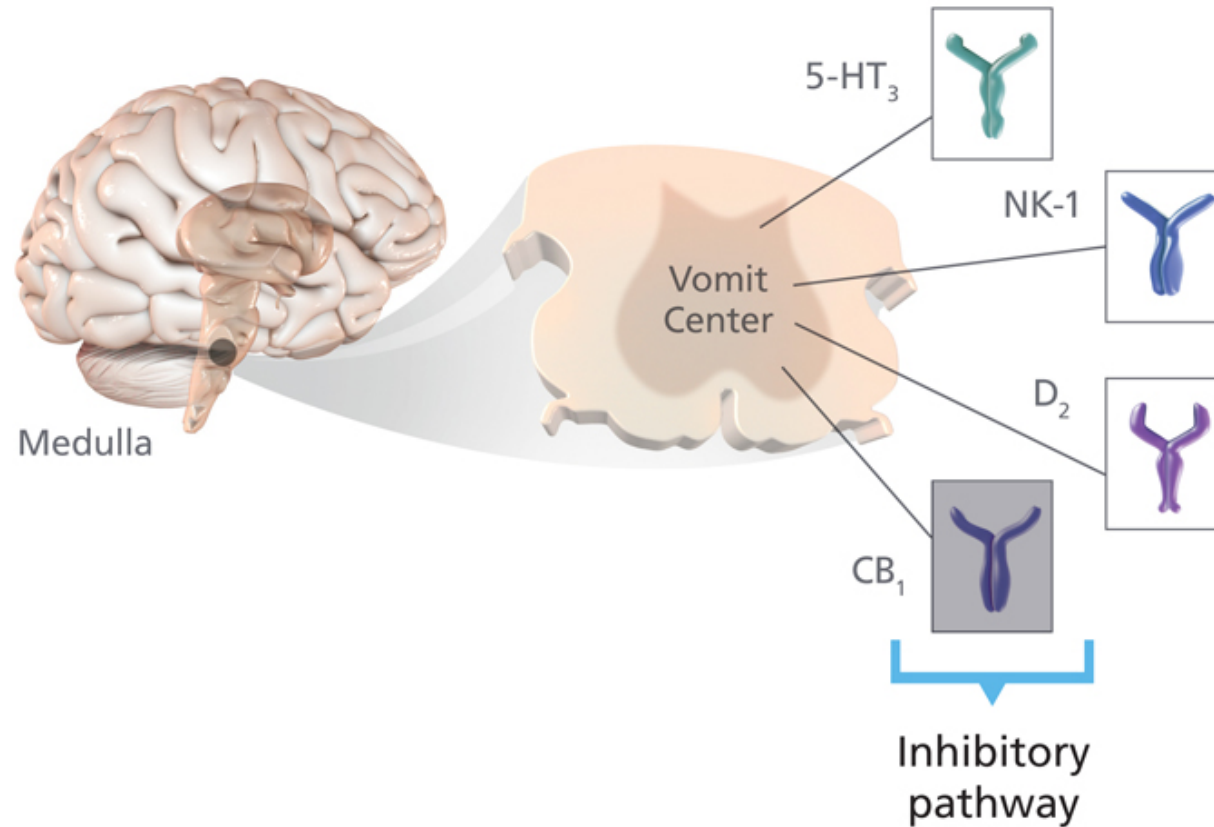
The facts

More than 25% of all surgical patients nationwide report an incidence of ponv

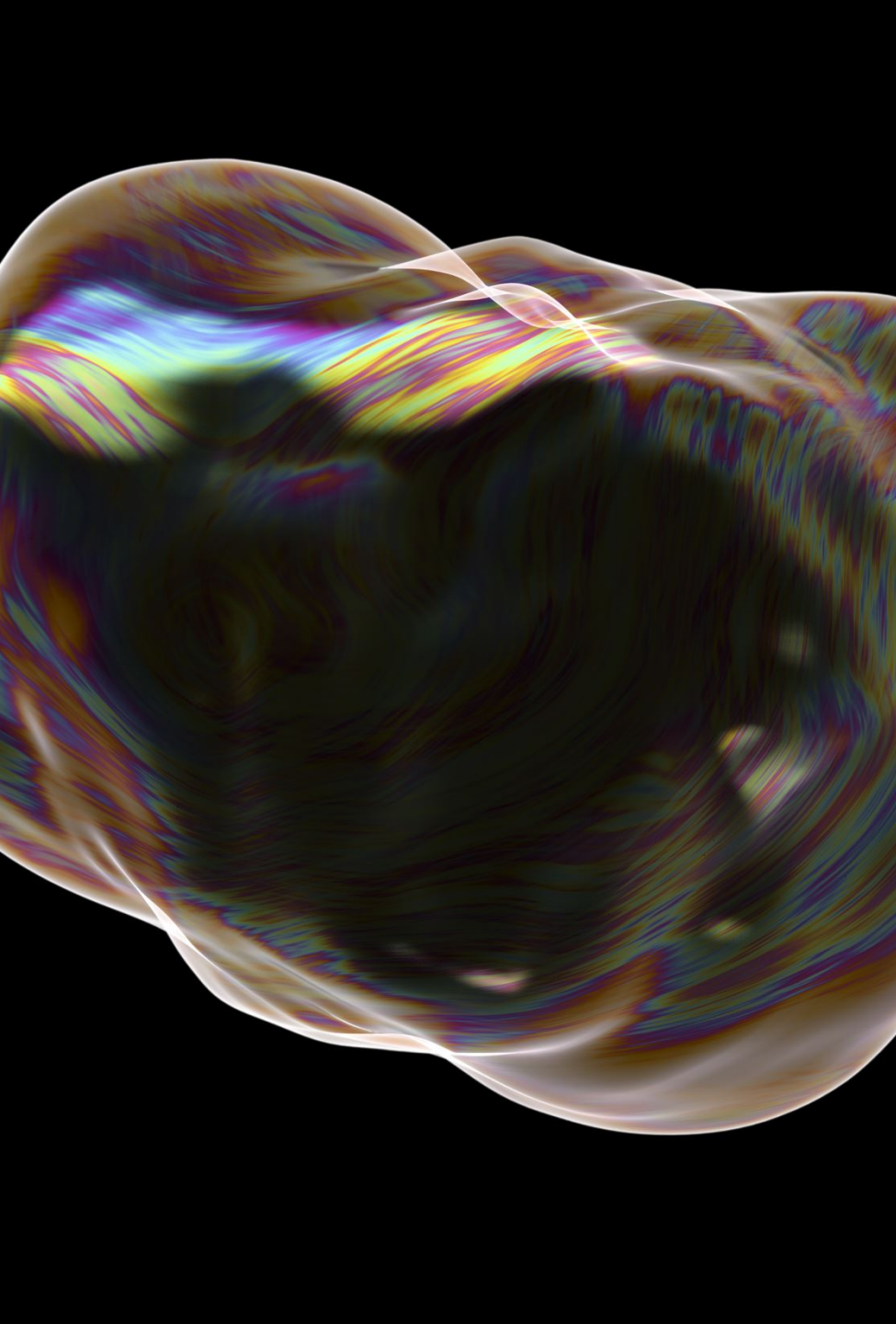
Greater than 80% in high risk population

Vomiting center

Located in the Medulla



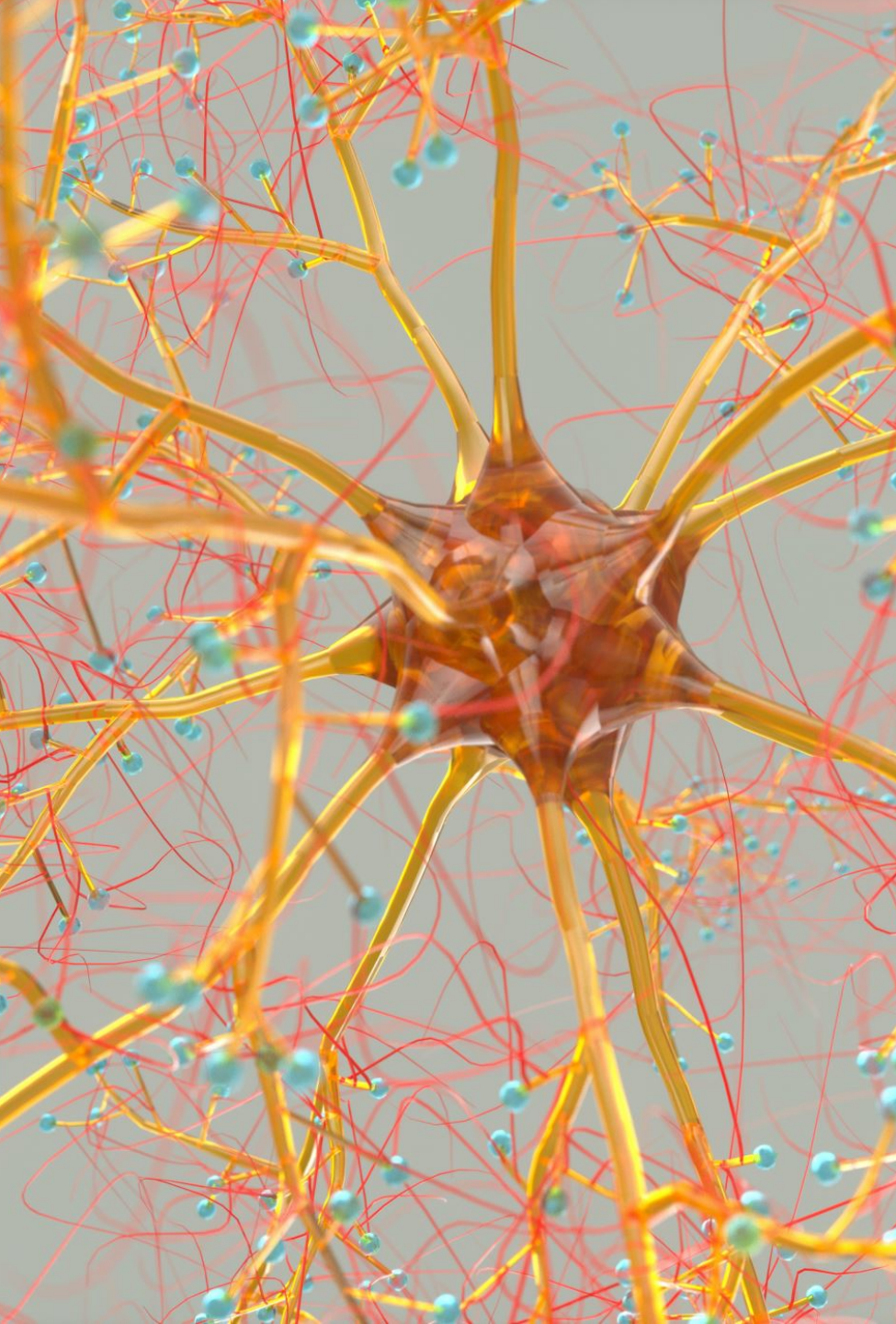
- Common sensations:
 - Tickling the back of the Throat
 - Gastric Distension
 - Vestibular Stimulation (Etoh)
 - Cerebral Cortex



Vomiting center

Composed of 3 nuclei

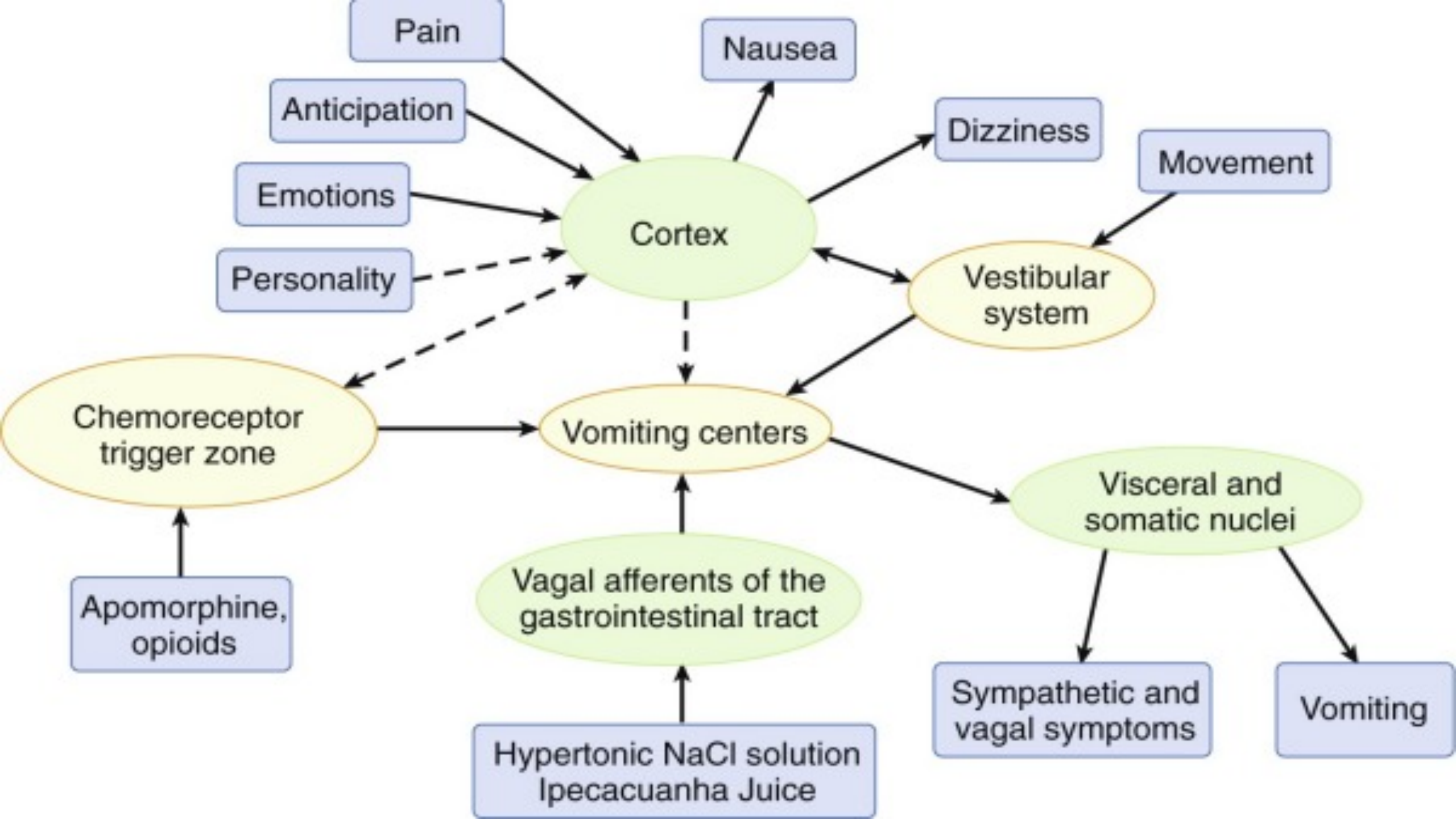
- Nucleus tractus solitaries (receives sensory info from afferent pathways)
- Dorsal motor nucleus of the vagus
- Nucleus ambiguus (involved in motor activity during vomiting)



Afferent pathways

5 primary afferent pathways:

1. Chemoreceptor trigger zone
2. Vagal mucosal pathway in gi system
3. Neuronal pathways from vestibular system
4. Reflex afferent pathways from cerebral cortex
5. Midbrain afferents



How is post op n/v triggered?

VIA 3 SEPARATE “SIGNALS”

First signal

Originates in the cns

Histamine and ach are the primary neurotransmitters

How to block transmission?

- Anticholinergic agents that block binding of ach (scopolamine)
- Benadryl (antihistamine)
- Promethazine and Haldol (MOA not understood)

Increased sensitivity

Anesthetic agents and opioids increase sensitivity of the vestibular center

Rapid position change

- Move patient slowly
- Having eyes closed can help

2nd signal- “A gut feeling”

Gi tract via vagus nerve

Gi distention and manipulation stimulates mechanoreceptors in wall of the gut releasing serotonin

How do I block this?

- Administer selective serotonin antagonist which decreases visceral info carried from gi tract to the vomiting center

the 3rd signal- “in the Zone”

Crtz located on the 4th ventricle in the brain

Fully formed by age 2

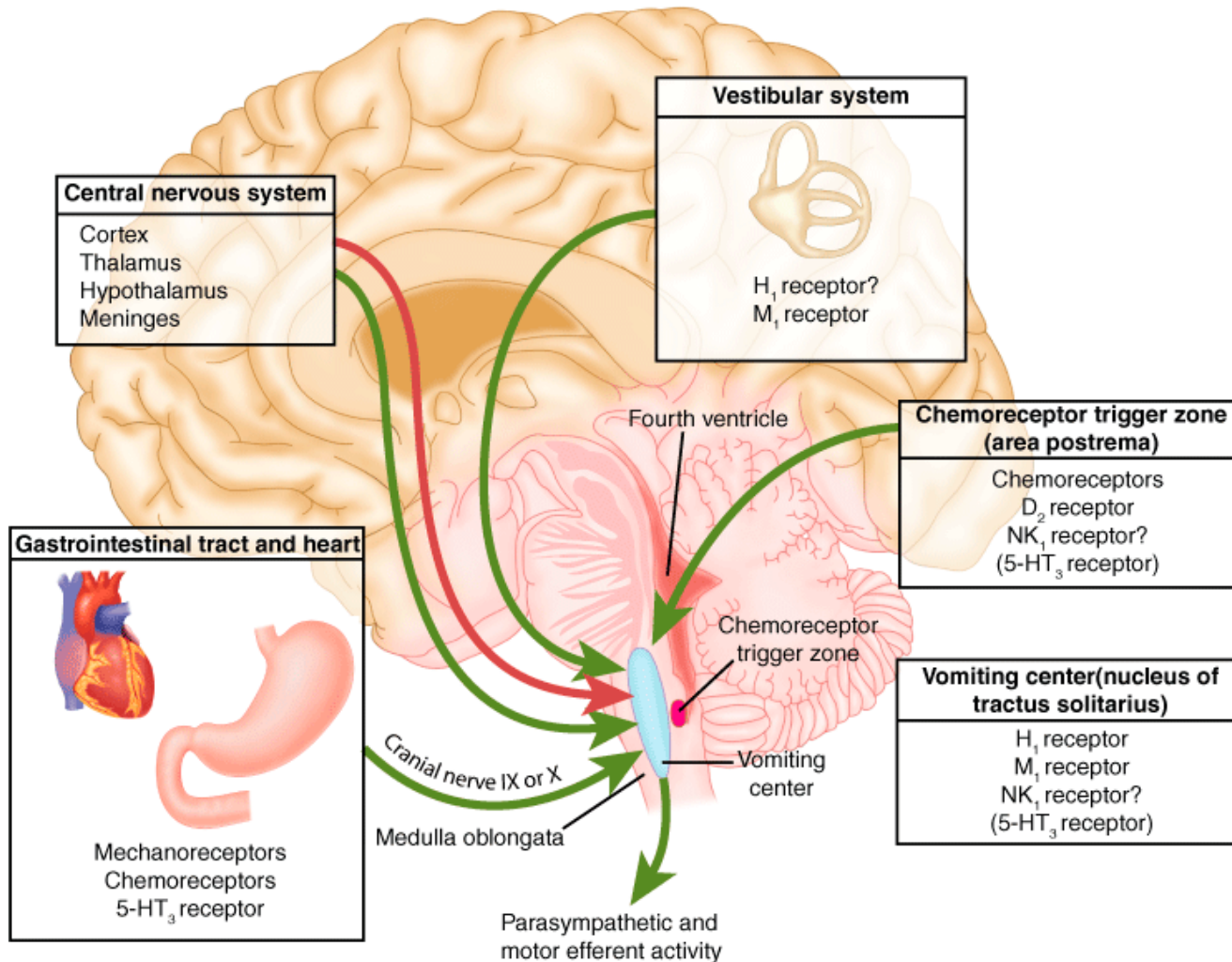
Close association with CSF

Large blood supply not protected by blood brain barrier

Detects presence of drugs or toxins in blood

Responsible for chemo associated nausea and vomiting

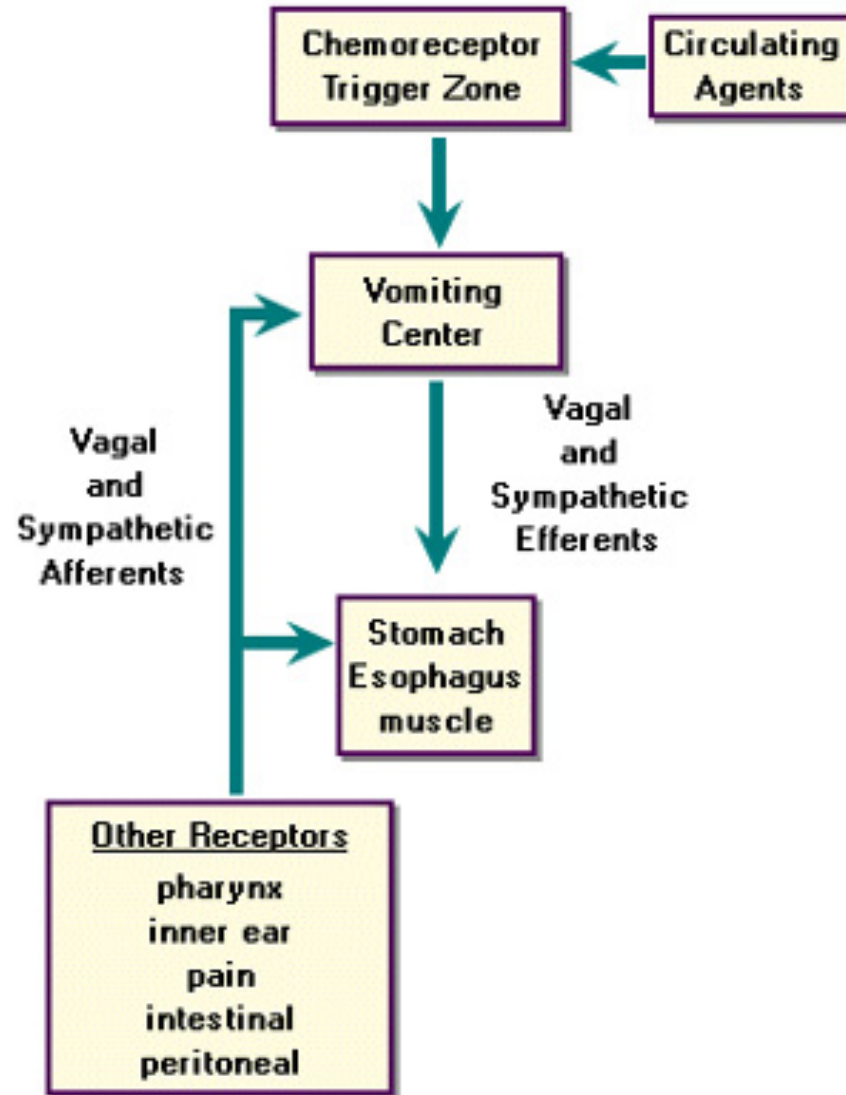
Chemoreceptor trigger zone



- Outside of the Blood brain barrier
- communicates with the vomiting center
- Triggered by Blood Borne Drugs or Hormones

Source: Katzung BG, Masters SB, Trevor AJ: *Basic & Clinical Pharmacology*, 11th Edition: <http://www.accessmedicine.com>

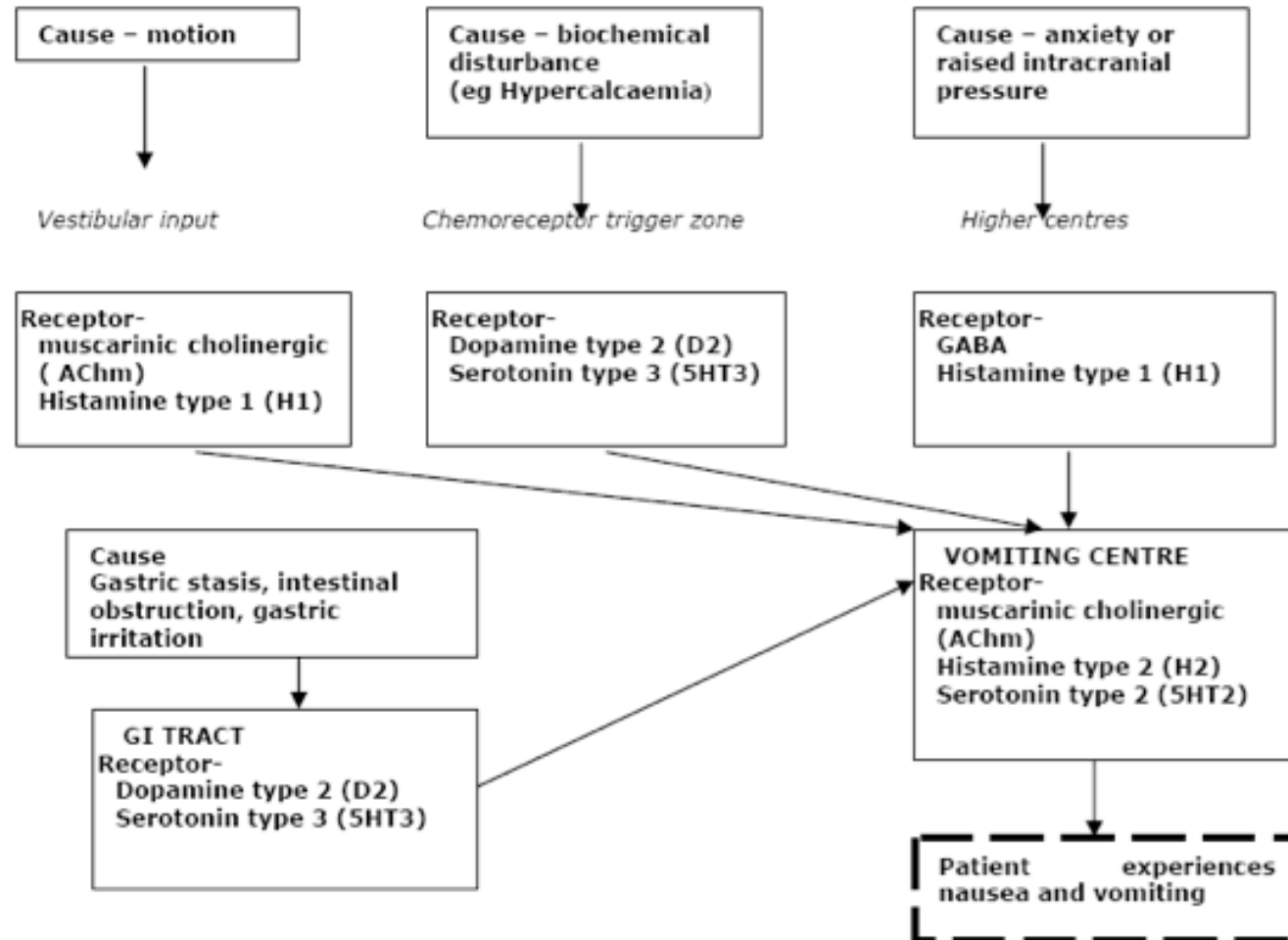
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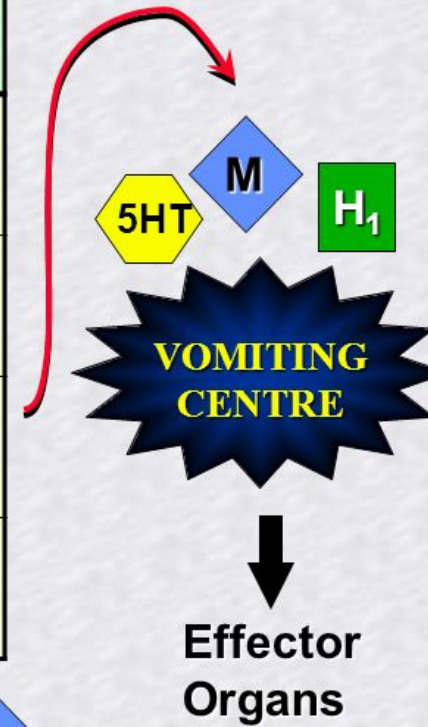
crtz

Neurotransmitters involved:

- Dopamine
- 5-HT
- Substance P
- Ach
- histamine



Stimulus	Area	Receptors
Drugs, Metabolic	Chemoreceptor trigger zone	D ₂ 5HT
Motion, Position	Vestibular	M H ₁
Visceral	Abdominal organs	D ₂ 5HT
↑ ICP	Cerebral cortex	H ₁



 **D₂**
 Dopamine
  **5HT**
 Serotonin
  **H₁**
 Histamine
  **M**
 Muscarinic

Anatomy of the Emetic Reflex

Central Coordination:
Orchestrated by the Central
Pattern Generator located
within the brainstem.

Afferent Highways:
Integrates high-speed
signals originating from the
central nervous system, gut
wall, and inner ear balance
centers.

Motor Efferents: Co-
activates coordinated
mechanical actions across
the diaphragm, abdominal
wall, and lower esophageal
sphincter.

The Central Command: Nucleus Tractus Solitarius (NTS)



Anatomical Localization: Embedded deep within the medulla oblongata framework.

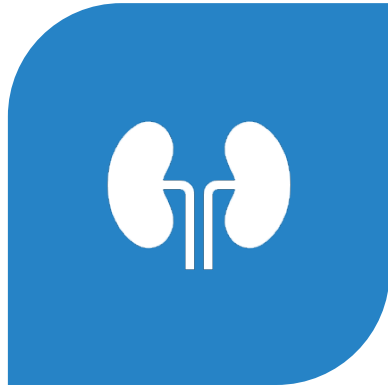


Receptor Infrastructure: Features dense concentrations of Neurokinin-1 (NK1), Serotonin (5-HT₃), and Muscarinic (M1) receptors.



Signal Processing: Collects inputs from various peripheral highways and acts as the gatekeeper for initiating somatic retching sequences.

The Chemoreceptor Trigger Zone (CTZ)



ANATOMICAL LOCATION: POSITIONED CLEANLY ON THE CAUDAL FLOOR OF THE FOURTH VENTRICLE WITHIN THE AREA POSTREMA.



BLOOD-BRAIN BARRIER ABSENCE: FENESTRATED CAPILLARY NETWORKS ALLOW CONTINUOUS MONITORING OF SYSTEMIC BIOCHEMICAL SHIFTS.



CHEMICAL DETECTION: SENSES BLOOD-BORNE EMETOGENIC COMPONENTS, INCLUDING CIRCULATING ANESTHETIC GASES, SYSTEMIC OPIOIDS, AND TOXINS.

Peripheral Highways: The Enteric-Vagal Network



Enterochromaffin Cells: Mucosal cells lining the gut lumen that react immediately to mechanical stretch, blood, or traction.



Serotonin Cascade: Local tissue irritation prompts a rapid release of visceral serotonin stores.



Vagal Propagation: Free serotonin stimulates 5-HT₃ receptors on vagal afferents, transmitting direct signals to the brainstem.

The Vestibular System & Balance Pathways



Labyrinthine Sensors: Detects changes in spatial orientation, acceleration, and angular motion mechanics.



Neurochemical Environment: Heavily driven by Histamine-1 (H1) and Muscarinic-1 (M1) receptor pathways.



PACU Triggers: Transporting patients on gurneys or changing head positions can spark sudden emetic episodes due to sensitized pathways.

Cortical Mechanisms: Anxiety, Pain, and Anticipation



Limbic Network: High preoperative anxiety and fear channel direct excitatory inputs to central emetic centers.



Sensory Triggers: Foul smells, sights, or intense operating room stressors project through the amygdala network.



Pain Paths: Severe, uncontrolled acute postoperative pain triggers cortical relays that lower the emetic threshold.

Neurotransmitter Deep Dive: Serotonin (5-HT3)



Receptor Class: Ionotropic ligand-gated ion channel responsible for rapid cellular depolarization.



Primary Anatomical Map: Distributed across the vagal afferents in the gut, solitary tract nucleus, and the CTZ.



Temporal Profile: Serves as the primary driver of acute-phase emesis, dominating the immediate 0 to 2 hour postoperative window.

Neurotransmitter Deep Dive: Substance P (NK1)



Receptor Class: G-protein coupled receptor (GPCR) that triggers long-term intracellular signaling cascades.



Primary Anatomical Map: Densely concentrated within the medullary Nucleus Tractus Solitarius.



Temporal Profile: Acts as the main mediator of delayed-phase emesis, keeping patients highly symptomatic up to 24-48 hours.

Neurotransmitter Deep Dive: Dopamine (D2)

Receptor Target: Strongly concentrated within the blood-exposed Chemoreceptor Trigger Zone.

Opioid Reinforcement: Systemic opioid administration activates these receptors, lowering the baseline emetic threshold.

Clinical Leverage: Antagonizing this specific path helps isolate the brainstem from circulating chemical triggers.

Neurotransmitter Deep Dive: Histamine (H1) & Acetylcholine (M1)



Primary Function: Governs internal balance and tracks motion updates through vestibular integration loops.



Anesthetic Sensitivity: Vapors and narcotics sensitize these pathways, turning minor movements into strong emetic triggers.



Therapeutic Value: Blocking these receptors prevents movement-induced nausea during early patient recovery.

The background of the slide features a grayscale, out-of-focus image of a laboratory petri dish. A glass pipette tip is positioned in the center, with a single drop of liquid about to fall into the dish. The lighting creates soft highlights and shadows, emphasizing the textures of the glass and the liquid.

Risk Factors & Screening Tools

• Patient specific risk factors

Reliable indication

Female gender

Non smoker

Previous history of ponv

Not as reliable

Age

Predisposing gastric disorders

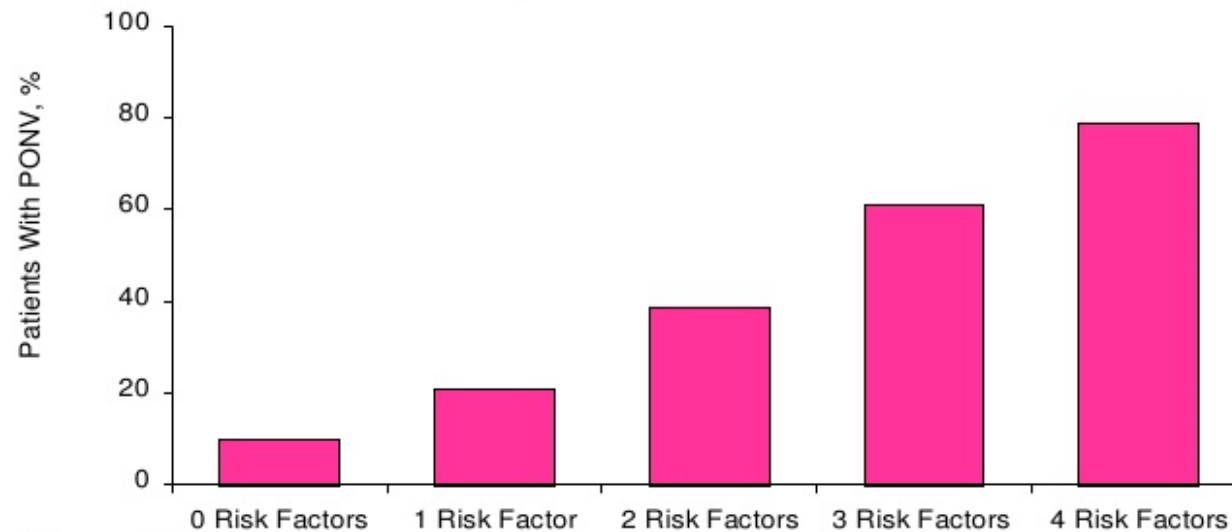
Preop anxiety

Obesity

Gastric distention

Impact of Multiple Patient-Related Risk Factors*

Risk of PONV Increased Based on Number of Primary Risk Factors Present



Primary Risk Factors:

History of PONV or motion sickness ■ Female sex ■ Nonsmoking ■ Use of postoperative opioids

*Validated in 2,722 adult patients receiving inhalational anesthesia.
Apfel CC et al. *Anesthesiology*. 1999;91:693-700.

Anesthetic risk factors

Use of volatile agents

Use of n2o

Use of intraop and post op opioids

Intubation

Etomidate

High doses of neostigmine

Surgical risk factors

Duration of surgery

- Each 30 min increase of surgery increases risk of ponv by 60%!

Early oral intake following surgery

Type of surgery:

Laparoscopy

Laparotomy

Breast

Strabismus (ophthalmologic)

Plastic surgery

Maxillofacial

Gyn

Abdominal

Neurologic

Urologic

Adult Risk Assessment: The Validated Apfel Score

The Four Primary Predictors: Female sex, non-smoker status, history of PONV or motion sickness, and anticipated post-op opioid use.



Risk Stratification: Each factor adds roughly 20% to baseline risk.



Scoring Distribution:



- 1 Factor: ~20% risk | 2 Factors: ~40% risk | 3 Factors: ~60% risk
| 4 Factors: ~80% risk

Apfel Component 1: Female Sex Breakdown

Primary Factor: Post-pubertal female sex stands as the strongest independent predictor of PONV.

Hormonal Interaction: Shifts in estrogen and progesterone alter receptor sensitivity within the CTZ.

Menstrual Cycle Cycles: Higher susceptibility noted during the luteal phase, though prophylaxis remains uniform.

Apfel Component 2: Non-Smoker Status Breakdown

The Protective Paradox: Active smoking significantly reduces the risk of developing PONV.

Enzyme Activation: Polycyclic hydrocarbons in smoke induce hepatic cytochrome P450 systems, accelerating clearance.

Receptor Shifts: Long-term nicotine exposure shifts baseline dopaminergic and nicotinic receptor sensitivity in the CTZ.

Apfel Component 3: History of PONV / Motion Sickness

Hyper-Reactive Phenotype: Suggests a lower genetic threshold for activating the central pattern generator.

Vestibular Overlap: High correlation between patients who get carsick and those who experience severe postanesthetic nausea.

Prior Outcomes: Past episodes of severe PONV represent a strong warning sign of a highly sensitive reflex loop.

Apfel

Component 4: Postoperative Opioid Anticipation

Dual Attack: Systemic postoperative narcotics trigger central receptors while simultaneously causing gastric stasis.

Dose-Dependent Trends: Overall emetic risk scales with the cumulative dose of intraoperative and postoperative opioids.

PCA Metrics: Patient-Controlled Analgesia can extend the emetic risk window if not paired with proper multi-receptor blocks.

Pediatric Risk Assessment: The POVOC / Eberhart Score



Applicability: Validated for children over the age of three (Postoperative Vomiting - POV).



Core Predictors: Age 3 years or older; history of POV or family history of PONV; strabismus, tonsillectomy, or hernia surgeries; surgical duration > 30 minutes.



Intervention Mandate: High POVOC scores require aggressive prevention due to elevated baseline pediatric vomiting risks.

Additional Emerging Patient Risk Factors

Age Cutoffs: Risk peaks in young adulthood (18-50) and drops significantly in geriatric cohorts.

The Obesity Myth: Large-scale clinical data confirms high BMI is not an independent risk factor for PONV on its own.

Gastric Conditions: Chronic conditions like diabetic gastroparesis slow down baseline clearance, raising emetic risk.

Surgical Triggers:
Laparoscopy &
Pneumoperitoneum

Mechanical Insufflation: CO₂ gas distends the peritoneal cavity and compresses abdominal organs.

Vagal Stimulation: Stretching activates mechanoreceptors on the vagal nerve, sending signals to the brainstem.

CO₂ Absorption: Systemic absorption can cause mild respiratory acidosis, further irritating central emetic centers.

Surgical Triggers: Gynecological & Breast Procedures

Neuroendocrine Axis: Procedures like hysterectomies trigger acute shifts in circulating sex hormones.

Tissue Innervation: Highly innervated breast tissues send strong afferent signals up the spinal cord when handled.

Risk Clustering: These surgeries often involve female patients who require post-op opioids, combining multiple risk factors.

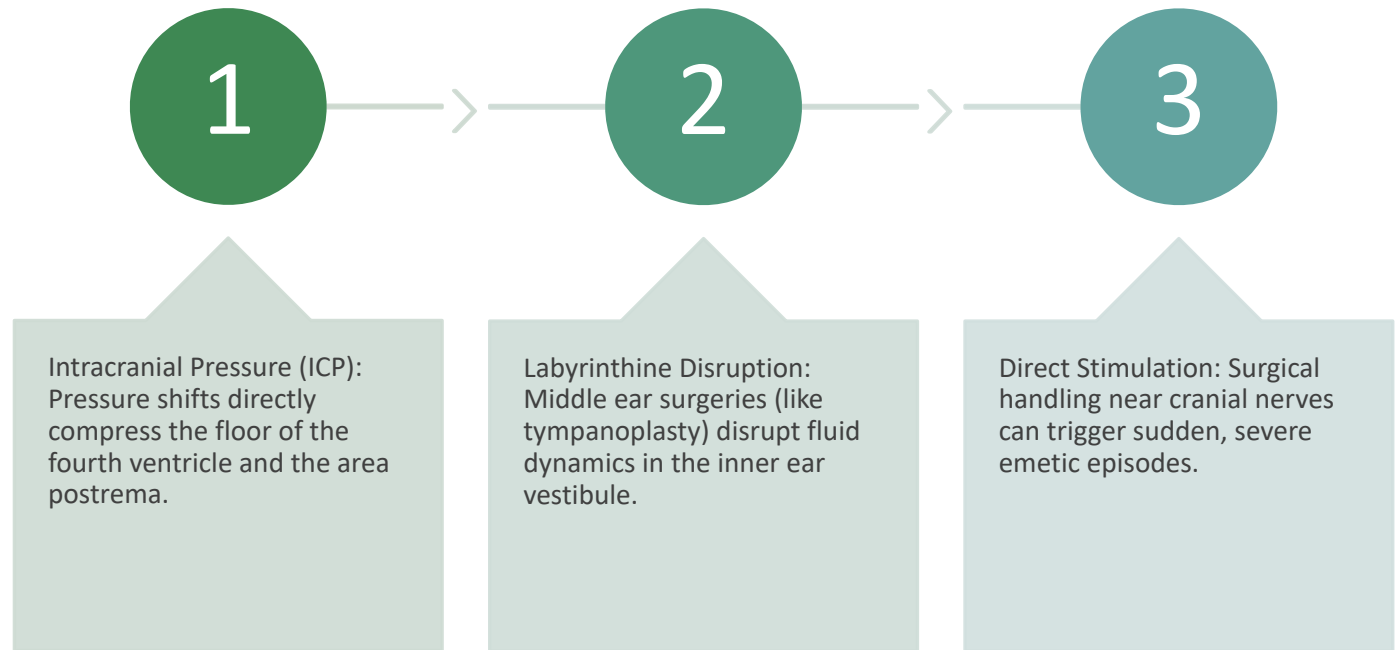
Surgical Triggers: Strabismus & Adenotonsillectomy

The Oculoemetic Reflex: Traction on extraocular muscles during strabismus repair triggers strong vagal discharge.

Blood Pools: Swallowed blood after tonsillectomy acts as a direct local irritant inside the stomach lining.

Airway Manipulation: Aggressive suctioning and pharyngeal irritation trigger local glossopharyngeal nerve networks.

Surgical Triggers: Neurosurgery & Middle Ear Procedures



Anesthetic Triggers: Volatile Halogenated Gases

Primary Culprit: Inhalational gases (Sevoflurane, Desflurane, Isoflurane) are the main cause of early PONV (0-2 hours).

Dose-Dependent Effect: Risk scales with the depth and overall duration of exposure to the gas.

Cellular Mechanism: Volatile gases irritate central emetic pathways and slow down gastrointestinal smooth muscle contractility.

Anesthetic Triggers: Nitrous Oxide (N₂O)

Space Expansion: Nitrous oxide diffuses rapidly into closed, air-filled spaces, increasing pressure in the middle ear.

Central Activation: Stimulates sympathetic networks and triggers dopamine pathways within the CTZ.

Risk Reduction: Avoiding nitrous oxide helps significantly reduce emetic rates in high-risk patient cohorts.

Anesthetic Triggers: Intraoperative Opioids

Central Inputs: Systemic opioids cross the area postrema to activate central dopamine and serotonin networks.

Peripheral Stasis: Narcotics bind to mu receptors in the gut wall, slowing down peristalsis and delaying gastric emptying.

Vestibular Sensitization: Lowers the threshold for motion-induced nausea, turning bed transfers into active triggers.

The Fifth Consensus Guidelines (Gan et al., 2026)

Proactive Paradigm: Recommends moving away from single-agent therapy toward early, aggressive multimodal combinations.

The Quality Standard: Minimizing PONV is treated as a critical metric for successful recovery and discharge.

ERAS Integration: Establishes combination prophylaxis as a core requirement for Enhanced Recovery After Surgery pathways.

Risk-Based Prophylaxis Architecture

Low Risk (0-1 Factors): Managed effectively with 1 to 2 baseline prophylactic interventions.

Medium Risk (2 Factors): Requires at least 2 to 3 distinct drug classes targeting different receptors.

High Risk (3-4 Factors): Mandates aggressive quadruple therapy or combining 3 drug classes with a propofol TIVA technique.

Baseline Risk Mitigation: Propofol TIVA



Direct Antiemetic Effect: Propofol actively suppresses the CTZ and reduces subcortical serotonin levels.



Elimination of Vapors: Switching to TIVA removes volatile gases, the main trigger for early post-op nausea.



Clinical Efficacy: Using TIVA instead of volatile gas has an NNT of roughly 5 to 11 for preventing PONV.

Opioid-Sparing Multimodal Analgesia

Regional Blocks: Using peripheral nerve blocks and epidurals to minimize or eliminate systemic opioids.

Non-Opioid Options:
Scheduled administration of IV Acetaminophen, NSAIDs, or Ketamine.

Recovery Impact:
Minimizes narcotic-induced gut stasis and keeps baseline emetic thresholds high.

Fluid Dynamics & Intraoperative Euvolemia



Hypovolemia Irritation: Poor gut perfusion triggers localized adenosine release, driving vagal emetic pathways.



Hydration Remedy: Giving 10 to 20 mL/kg of balanced crystalloids reduces baseline nausea in ambulatory outpatients.



Euvolemia Goal: Targeted fluid administration maintains stable bowel perfusion without causing tissue edema.

Neuromuscular Reversal: Sugammadex vs. Neostigmine



The Neostigmine Problem: Anticholinesterases increase systemic acetylcholine, stimulating muscarinic receptors in the gut.



The Sugammadex Mechanism: Encapsulates neuromuscular blockers directly without altering systemic acetylcholine levels.



Clinical Evidence: Utilizing sugammadex over neostigmine helps lower the incidence of early postoperative vomiting.

Pharmacologic Guide: Ondansetron (5-HT₃)



Standard Adult Dose: 4 mg IV administered during emergence or toward the end of surgery.



Elimination Half-Life: Roughly 3 to 5 hours; primarily protects against early-phase recovery emesis.



Safety Considerations: Can cause dose-dependent QT interval prolongation; requires caution in high-risk cardiac patients.

Pharmacologic Guide: Palonosetron (5-HT₃)

Standard Adult Dose:
0.075 mg IV administered
during anesthesia
induction.

Extended Half-Life:
Features an impressive
40-hour half-life with high
receptor binding affinity.

Clinical Benefit: Provides
long-lasting protection
that helps prevent both
early recovery emesis and
post-discharge nausea.

Pharmacologic Guide: Dexamethasone (Corticosteroid)

Standard Adult Dose: 4 to 5 mg IV administered immediately after induction.

Mechanism of Action:
Reduces central prostaglandin synthesis and dampens inflammatory shifts in the brainstem.

Timing Requirement: Must be given early at induction due to its slow, gene-mediated onset of action.

Pharmacologic Guide: Aprepitant (NK1)

Standard Adult Dose:
40 mg orally
administered within 3
hours prior to surgical
induction.

Target Phase: Highly
effective at preventing
delayed-phase nausea
and post-discharge
vomiting.

Synergistic Power: Pairs
exceptionally well with
5-HT3 antagonists to
provide comprehensive
dual-phase coverage.

Pharmacologic Guide: Droperidol (D2 Antagonist)

Standard Adult Dose:
0.625 to 1.25 mg IV
administered toward the
end of surgery.

Target Site: Provides
strong, competitive
binding at dopamine-2
receptors within the
CTZ.

Safety Profile: Low-dose
administration carries an
excellent safety profile
with minimal risk of
dysrhythmias.

Pharmacologic Guide: Transdermal Scopolamine (M1)

Standard Adult Dose: 1 transdermal patch (1.5 mg) applied behind the ear.

Optimal Timing: Should be placed the night before surgery or at least 2 hours prior to induction.

Side Effect Profile: Can cause mild dry mouth, blurred vision, or transient confusion in elderly patients.

Breakthrough PACU Management: Rules of Rescue

The Rotation Rule: Never repeat a drug class that was already used for prophylaxis within the previous 6 hours.

Receptor Fatigue: Re-administering the same drug class adds zero clinical benefit and increases side effects.

Alternative Options: Pivot immediately to an unused mechanism, such as a dopamine antagonist or a low dose of propofol.

Non- Pharmacologic Alternative Options



P6 Acustimulation: Stimulating the Pericardium 6 meridian helps reduce central emetic signaling.



Isopropyl Alcohol Inhalation: Inhaling alcohol vapor provides rapid, temporary relief while preparing IV rescue drugs.



Clinical Acceptance: Both techniques are fully supported by the latest international consensus guidelines.

Post-Discharge Nausea and Vomiting (PDNV)



The At-Home Challenge: Affects a significant number of outpatients once intraoperative blocks wear off.



Risk Drivers: Strongly driven by age under 50, history of PONV, and ongoing opioid use at home.



Extended Protection: Recommends using long-acting drugs like Palonosetron or oral Aprepitant for high-risk outpatients.

AANA Position Statements on ERAS Protocols

Opioid Minimization: Advocates for opioid-sparing techniques as a core safety standard for anesthesia care.

Protocolized Care: Supports standardized, evidence-based approaches to eliminate variance in patient outcomes.

Patient-Centered Metrics: Ties aggressive PONV prevention directly to faster recovery and higher patient satisfaction.

ASA Practice Guidelines on Postanesthetic Care

Routine Assessment: Mandates regular evaluation of emetic symptoms during recovery.

Multimodal Approach: Strongly supports combination prophylaxis for patients with moderate-to-high risk profiles.

Discharge Standards: Requires stable emetic control before a patient can be safely cleared for discharge.

Summary & Clinical Workflow Checklist



Assess

Assess Baseline Risk: Score every patient using the Apfel or POVOC matrix before surgery.



Incorporate

Incorporate Mitigation: Use TIVA, regional blocks, and proper hydration whenever possible.



Combine

Combine Drug Classes: Deploy 2 to 4 different drug classes for medium and high-risk patients.



Rotate

Rotate Rescue Agents: Treat breakthrough symptoms with a completely fresh drug mechanism.

Questions?



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