HARRISON

EDITION 22nd

GASTROENTEROLOGY

Gastroenterology

Approach To The Patient With Gastrointestinal Diseases	Completed
Diseases Of The Esophagus	Completed
Peptic Ulcer Disease And Disorders	Completed
Disorders Of Absorption	Completed
Gastrointestinal Endoscopy	Completed
Inflammatory Bowel Disease	Completed
Acute Intestinal Obstruction	Completed
Diverticular Disease, Anorectal Disorders And Appendix	Completed
Inflammatory Bowel Disease Part 2	Completed
Irritable Bowel Syndrome	Completed
Mesenteric Vascular Insufficiency	Completed
Nutrition	Completed
Approach To Liver Disease And Liver Function Tests	Completed

Viral Hepatitis	Completed
Liver Disorders	Completed
Acute And Chronic Pancreatitis	Completed
Cirrhosis And Its Complications	Completed
Approach To The Patient With Pancreatic Disease	Completed

Section 6 Alterations in Gastrointestinal Function

47 Dysphagia

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False about globus pharyngeus

- A- Never interfere with swallowing
- B- Relived after swallowing but only sometime
- C- Fear of swallowing due to foreign body sensation
- D- counselling and anti anxiety sometime help

Ans- c

- Aphagia (inability to swallow) - in the acute setting of a food bolus or foreign body impaction.
- Odynophagia - refers to painful swallowing, typically resulting from mucosal ulceration within the oropharynx or esophagus.
- Globus pharyngeus - foreign body sensation localized in the neck that does not interfere with swallowing and sometimes is relieved by swallowing.
- Transfer dysphagia frequently results in nasal regurgitation or pulmonary aspiration during swallowing and is characteristic of oropharyngeal dysphagia.
- Phagophobia (fear of swallowing) and refusal to swallow may be psychogenic or related to anticipatory anxiety about food bolus obstruction, odynophagia, or aspiration.

Physiology of swallowing

- Bolus entry into the hypopharynx initiates the **pharyngeal swallow response**, which is centrally mediated and involves a complex series of actions, the **net result of which is to propel food through the pharynx into the esophagus while preventing its entry into the airway.**
- **To accomplish this, the larynx is elevated and pulled forward, actions that also facilitate upper esophageal sphincter (UES) opening.**
- **Tongue pulsion then propels the bolus through the UES, followed by a peristaltic contraction that clears residue from the pharynx and through the esophagus.**

Which of the following is non peristaltic esophageal contraction ??

- A- primary
- B- secondary
- C- Tertiary
- D- both secondary and tertiary

Ans- C

- **The lower esophageal sphincter (LES) relaxes as the food enters the esophagus and remains relaxed until the peristaltic contraction has delivered the bolus into the stomach.**
- **Peristaltic contractions elicited in response to a swallow are called primary peristalsis and involve sequenced inhibition followed by contraction of the musculature along the entire length of the esophagus.**
- **The inhibition that precedes the peristaltic contraction is called deglutitive inhibition.**



- **Local distention of the esophagus anywhere along its length, as may occur with gastroesophageal reflux, activates secondary peristalsis** that begins at the point of distention and proceeds distally.

- **Tertiary esophageal contractions are nonperistaltic, disordered esophageal contractions that may be observed to occur spontaneously during fluoroscopic observation**

- **Oral cavity muscles are innervated by the fifth (trigeminal) and seventh (facial) cranial nerves; the tongue, by the 12th (hypoglossal) cranial nerve.**

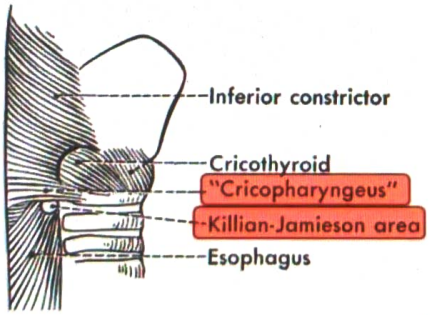
- **Pharyngeal muscles are innervated by the ninth (glossopharyngeal) and tenth (vagus) cranial nerves.**

- **UES opening during swallowing involves both cessation of vagal excitation to the cricopharyngeus and simultaneous contraction of the suprahyoid and geniohyoid muscles**

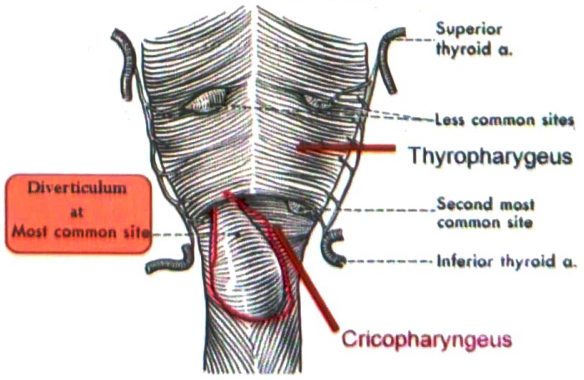
- **Distal esophagus and LES are composed of smooth muscle and are controlled by excitatory and inhibitory neurons within the esophageal myenteric plexus.**

- Neurotransmitters of the excitatory ganglionic neurons - acetylcholine and substance P
- Inhibitory neurons - vasoactive intestinal peptide and nitric oxide.

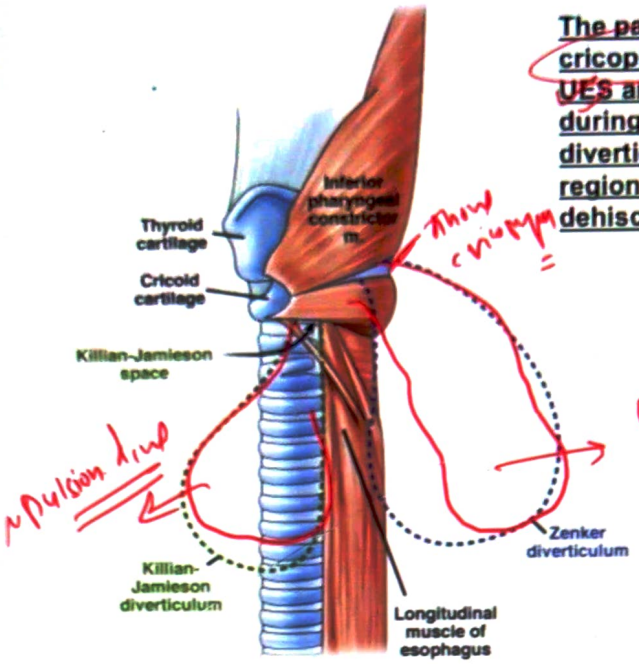
- Scleroderma commonly presents with absent peristalsis as well as a weakened LES that predisposes patients to peptic stricture formation.



- As the larynx descends with aging the inferior constrictor pharyngeal musculature takes an oblique course
- Killian's dehiscence develops between the "thyropharyngeus" and "cricopharyngeus" portions of

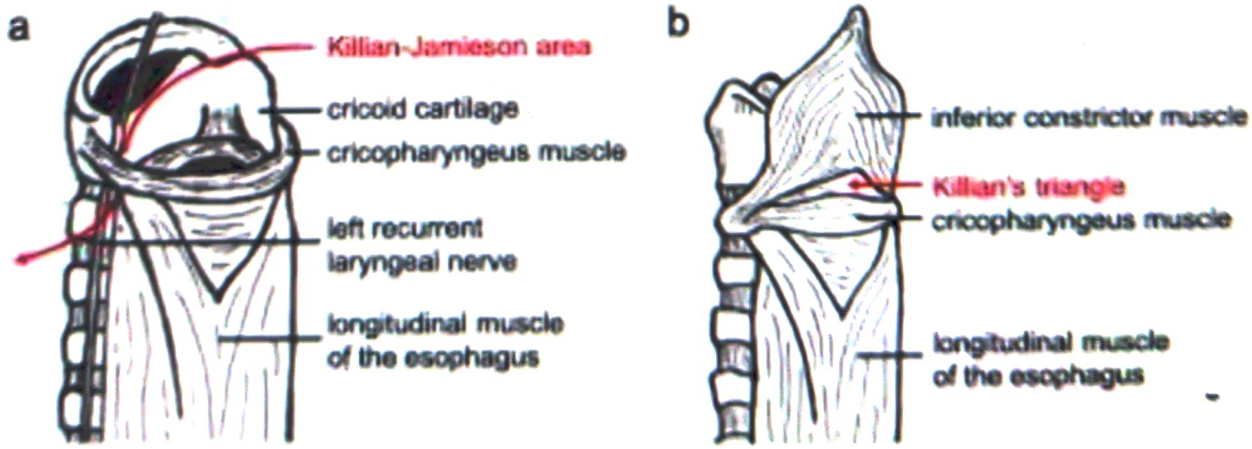


The pathogenesis is related to stenosis of the cricopharyngeus that causes diminished opening of the UES and results in increased hypopharyngeal pressure during swallowing with development of a pulsion diverticulum immediately above the cricopharyngeus in a region of potential weakness known as Killian's dehiscence.



Left lateral view

	Killian Jamieson (KJ) diverticulum	Zenker diverticulum (ZD)
Location	Lateral wall of pharyngoesophageal junction in space below cricopharyngeus muscle and lateral to longitudinal muscular fiber of esophagus	Posterior wall of pharnygoesophageal junction below superior pharyngeal constrictor muscle and above cricopharyngeus muscle
Prevalence	Rare	More Common
Laterality	Left	Left
Gender	Female	Male
Age average	58 years	70 years
Size	Small	Medium to Large
Symptoms	Dysphagia, regurgitation, globus sensation At least one-third have no aerodigestive symptoms	Dysphagia, cervical borborygmus, weight loss, hoarseness, cough, aspiration pneumonia



Esophageal Dysphagia

- When distended, the esophageal lumen has internal dimensions of about 2 cm in the anteroposterior plane and 3 cm in the lateral plane
- Solid food dysphagia becomes common when the lumen is narrowed to <13 mm
- The most common structural causes of dysphagia are Schatzki's rings, eosinophilic esophagitis, and peptic strictures.

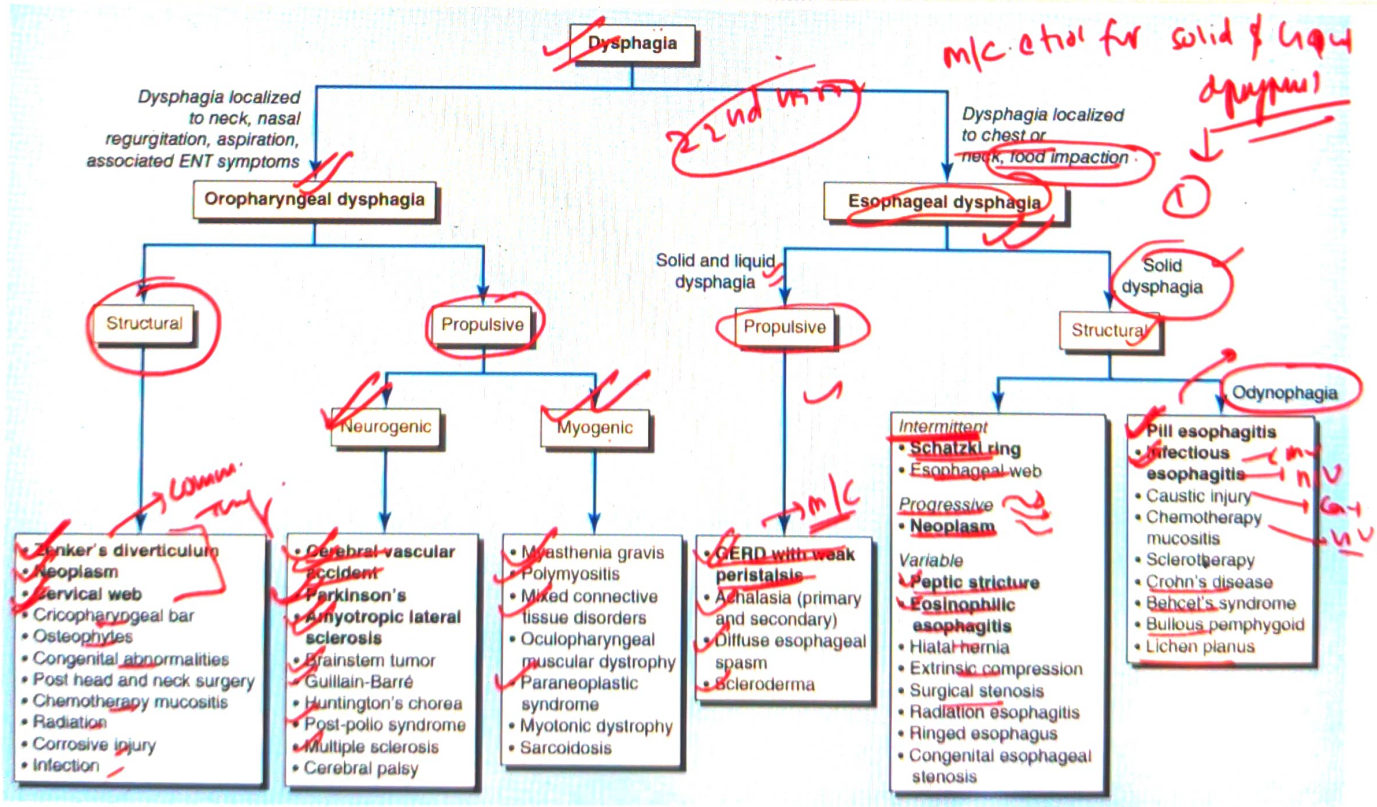


FIGURE 47-2 Approach to the patient with dysphagia. Etiologies in bold print are the most common. ENT, ear, nose, and throat; GERD, gastroesophageal reflux disease.

Intermittent dysphagia that occurs only with solid food implies structural dysphagia, whereas constant dysphagia with both liquids and solids strongly suggests an esophageal motor abnormality.

Two caveats to this pattern are that despite having a motor abnormality, patients with scleroderma generally develop mild dysphagia for solids only and that patients with oropharyngeal dysphagia often have greater difficulty managing liquids than solids.

Dysphagia that is progressive over the course of weeks to months raises concern for neoplasia.

Episodic dysphagia to solids that is unchanged or slowly progressive over years indicates a benign disease process such as a Schatzki ring or eosinophilic esophagitis.

Food impaction with a prolonged inability to pass an ingested bolus even with ingestion of liquid is typical of structural dysphagia.

48 Nausea, Vomiting, and Indigestion

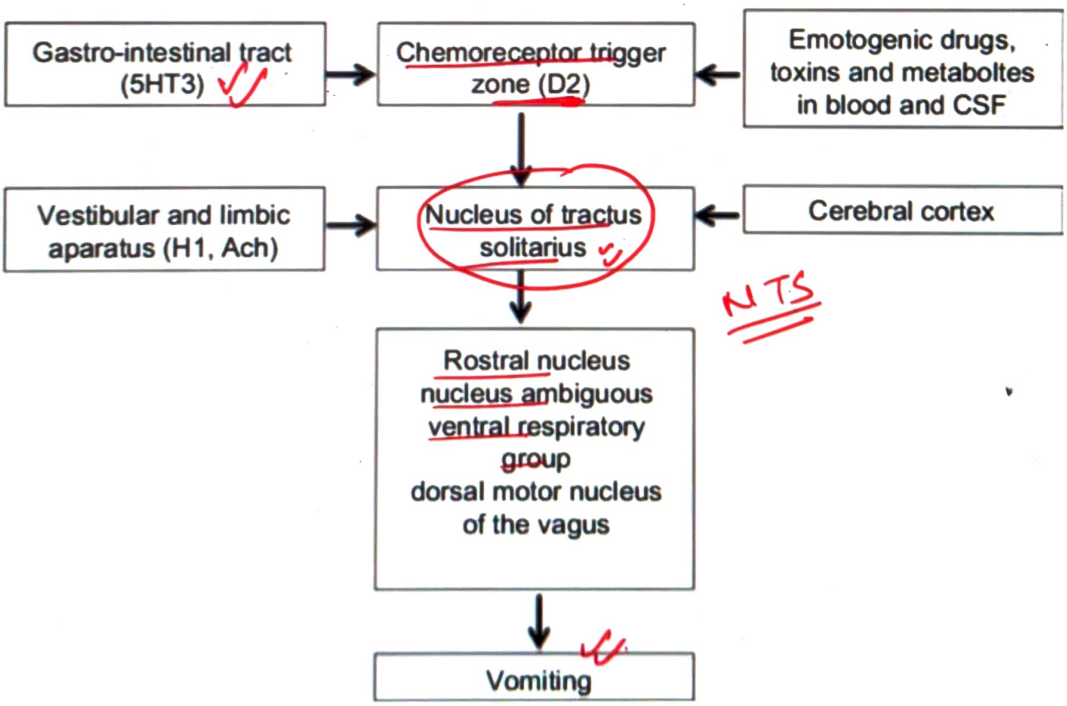
William L. Hasler

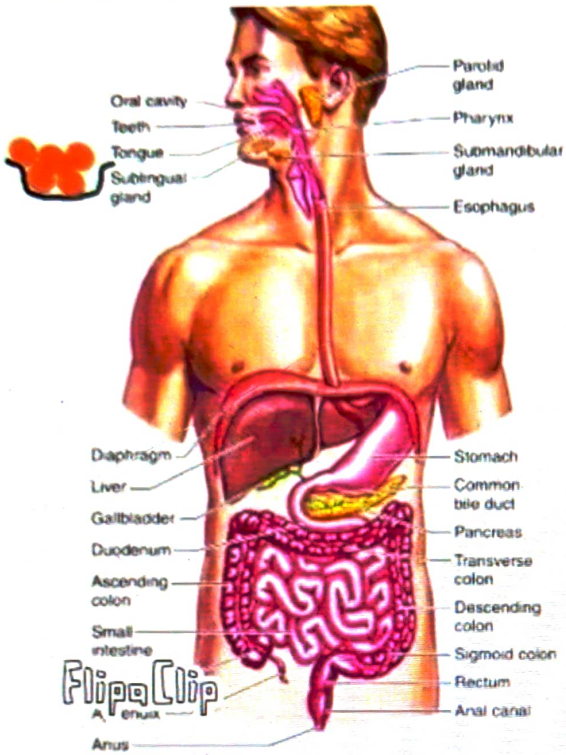
Nausea is the feeling of a need to vomit. *Vomiting* (emesis) is the oral expulsion of gastrointestinal contents resulting from gut and thoracoabdominal wall contractions. Vomiting is contrasted with *regurgitation*, the effortless passage of gastric contents into the mouth. *Rumination* is the repeated regurgitation of food residue. In contrast to

Under normal condition what is distal migratory gut contraction.

pathways.

Somatic and visceral muscles respond stereotypically during emesis. Inspiratory thoracic and abdominal wall muscles contract, increasing intrathoracic and intraabdominal pressures to evacuate the stomach. Under normal conditions, distally migrating gut contractions are coordinated by an electrical phenomenon, the slow wave, which cycles at 3 cycles/min in the stomach and 11 cycles/min in the duodenum. During emesis, slow waves are abolished and replaced by orally propagating spikes that evoke retrograde contractions to facilitate expulsion of gut contents.





How does vomiting starts?

1- Vomiting begins with salivation

2-And the sensation of nausea.

3-Reverse peristalsis empties material from the Upper part of the small intestine into the stomach.

4-The glottis closes preventing aspiration of vomitus into the trachea.

5-The breath is held in mild inspiration.

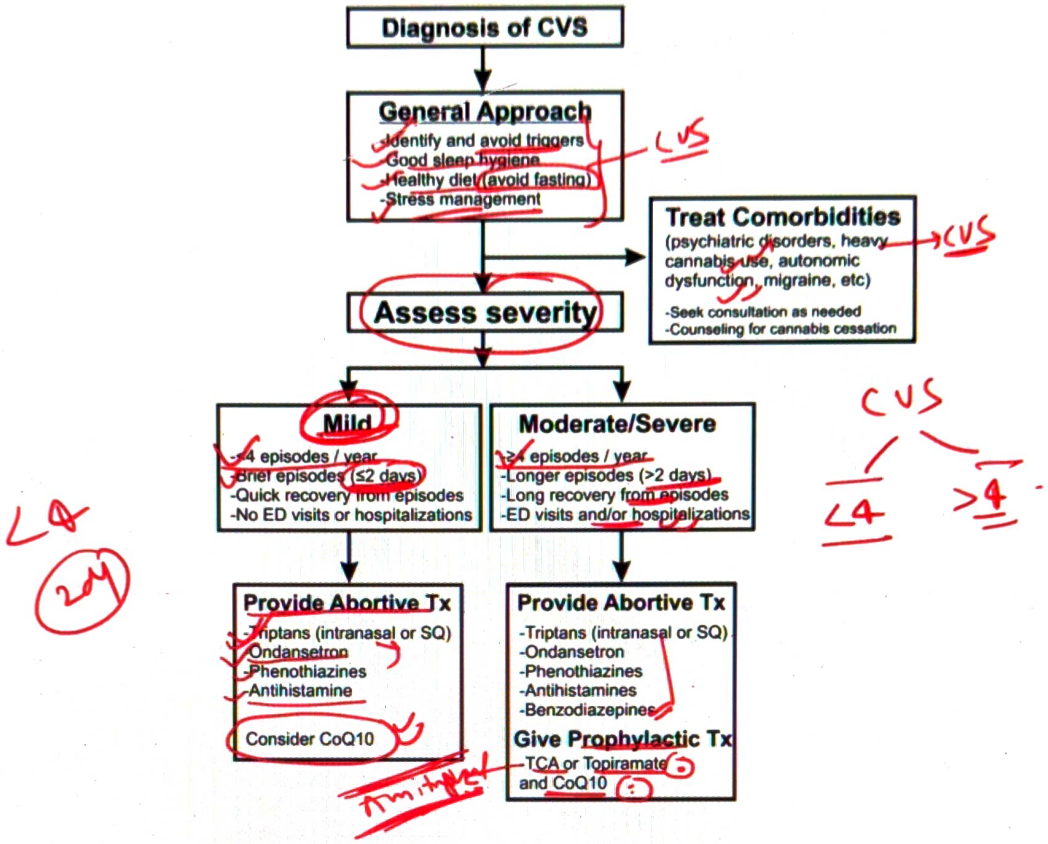
6- The muscles of the abdominal wall contract, and because the chest is held in fixed position, the contraction increases intra-abdominal pressure.

INTRAPERITONEAL	EXTRAPERITONEAL	MEDICATIONS/ METABOLIC DISORDERS
Obstructing disorders	Cardiopulmonary disease	Drugs
Pyloric obstruction	Cardiomyopathy	Cancer chemotherapy
Small-bowel obstruction	Myocardial infarction	Analgesics
Colonic obstruction	Labyrinthine disease	Opioids
Superior mesenteric artery syndrome	Motion sickness	Antibiotics
Enteric infections	Labyrinthitis	Cardiac antiarrhythmics
Viral	Malignancy	Digoxin
Bacterial	Intracerebral disorders	Oral hypoglycemics
Inflammatory diseases	Malignancy	Oral contraceptives
Cholecystitis	Hemorrhage	Antidepressants
Pancreatitis	Abscess	Restless legs/ Parkinson's therapies
Appendicitis	Hydrocephalus	Smoking cessation agents
Hepatitis	Psychiatric illness	Endocrine/metabolic disease
Altered sensorimotor function	Anorexia and bulimia nervosa	Pregnancy
Gastroparesis	Depression	Uremia
Intestinal pseudoobstruction	Postoperative vomiting	Ketoacidosis
Gastroesophageal reflux		Thyroid and parathyroid disease
Chronic nausea vomiting syndrome		Adrenal insufficiency
Cyclic vomiting syndrome		Toxins
Cannabinoid hyperemesis syndrome		Liver failure
Rumination syndrome		Ethanol
Mesenteric insufficiency		
Celiac artery stenosis		
Median arcuate ligament syndrome		
Biliary colic		
Abdominal irradiation		

Idiopathic gastroparesis is the most prevalent etiology; it occurs in the absence of systemic illness and follow a viral illness in ~15–20% of cases.

Intestinal pseudoobstruction may be idiopathic, inherited, result from systemic disease like scleroderma or an infiltrative process like amyloidosis, or occur as a paraneoplastic consequence of malignancy (e.g., small-cell lung carcinoma)

Cyclic vomiting syndrome (CVS) causes 3–14% of cases of unexplained nausea and vomiting and presents with discrete episodes of relentless vomiting and is associated with migraines.



Which drug can be used in chemotherapy induced nausea and vomiting

- A- Gabapentin
- B- Amitryptyline
- C- Mirtazepine
- D- Buspirone

Ans- A

Drug used in intestinal pseudo obstruction in scleroderma

- A- Octreotide
- B- Neostigmine
- C- Metoclopromide
- D- Erythromycin

Ans- A

Not used in CVS

- A- Levetriecetam
- B- sumatriptan
- C- Haloperidol
- D- Toprimate

Chronic nausea vomiting syndrome is defined as bothersome nausea and/ or one or more vomiting episodes at least weekly

CVS , CHS

- Cyclic vomiting syndrome (CVS) presents with discrete episodes of relentless vomiting, has a prevalence of 1.4%, and is associated with migraines, autonomic dysfunction, and menstrual cycling. Some cases exhibit rapid gastric emptying.
- A related condition, cannabinoid hyperemesis syndrome (CHS), presents with cyclical vomiting in individuals with long-standing (>1 year) use of large quantities of cannabis at least 4 days weekly and resolves with its discontinuation for ≥6 months.
- Rumination syndrome is often misdiagnosed as refractory vomiting.

DOC for acute attack of CHS

- A- Levetriecetam
- B- sumatriptan → D.O.C for CVS
- C- Haloperidol
- D- Toprimate

Cannabinoid hyperemesis syndrome

C,C

Managing CVS and CHS is challenging. Prophylaxis with tricyclic antidepressants or anticonvulsants (topiramate, zonisamide, levetiracetam) reduces the severity and frequency of CVS attacks in uncontrolled reports. Combining intravenous 5-HT_3 antagonists with the sedating effects of a benzodiazepine like lorazepam are mainstays for aborting acute flares. Small studies report benefits with aprepitant and injectable or intranasal forms of the 5-HT_1 agonist sumatriptan to manage acute CVS episodes. These treatments are reportedly less effective for CHS, but haloperidol and topical capsaicin cream may reduce acute CHS attacks

Or Haloperidol - used for acut attack of CHS

TABLE 48-2 Treatment of Nausea and Vomiting

TREATMENT	MECHANISM	EXAMPLES	CLINICAL INDICATIONS
Antiemetic agents	Antihistaminergic	Dimenhydrinate, meclizine	Motion sickness, inner ear disease
	Anticholinergic	Scopolamine	Motion sickness, inner ear disease
	Antidopaminergic	Prochlorperazine, thiethylperazine, haloperidol	Medication-, toxin-, or metabolic-induced emesis, chemotherapy-induced emesis,? cannabinoid hyperemesis syndrome
	5-HT_3 antagonist	Ondansetron, granisetron	Chemotherapy- and radiation-induced emesis, postoperative emesis, opioid-induced nausea and vomiting
	Cannabinoids	Tetrahydrocannabinol, cannabidiol	Chemotherapy-induced emesis, gastroparesis
	Tricyclic antidepressant	Amitriptyline, nortriptyline	Chronic nausea vomiting syndrome, cyclic vomiting syndrome,? gastroparesis
	Other antidepressant/atypical antipsychotic	Mirtazapine, olanzapine	Functional dyspepsia, chemotherapy-induced emesis,? gastroparesis
	Neuropathic modulator	Gabapentin	Chemotherapy-induced emesis
	Neurokinin (NK_1) receptor antagonists	Aprepitant, fosaprepitant, netupitant, rolapitant	Chemotherapy-induced emesis
Prokinetic agents	5-HT_4 agonist and antidopaminergic	Metoclopramide	Gastroparesis
	Motilin agonist	Erythromycin	Gastroparesis,? intestinal pseudoobstruction
	Peripheral antidopaminergic	Domperidone	Gastroparesis
	Pure 5-HT_4 agonist	Prucalopride	Idiopathic gastroparesis
	Somatostatin analogue	Octreotide	Intestinal pseudoobstruction
	Acetylcholinesterase inhibitor	Pyridostigmine	?Small-intestinal dysmotility/pseudoobstruction
Special settings	Benzodiazepines	Lorazepam	Anticipatory nausea and vomiting with chemotherapy, cyclic vomiting syndrome
	5-HT_{1A} agonist	Buspirone, tandospirone	Functional dyspepsia
	Glucocorticoids	Methylprednisolone, dexamethasone	Chemotherapy-induced emesis
	Anticonvulsants	Topiramate, zonisamide, levetiracetam	Cyclic vomiting syndrome
	Antimigraine agents	Sumatriptan	Cyclic vomiting syndrome
	Topical analgesic	Capsaicin cream	Cannabinoid hyperemesis syndrome

Note:? , indication is uncertain.

GENERAL PRINCIPLES

Therapy of vomiting is tailored to correct remediable abnormalities if possible. Patients with severe dehydration should be hospitalized if oral fluid replenishment is unsustainable. Once oral intake is tolerated, low-fat liquid nutrients are restarted because lipids delay gastric emptying. Low-residue, small-particle diets have shown efficacy in gastroparesis. Glycemic control should be optimized to reduce diabetic gastroparesis symptoms.

G- POEM used for

- A- Gastroparesis
- B- Achalasia type 3
- C- GERD
- D- GOO

Ans- A

uncontrolled studies, but small controlled trials observed benefits no greater than sham treatments. Surgical pyloroplasty and gastric peroral endoscopic myotomy (G-POEM) of the pylorus improved symptoms in case series. Enteral feedings through a jejunostomy reduce hospitalizations and improve overall health in some patients with refractory gastroparesis. Subtotal gastric resection may improve some cases of postvagotomy gastroparesis, but its utility for other gastroparesis etiologies is unproven. Implanted gastric electrical stimulators may reduce symptoms, enhance nutrition, improve quality of life, and decrease health care expenditures in medication-refractory gastroparesis; a controlled trial has confirmed modest improvement in vomiting.

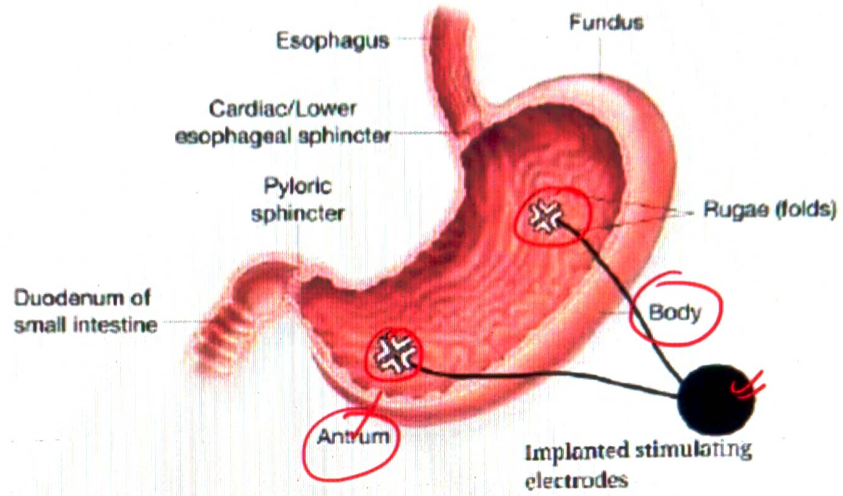


Figure 11: Ideal electrode configuration of gastric stimulator.

■ DIFFERENTIAL DIAGNOSIS

Gastroesophageal Reflux Disease Heartburn or regurgitation is reported weekly by 18–28% of the population, highlighting the prevalence of gastroesophageal reflux disease (GERD). Most cases of heartburn result from excess acid reflux, but reflux of weakly acidic or nonacidic fluid can produce similar symptoms. Alkaline reflux esophagitis elicits GERD symptoms in patients who have had surgery for peptic ulcer disease. Ten percent of patients with heartburn exhibit no acidic or nonacidic esophageal reflux and are considered to have functional heartburn.

Functional Dyspepsia Approximately 20% of the populace has

TABLE 45-3 Alarm Symptoms in Gastroesophageal Reflux Disease

- Odynophagia or dysphagia
- Unexplained weight loss
- Recurrent vomiting
- Occult or gross gastrointestinal bleeding
- Jaundice
- Palpable mass or adenopathy
- Family history of gastroesophageal malignancy

TREATMENT

Indigestion

LIFESTYLE, DIET, AND NONMEDICATION RECOMMENDATIONS

Patients with mild indigestion can be reassured that a careful evaluation revealed no serious disease and are offered no other intervention. If possible, drugs that cause gastroesophageal reflux or dyspepsia should be stopped. GERD patients should limit ethanol, caffeine, chocolate, and tobacco use and can ingest a low-fat diet, avoid snacks before bedtime, and elevate the head of the bed. Functional dyspepsia patients can be advised to reduce intake of fat, spicy foods, caffeine, and alcohol. Dietary lactose restriction is appropriate for lactase deficiency, while gluten exclusion is indicated for celiac disease. Low FODMAP (fermentable oligosaccharide, disaccharide, monosaccharide, and polyol) diets are effective for gaseous symptoms in IBS. In a systematic review, FODMAP intake correlated with functional dyspepsia symptoms, suggesting potential utility in this disorder as well.

Not useful in functional dyspepsia

- A- SSRI
- B- Amitryptyline
- C- Buspirone
- D- Pregabalin