

Gastric Electrical Stimulation

Effective: August 1, 2021

Next Review: April 2022

Last Review: June 2021

IMPORTANT REMINDER

Medical Policies are developed to provide guidance for members and providers regarding coverage in accordance with contract terms. Benefit determinations are based in all cases on the applicable contract language. To the extent there may be any conflict between the Medical Policy and contract language, the contract language takes precedence.

PLEASE NOTE: Contracts exclude from coverage, among other things, services or procedures that are considered investigational or cosmetic. Providers may bill members for services or procedures that are considered investigational or cosmetic. Providers are encouraged to inform members before rendering such services that the members are likely to be financially responsible for the cost of these services.

DESCRIPTION

Gastric electrical stimulation (GES) is performed using an implantable device designed to treat chronic drug-refractory nausea and vomiting secondary to gastroparesis of diabetic or idiopathic etiology. Gastric electrical stimulation is also proposed as a treatment of obesity. The device may also be referred to as a gastric pacemaker or gastric pacing.

MEDICAL POLICY CRITERIA

- I. Gastric electrical stimulation may be considered **medically necessary** in the treatment of chronic intractable nausea and vomiting secondary to gastroparesis of diabetic, idiopathic or post-surgical etiology when all of the following (A – C) Criteria are met:
 - A. Significantly delayed gastric emptying as documented by standard scintigraphic imaging of solid food; and
 - B. Patient is refractory or intolerant of 2 out of 3 classes of prokinetic medications and 2 out of 3 antiemetic medications. (see Appendices for classes); and
 - C. Patient's nutritional status is sufficiently low that weight has decreased to 90 percent or less of normal body weight for a patient's height and age in comparison with pre-illness weight.

- II. The replacement of an existing gastric electrical stimulator and/or generator is considered **medically necessary** when the existing gastric electrical stimulator and/or generator is malfunctioning, cannot be repaired, and is no longer under warranty.
- III. Replacement of a gastric electrical stimulator and/or generator is considered **not medically necessary** when Criterion II. is not met.
- IV. Gastric electrical stimulation for the treatment of chronic intractable nausea and vomiting secondary to gastroparesis of diabetic, idiopathic or post-surgical etiology is considered **not medically necessary** when Criterion I. is not met.
- V. Gastric electrical stimulation is **investigational** for all other indications including but not limited to the treatment of obesity.

NOTE: A summary of the supporting rationale for the policy criteria is at the end of the policy.

LIST OF INFORMATION NEEDED FOR REVIEW

It is critical that the list of information below is submitted for review to determine if the policy criteria are met. If any of these items are not submitted, it could impact our review and decision outcome.

- History and Physical/Chart Notes
- Current Symptomology
- Prokinetic and Antiemetic Medications given and response
- Replacement and Revisions
 - Name and type of device requested
 - Documentation of specifically why the stimulator is no longer able to perform its basic function
 - Documentation that the current device cannot be repaired or adapted adequately to meet the patient's needs

CROSS REFERENCES

1. [Bariatric Surgery](#), Surgery, Policy No. 58
2. [Vagus Nerve Stimulation](#), Surgery, Policy No. 74
3. [Vagus Nerve Blocking Therapy for Obesity](#), Surgery, Policy No. 200

BACKGROUND

A subcutaneously implanted pulse generator delivers electrical stimulation to the stomach via intramuscular leads that are implanted on the outer surface of the greater curvature of the stomach either laparoscopically or during a laparotomy. Stimulation parameters are typically programmed at an “on time” (ON) (e.g., 0.1 second) alternating with an “off time” (OFF) (e.g., 5.0 seconds).

GASTRIC STIMULATION FOR THE TREATMENT OF INTRACTABLE NAUSEA AND VOMITING DUE TO GASTROPARESIS

Gastroparesis is a chronic disorder of gastric motility characterized by delayed emptying of a solid meal. Symptoms include bloating, distension, nausea, and vomiting. When severe and chronic, gastroparesis can be associated with dehydration, poor nutritional status, and poor

glycemic control in diabetics. While most commonly associated with diabetes, gastroparesis is also found in chronic pseudo-obstruction, connective tissue disorders, Parkinson disease, and psychological pathology. Idiopathic gastroparesis refers to symptoms of gastroparesis which are not associated with an identifiable cause. Treatment of gastroparesis includes prokinetic agents such as metoclopramide, and antiemetic agents such as metoclopramide, granisetron, or ondansetron. Severe cases may require enteral or total parenteral nutrition.

GASTRIC STIMULATION FOR THE TREATMENT OF OBESITY

GES has also been investigated as a treatment of obesity as a technique to increase a feeling of satiety with subsequent reduced food intake and weight loss. The exact mechanisms resulting in changes in eating behavior are uncertain but may be related to neurohormonal modulation and/or stomach muscle stimulation.

REGULATORY STATUS

The Enterra™ Therapy System (formerly named Gastric Electrical Stimulation [GES] System; manufactured by Medtronic) is the only device approved for treatment of chronic refractory gastroparesis. It received approval for marketing from the U.S. Food and Drug Administration (FDA) in 2000 through the humanitarian device exemption (HDE) process.^[1] This process requires the manufacturer to provide adequate information for the FDA to determine that the device has “probable” benefit but does not pose an unreasonable or significant risk; it does not require data confirming the efficacy of the device. The HDE process is available for devices treating conditions that affect fewer than 4,000 Americans per year.

EVIDENCE SUMMARY

GASTRIC STIMULATION FOR THE TREATMENT OF INTRACTABLE NAUSEA AND VOMITING DUE TO GASTROPARESIS

Systematic Reviews

Several systematic reviews of studies of gastric electrical stimulation (GES) for gastroparesis have been published, the most recent and comprehensive of which was conducted by Levinthal in 2017.^[2-4]

To be included in the Levinthal review, studies had to include adults with established gastroparesis, report patient symptom scores and administer treatment for at least one week. Five randomized controlled trials (RCTs) and 13 non-RCTs meeting criteria were identified. Pooled analysis of data from the five RCTs (n=185 patients) did not find a statistically significant difference in symptom severity when the GES was turned on versus off (standardized mean difference [SMD], 0.17; 95% confidence interval [CI], -0.06 to 0.40; p=0.15). Another pooled analysis did not find a statistically significant difference in nausea severity scores when the GES was on or off (SMD = -0.143; 95% CI, -0.50 to 0.22; p=0.45). In a pooled analysis of 13 open-label single-arm studies and data from open-label extensions of three RCTs, mean total symptom severity score decreased 2.68 (95% CI, 2.04 to 3.32) at follow-up from a mean of 6.85 (95% CI, 6.28 to 7.42) at baseline. The rate of adverse events in the immediate postoperative period (reported in seven studies) was 8.7% (95% CI, 4.3% to 17.1%). The in-hospital mortality rate within 30 days of surgery was 1.4% (95% CI, 0.8% to 2.5%), the rate of reoperations (up to 10 years of follow-up) was 11.1% (95% CI, 8.7% to 14.1%), and the rate of device removal was 8.4% (95% CI, 5.7% to 12.2%).

Randomized Controlled Trials

The data presented to the FDA documenting the “probable benefit” of the GES (Enterra™) system was based on a multicenter double-blind cross-over study referred to as the Worldwide Anti-Vomiting Electrical Stimulation Study (WAVESS).^[1] The study included 33 patients with intractable idiopathic or diabetic gastroparesis. The primary endpoint of the study was a reduction in vomiting frequency, as measured by patient diaries. In the initial phase of the study, all patients underwent implantation of the stimulator and were randomly and blindly assigned to stimulation ON or stimulation OFF for the first month, with crossover to OFF and ON during the second month. The baseline vomiting frequency was 47 episodes per month, which significantly declined in both ON and OFF groups to 23 and 29 episodes, respectively. However, there were no significant differences in the number of vomiting episodes between the two groups, suggesting a placebo effect.

After the first two months of therapy, patients were asked which month of the cross-over stimulation they preferred. Twenty-one of the 33 patients selected the ON mode as their preferred month, compared to 7 who preferred the OFF mode, and 5 who had no preference. The greater preference for ON stimulation suggested some short-term effect that was not placebo.

In a continuing open phase of the trial, the patients then received the stimulation consistent with their preference. However, by four months all patients had the device turned ON (it was not clear whether this phase was by preference or design). At 6 and 12 months follow-up, the mean number of vomiting episodes continued to decline, although only 15 patients were followed for a period of 12 months. Data regarding quality of life were also obtained at 6 and 12 months and showed improvement. At 6 months, there was a significant improvement in 2-hour gastric retention (from 80% retention to 60% retention), but not in 4-hour gastric retention. (Fifty percent gastric retention at two hours was considered the upper limits of normal.)

The results of the randomized portion of the study suggest a placebo effect. Therefore, long-term results of GES must be validated in a longer-term randomized trial. It is interesting to note that GES did not return gastric emptying to normal in the majority of the patients tested. In as much as the device is intended to improve gastric emptying, as a proof of principle, it would be interesting to investigate the correlation between the degree of gastric emptying and symptom improvement.

In a 2003 update to WAVESS, Abell reported 12-month outcomes for all of the patients.^[5] Statistically significant improvements were found for weekly vomiting frequency, total abdominal symptom score, and scintigraphic solid food emptying. At baseline the median vomiting frequency was 17.3 episodes per week with gastroparetic symptoms over a mean of 6.2 years. All patients had scintigraphic evidence of delayed gastric emptying at 2 and 4 hours, all patients were refractory to prokinetic and antiemetic medications, and 14 required some form of parenteral or enteral feedings. Results at the end of phase 1 (the blinded phase) showed a 50% decreased vomiting frequency for patients whose devices were ON compared to patients whose devices were OFF ($p=0.05$).

Symptom severity trended toward improvement in the ON versus OFF period, although these changes did not reach statistical significance in phase 1. In a second phase of the study all patients were switched to the ON position with 6- and 12- months follow-up. Vomiting at 12 months was compared to baseline; 72% for the combined group, 63% for diabetics with gastroparesis, and 83% for patients with idiopathic gastroparesis. Total symptom score

improved significantly ($p < 0.05$) at 6 and 12 months. Physical and mental quality of life scores improved significantly compared to baseline ($p =$ less than 0.025). Baseline gastric retention was 78% at 2 hours. This decreased significantly with electrical stimulation to 65% at 6 months and 56% at 12 months for the combined group. The changes in 2-hour gastric emptying were not significant for the diabetic and idiopathic groups separately. Four-hour gastric emptying improved from 34% retention at baseline to 22% retention at 12 months. The difference was statistically significant for the combined group as well as the diabetic and idiopathic groups separately.

McCallum (2010) performed a multicenter prospective study to evaluate Enterra™ therapy in patients with chronic intractable nausea and vomiting from diabetic gastroparesis (DGP).^[6] In this study, 55 patients with refractory DGP (5.9 years of DGP) were implanted with the Enterra™ system. After surgery, all patients had the stimulator turned ON for 6 weeks and then were randomly assigned to groups that had consecutive 3-month cross-over periods with the device ON or OFF. After this period, the device was turned ON in all patients and they were followed up unblinded for 4.5 months. During the initial 6-week phase with the stimulator turned ON, the median reduction in weekly vomiting frequency (WVF) compared with baseline was 57%. There was no difference in WVF between patients who had the device turned ON or OFF during the 3-month cross-over period. At 1 year, the WVF of all patients was significantly lower than baseline values (median reduction, 68%; $P < 0.001$). One of the patients had the device removed due to infection; 2 patients required surgical intervention due to lead-related problems.

In a later study, McCallum (2013) evaluated GES (Enterra™ system) in patients with chronic vomiting due to idiopathic gastroparesis in a randomized, double-blind crossover trial.^[7] In this study, 32 patients with nausea and vomiting associated with idiopathic gastroparesis, which was unresponsive or intolerant to prokinetic and antiemetic drugs, received Enterra™ implants and had the device turned on for 6 weeks. Subsequently, 27 of these patients were randomized to have the device turned on or off for 2 consecutive 3 month periods. Twenty five of these subjects completed the randomized phase; of note, 2 subjects had the device turned on early, 2 subjects had randomization assignment errors, and 1 subject had missing diaries. During the initial 6-week on period, all subjects demonstrated improvements in their WVF, demonstrating a median reduction of 61.2% compared with baseline (17.3 episodes/week at baseline vs 5.5 episodes/week at 6 week postimplant, $p < 0.001$). During the on-off crossover phase, subjects demonstrated no significant differences between the on and off phase in the study's primary end point, median WVF (median 6.4 in the on phase vs 9.8 in the off phase; $p = 1.0$). Among the 19 subjects who completed 12 months of follow up, there was an 87.1% reduction in median WVF compared with baseline (17.3 episodes/week at baseline vs 2 episodes/week at 12-month follow-up, $p < 0.001$). Two subjects required surgical intervention for lead migration/dislodgement or neurostimulator migration.

Nonrandomized Studies

Laine (2018) published a retrospective, multicenter analysis of patients with severe, medically refractory gastroparesis who received GES.^[8] Fourteen patients (11 diabetic, 1 idiopathic, and 2 postoperative) treated in Finland between 2007 and 2015 were included; median follow-up was 3 years. Eight (57.1%) patients experience marked relief of gastroparesis symptoms, while 3 (21.4%) patients experience partial relief. There was a median weight gain of 5.1 kg in 11 (78.6%) patients after GES implantation, and, at last possible follow-up, 5 out of 10 (50%)

patients were without medication for gastroparesis. The study was limited by its retrospective nature, small population size, and relatively short follow-up time.

Shada (2018) published a prospective study of patients with medically refractory gastroparesis who underwent implantation of GES between 2005 and 2016.^[8] One hundred nineteen patients (64 diabetic, 55 idiopathic), with mean follow-up of 39.0 ± 32.0 months, were included in the analysis. Before GES placement, operatively placed feeding tubes were present in 22% of diabetic and 17% of idiopathic patients, however, after GES placement, 67% of feeding tubes were removed. Due to a perceived lack of benefit, 8 patients decided to have their GES device removed after a mean time of 36 ± 29 months. Also, there was significant improvement in GCSI scores for both diabetic ($p=0.01$) and idiopathic ($p=0.003$) subgroups at ≥ 2 years after implantation. The study was limited by its not all patients being administered the GCSI before GES, and a number of patients being lost to follow-up.

In 2016, Heckert reported on GES as a treatment for refractory symptoms of gastroparesis in 138 patients (65 diabetic, 68 idiopathic, and 5 other) with delayed gastric emptying at one-year follow-up (1.4 ± 1.0 years).^[9] Patients reported their response to GES using the Clinical Patient Grading Assessment Scale (CPGAS), of which, 75% of patients felt their symptoms had improved, and 25% felt their symptoms were the same or worsened (diabetics had a greater response than idiopathic patients). Symptom severity was assessed by analyzing Patient Assessment of GI Symptoms (PAGI-SYM) questionnaires, before insertion of GES and at the last follow-up visit. PAGI-SYM scores were improved for all symptoms, though the authors report nausea, early satiety and loss of appetite to have been most improved; and constipation, diarrhea, and abdominal distension to have been least improved. In this selected group of patients, the authors concluded GES to be beneficial in the majority of patients.

In 2013, Keller reported complication rates and need for a second surgery in 233 patients who had GES implantation surgery over a ten year period at a single institution.^[10] Additional surgery was required in 58% of patients. The majority of reoperations were due to the following complications: nutritional access (45 patients, requiring 77 procedures), subcutaneous pocket issues ($n = 21$), gastroparetic symptoms ($n = 11$), mechanical issues ($n = 9$) and infection ($n = 4$). The study reported that patient BMI was predictive of additional surgeries, with 4.45 overall increased risk of pocket revision surgery. Although 70% of patients reported improved symptoms of pain, bloating and nausea, GES had a significantly high reoperation rate due to complications associated with the initial procedure.

In 2007, Anand reported on a study of 214 consecutive drug-refractory patients with the symptoms of gastroparesis (146 idiopathic, 45 diabetic, 23 after surgery).^[11] A GES device was implanted in 156 patients. The remaining 58 patients, designated as the control group, were either on the waiting list for permanent implantation or consented to not receive a permanent implant. At last follow-up (median 4 years), most patients who received implants (135 of 156) were alive with intact devices, significantly reduced gastrointestinal symptoms, and improved health-related quality of life, with evidence of improved gastric emptying. Also, 90% of the patients had a response in at least 1 of 3 main symptoms. Most patients that explanted, usually for pocket infections, were later successfully reimplanted.

GES placement using minimally invasive surgical approaches has also been evaluated in several publications. Laparoscopy has been reported in at least two studies as a feasible approach in placement of GES for patients with medically refractory diabetic or idiopathic gastroparesis.^[12, 13]

Several small case series and retrospective reviews have been reported, some with long-term outcomes up to 5 years.^[12, 14-30] The data indicate that GES may be associated with improvements in gastrointestinal symptom scores, nutrition and quality-of-life for patients; these improvements were sustained over time. However, gastric emptying rates were mixed.

Adverse Events

In 2017, Bielefeldt analyzed the number, severity and type of voluntarily reported adverse events related to Enterra™ in the Manufacturer and User Device Experience (MAUDE) databank of the FDA.^[31] Data were retrieved for 2001 through October 31, 2015, of which 1472 reports were abstracted. Thirty-six perioperative complication reports were reviewed; six were serious events, including three deaths (one due to cardiac arrest, two due to septic complications with resulting multi organ failure), one stroke, and one myocardial infarction complicated further by a pulmonary embolism. Overall, most of the reports were regarding patient concerns, local complications, or system failure. Limitations of these findings include reporting bias (the MAUDE data are voluntarily submitted), and report misclassification bias (MAUDE data sources vary from patient reports to published articles and inconsistencies in reporting have been found). Risk-benefit could not directly be assessed given the nature of the MAUDE database, though the author cites other studies for outcomes measurement, most of which are included in the other sections of this evidence review. Overall, 35% of the reported adverse events prompted an additional surgery.

Section Summary

The evidence regarding the clinical utility of GES for gastroparesis due to intractable nausea and vomiting is limited to three small crossover RCTs. However, longer-term data suggest improvements in gastrointestinal symptom scores, nutrition, and quality-of-life scores, suggesting some benefit with GES treatment. Given the lack of alternative treatment options in this specific patient population, GES may be considered reasonable treatment of symptoms of gastroparesis.

GASTRIC STIMULATION FOR THE TREATMENT OF OBESITY

Systematic Review

In 2014, Cha published a review of 33 studies evaluating various methods of gastric stimulation as a treatment of obesity, including implantable GES.^[32] The majority of included studies were small in nature with 24 studies evaluating 30 or fewer patients. In addition, many of the studies reported high dropout rates of more than 50% of patients at the end of the study follow-up period. A major limitation of the review was the inclusion of studies which did not include the treatment of obesity (i.e., BMI or weight loss) as a primary outcome measure. Furthermore, there were methodological difference in the patient inclusion criteria and most of the studies included in the review were limited by short-term follow-up of less than one year. The authors concluded that the level of evidence regarding GES as a treatment of obesity was low. Long-term RCTs which compare GES to other treatments of obesity and sham are needed in order to assess the safety and efficacy of GES in this population.

Randomized Controlled Trials

There is one published RCT on GES for the treatment of obesity. In 2009, Shikora reported on a randomized controlled, double-blind study (SHAPE trial) to evaluate GES for the treatment of obesity.^[33] All 190 patients participating in the study received an implantable gastric stimulator

and were randomized to have the stimulator turned on or off. All patients were evaluated monthly, participated in support groups and reduced their diet by 500-kcal/day. At 12-month follow-up, there was no difference in excess weight loss between the treatment group (weight loss of 11.8% +/- 17.6%) and the control group (weight loss of 11.7% +/- 16.9%) using intention-to-treat analysis ($p=0.717$).

Nonrandomized Studies

Additional, small studies – including one patient population with comorbidities of gastroparesis and morbid obesity – have reported positive outcomes in weight loss and maintenance of weight loss along with minimal complications.^[34-39] However, due to lack of long-term outcomes from well-designed randomized clinical trials, conclusions cannot be made concerning the safety and efficacy of chronic gastric stimulation as a treatment for morbid obesity.

PRACTICE GUIDELINE SUMMARY

AMERICAN COLLEGE OF GASTROENTEROLOGY^[40]

The American College of Gastroenterology (ACG) published a clinical practice guideline on management of gastroparesis in 2013. The recommendations for this guideline were based on review of the evidence-base through 2011. The ACG concluded that GES treatment does not adequately address the clinical needs of these patients, but that, “GES may be considered for compassionate treatment in patients with refractory symptoms, particularly nausea and vomiting. Symptom severity and gastric emptying have been shown to improve in patients with diabetic gastroparesis (DG), but not in patients with idiopathic gastroparesis (IG) or postsurgical gastroparesis (PSG). (Conditional recommendation, moderate level of evidence.)”

SUMMARY

It appears that gastric electrical stimulation (GES) may improve intractable nausea and vomiting for patients with gastroparesis. Clinical guidelines based on research state GES may be considered for compassionate treatment in patients with refractory symptoms, particularly nausea and vomiting. Therefore, given the lack of treatment options in this very specific patient population, GES may be medically necessary in carefully selected patients with gastroparesis when policy Criteria are met. GES for the treatment of chronic intractable nausea and vomiting secondary to gastroparesis of diabetic, idiopathic or post-surgical etiology is considered not medically necessary when policy Criteria are not met.

There is limited evidence on the efficacy and safety gastric electrical stimulation for any other indication including but not limited to the treatment of obesity. There are no clinical practice guidelines that recommend the use of gastric electrical stimulation for any other indication. Therefore, the use of electrical gastric stimulation for all other indications including treatment for obesity are considered investigational.

In certain situations, a stimulator may require revision after it has been placed. In these cases, revision may be medically appropriate to allow for the proper functioning of the device. Therefore, revision(s) to an existing gastric electrical stimulator may be considered medically necessary after the device has been placed.

In certain situations, a gastric electrical stimulator may no longer be able to perform its basic function due to damage or wear. When a gastric electrical stimulator is out of its warranty period and cannot be repaired adequately to meet the patient's medical needs, replacement of the device may be medically appropriate. Therefore, replacement of all or part of a gastric electrical stimulator may be considered medically necessary when device replacement Criteria are met.

When a gastric electrical stimulator is in its warranty period or can be repaired or adapted adequately to meet the patient's medical needs, replacement of the device is not medically appropriate. Therefore, replacement of all or part of a gastric electrical stimulator is considered not medically necessary when device replacement Criteria are not met.

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CODES

NOTES:

- The CPT coding manual indicates that procedures related to laparoscopic gastric stimulation electrodes for morbid obesity should be reported using code 43659 - Unlisted laparoscopy procedure, stomach
- HCPCS code C1823 is NOT the correct code to use for reporting these services. Please refer to the codes listed below for guidance.

Codes	Number	Description	
CPT	43647	Laparoscopy, surgical; implantation or replacement of gastric neurostimulator electrodes, antrum	
	43648	Laparoscopy, surgical; revision or removal of gastric neurostimulator electrodes, antrum	
	43659	Unlisted laparoscopy procedure, stomach	
	43881	Implantation or replacement of gastric neurostimulator electrodes, antrum, open	
	43882	Revision or removal of gastric neurostimulator electrodes, antrum, open	
	43999	Unlisted procedure, stomach	
	64590	Insertion or replacement of peripheral or gastric neurostimulator pulse generator or receiver, direct or inductive coupling.	
	64595	Revision or removal of peripheral or gastric neurostimulator pulse generator or receiver	
	95980	Electronic analysis of implanted neurostimulator pulse generator system (eg, rate, pulse amplitude and duration, configuration of wave form, battery status, electrode selectability, output modulation, cycling, impedance and patient measurements) gastric neurostimulator pulse generator/transmitter; intraoperative, with programming	
	95981	;subsequent, without programming	
	95982	;subsequent, with reprogramming	
	HCPCS	C1767	Generator, neurostimulator (implantable), nonrechargeable
		C1778	Lead neurostimulator
		C1883	Adaptor/Extension, pacing lead or neurostimular lead (implantable)
C1897		Lead neurostimulator test kit (implantable)	
E0765		FDA approved nerve stimulator, with replaceable batteries, for treatment of nausea and vomiting	
L8679		Implantable neurostimulator, pulse generator, any type	
L8680		Implantable neurostimulator electrode, each	
L8685		Implantable neurostimulator pulse generator, single array, rechargeable, includes extension	
L8686		;non-rechargeable, includes extension	
L8687		Implantable neurostimulator pulse generator, dual array, rechargeable, includes extension	
L8688	;non-rechargeable, includes extension		

Appendix 1: Prokinetic Medications

Class	Common Examples
Cholinergic Agonists	dexpanthenol (Ilopan®), bethanechol (Urecholine®)
Motilin receptor agonists	erythromycin
Dopamine receptor antagonists	metoclopramide (Reglan®)

Appendix 2: Antiemetic Medications

Class	Common Examples
Antihistamines	diphenhydramine (Benadryl®), dimenhydrinate (Dramamine®), meclizine (Antivert®), hydroxyzine (Vistaril®), trimethobenzamide (Tigan®)
Serotonin (5HT ₃) receptor antagonists	ondansetron (Zofran®), granisetron (Kytril®), dolasetron (Anzemet®)
Dopamine receptor antagonists	Metoclopramide (Reglan®), perphenazine (Trilafon®), prochlorperazine (Compazine®),

Appendix 2: Antiemetic Medications

	promethazine (Phenergan®), thiethylperazine (Torecan®), cyclizine (Marezine®)
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Date of Origin: February 2001