



Horizon Scanning Technology Prioritising Summary

Enterra® Therapy Gastric Electrical Stimulation
(GES) system for the treatment of the
symptoms of medically refractory gastroparesis

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This Horizon scanning prioritising summary was prepared by staff from the Australian safety and Efficacy Register of New Interventional Procedures – Surgical (ASERNIP-S).



Horizon Scanning Technology Prioritising Summary

Name of Technology:

Enterra® Therapy Gastric Electrical Stimulation (GES) system for the treatment of the symptoms of medically refractory gastroparesis.

Purpose and Target Group:

Implanted through laparoscopy or laparotomy, the Enterra® Therapy system acts as an electrical 'pacemaker' for the stomach, stimulating smooth muscle contractions and alleviating the symptoms of delayed gastric emptying. The system is indicated for the treatment of intractable nausea and vomiting symptoms in gastroparetic patients refractory to conventional medication

(http://www.cpmc.org/services/gi/services/gastric_electrical_stim.pdf). It has particular application in cases of severe gastroparesis, where symptoms have been refractory to medical treatment for at least 12 months (Smith & Ferris 2003).

Stage of Development (in Australia): Not yet emerged in Australia.

- Experimental
- Investigational
- Nearly established
- Established
- Established but changed indication or modification of technique
- Should be taken out of use

The Enterra® system is not listed or registered in the Australian Register of Therapeutic Goods (ARTG).

International Utilisation:

COUNTRY	LEVEL OF USE		
	Trials underway	Limited use	Widely diffused
U.S.		✓	
Europe		✓	

Impact Summary:

Background

Gastroparesis is a chronic condition of the stomach, which is thought to be caused by injury to the vagus nerve (most commonly due to the effects of diabetes, but also from surgical and idiopathic causes). The condition is characterised by impaired gastric motility, which results in delayed gastric emptying. Weakened gastric muscle contractions and slow stomach emptying produce chronic dyspeptic symptoms, including nausea, vomiting, excessive



fullness and reflux (Lin & McCallum 2003; gihealth 2005). In severe cases, chronic vomiting and discomfort associated with food intake can lead to malnutrition and dehydration (Lin & McCallum 2003). In such cases, long-term medical intervention is usually required in order to sustain a patient's nutrition and/or alleviate symptoms.

Conventional treatment options for gastroparesis currently involve lifestyle and nutritional modifications, antiemetic and prokinetic drug therapy, and 'last resort' surgical treatment and nutritional support (Buckles & Forster 2003; Smith & Ferris 2003). Modifications to diet and lifestyle aim to maintain adequate levels of nutrition and hydration (Buckles & Forster 2003). However, this approach may not be sufficient to prevent chronic nausea and vomiting in more severe cases of gastroparesis.

The symptoms of gastroparesis have traditionally been treated through pharmacotherapy. Antiemetic medication prevents nausea and vomiting in gastroparetic patients, while prokinetic agents help to improve gastric motility (Buckles & Forster 2003). Currently, drugs used for treating gastroparesis in Australia include metoclopramide, erythromycin, domperidone and cisapride (Buckles & Forster 2003). However, it should be noted that the drug cisapride was deleted from the Pharmaceutical Benefits Schedule on August 1 2004 (Department of Health and Ageing 2004). While pharmacotherapy may be useful in treating the symptoms of gastroparesis, limited evidence exists on the efficacy of these drugs (Smith & Ferris 2003). Moreover, not all patients respond well to drug treatment. Side-effects, drug availability and safety issues (such as the slight risk of cardiotoxicity in patients using cisapride) can be prohibitive factors, and patients with severe gastroparesis may simply be refractory to pharmacotherapy (Hebbard *et al.* 2001).

Surgical interventions and nutritional support have been used as a 'last resort' in cases of severe gastroparesis. Endoscopic or surgical venting gastrostomies are sometimes considered in the treatment of drug refractory gastroparesis. This procedure enables periodic venting of the stomach and alleviation of symptoms. Although studies have shown this procedure to be effective, it also carries a significant risk of infection, with as many as 25% of patients experiencing an infection within the first month of percutaneous tube placement (Smith & Ferris 2003). Gastrectomy with Roux-en-Y reconstruction surgery has also occasionally been considered in the treatment of gastroparesis. While this radical procedure has been helpful in some diabetic patients with severe gastroparesis, it is considered a last resort only (Smith & Ferris 2003).

J-tube feeding aims to restore nutrition and hydration and improve quality of life in frequently hospitalised patients refractory to medication and surgical treatment. Once implanted, J-tubes facilitate enteral nutritional intake at night, thus enabling patients to function during the day (Buckles & Forster 2003). While J-tubes can be useful for restoring nutrition in severe cases, they are frequently dislodged, and the risk of sepsis and other complications increases with long-term tube placement. More importantly, J-tube feeding



does nothing to address the symptoms of gastroparesis (Buckles & Forster 2003; Smith & Ferris 2003).

The concept of ‘gastric pacing’ for the treatment of impaired motility dates back to early studies published in the 1960s (Lin & McCallum 2003; Smith & Ferris 2003). Since gastric motility is regulated by a natural pacemaker, it was hypothesised that disorders of gastric motility could be treated by entraining the natural gastric myoelectric rhythm (GMA) with an artificial pacemaker (Lin & McCallum 2003; Buckles & Forster 2003). This natural GMA is known as the ‘gastric slow wave’. Contractions of gastric smooth muscles are generated whenever spikes of GMA interact with this background slow wave cycle (Bortolotti 2002; Lin & McCallum 2003). In patients with gastroparesis, the slow wave cycle is impaired or irregular, effectively reducing the contractility of the stomach (Lin & McCallum 2003; Strickland 2003).

Enterra® Therapy was developed with a similar concept in mind. By delivering high frequency/low energy electrical impulses to nerves in the lower stomach, Enterra® Gastric Electrical Stimulation (GES) may be able to relieve symptoms associated with delayed gastric emptying (Strickland 2003; Lin & McCallum 2003). However, the correlation between symptom outcomes and gastric motility after GES treatment remains unclear. While studies have generally shown a decrease in symptoms following GES treatment, most patients experience no long-term improvement in gastric motility (Buckles & Forster 2003; Bouras 2005). One theory is that GES may have a neural effect, creating changes in the autonomic nervous system and hormone levels which might play a role in regulating nausea and vomiting (Buckles & Forster 2003).

The Enterra® GES system consists of a 2.6 cm long, 5.8 cm wide stimulation device and two intramuscular electrode leads

(http://www.cpmc.org/services/gi/services/gastric_electrical_stim.pdf).

The stimulator is implanted in the abdominal wall by laparoscopy or laparotomy, and the leads are typically implanted in the smooth muscle along the greater curvature of the antrum (Buckles & Forster 2003). Endoscopic monitoring during implantation is required in order to ensure proper placement and prevent lead penetration (Buckles & Forster 2003).

Clinical Need and Burden of Disease

Gastroparesis is more prevalent among women than men, and commonly occurs as a complication of longstanding type 1 and type 2 diabetes (Buckles & Forster 2003; Samson *et al.* 2003; Smith & Ferris 2003). It is thought to affect 20 to 50% of diabetic patients, although the disorder tends to have a higher prevalence in groups with type 1 and/or poorly managed diabetes (Buckles & Forster 2003; gihealth 2005). Gastroparesis can also develop as a result of surgical and idiopathic causes, as well from conditions such as Parkinson’s disease, scleroderma, and intestinal pseudo-obstruction (Buckles & Forster 2003). Idiopathic gastroparesis accounted for approximately 36% of 146 cases of gastroparesis in a long-term



study by Soykan *et al.* (1998). Viral infection is thought to be a major cause of gastroparesis in idiopathic cases (Buckles & Forster 2003).

Severe gastroparesis results in significant impairments in health and quality of life. In diabetic patients, gastroparesis can compound the ill-effects of diabetes and contribute to mortality by increasing blood glucose fluctuations (gihealth 2005). More generally, gastroparesis can result in malnutrition, dehydration, anorexia and weight loss, frequent hospitalisation, difficulties in maintaining employment, and an overall loss of freedom and mobility (Lin & McCallum 2003).

Estimated Speed, Geographic and Practitioner Use, Patterns of Diffusion in the Health System

It is estimated that as many as 50% of all patients referred for treatment of severe gastroparesis are eligible for Enterra® Therapy (Medtronic 2003; Buckles & Forster 2003). However, at its current stage of distribution, it is unlikely that Enterra® Therapy is used in such a high percentage of cases.

The FDA approved the Enterra® Therapy device for limited use in the United States under the Humanitarian Use Device (HUD) exemption in April 2000 (Strickland 2003). Under this exemption, Medtronic Inc. is permitted to manufacture Enterra® without the usual requirement of establishing evidence for the efficacy of the device (Strickland 2003). This is on the condition that Enterra® is manufactured and sold for use in treating no more than 4000 new cases of gastroparesis per year in the United States (Strickland 2003). Medtronic Inc. is currently in the process of establishing an efficacy trial which will help the company to apply to the FDA for full premarket approval (PMA) of Enterra® (Keith-Ferris 2004). If PMA is obtained, it will enable increased use of Enterra® Therapy throughout the U.S.

In 2002, Enterra® received CE Mark approval for commercial use in Europe (Medtronic 2003). It is unclear how widely Enterra® Therapy is employed for the treatment of gastroparesis in European countries, although it does seem to be used in the U.K. at present (<http://www.abc.net.au/rn/talks/8.30/helthrpt/stories/s1232837.htm>).

Existing Comparators

- Dietary and behavioural modifications
- Pharmacotherapy (prokinetic and antiemetic medications)
- Surgery (gastrotomies and gastrectomies)
- Nutritional support through J-tube feeding

Estimated Cost Impact

The current cost of implanting the Enterra® system in the U.S. is approximately US\$24 000 per patient per year (Friedman 2002). Inclusive of total hospital costs, Enterra® Therapy



treatment amounts to around £15 000 to £16 000 per patient in the U.K (Australian Broadcasting Corporation 2004). However, these high initial costs could be offset by long-term reductions in hospitalisation and health care costs. Reduced hospital stay from GES treatment is estimated to amount to a saving of US\$60 000 per patient per year (Friedman 2002; Strickland 2003). A recent study by Cutts *et al.*. (2005) comparing gastroparetic patients receiving GES and those receiving pharmacotherapy also found that health care costs for the GES group decreased over time ($P < 0.001$), while those receiving standard medical treatment encountered no such decrease ($P = 0.19$).

Costs associated with this new product in Australia are not currently available. The reimbursement fee for percutaneous gastrostomy is approximately \$302.95 (Medicare Benefits Schedule item number 30481) (<http://www.health.gov.au>). An estimate of the reimbursement fees for implantation of the Enterra® system (if it becomes an established and routine procedure in Australia) can be obtained by examining other examples of nerve stimulation (eg. Sacral nerve stimulation) within the Medicare Benefits Schedule. The placement of sacral nerve leads (Item number 32213), neurostimulators/receivers (Item number 32214), and adjustment of sacral nerve electrodes (Item number 32215) for the treatment of faecal incontinence has MBS reimbursements of \$572.05, \$289.00 and \$108.05. The reimbursement fee for Enterra® may be similar to these procedures.

Even assuming that about 50% of diabetic patients have gastroparesis in some form, it is difficult to arrive at a general figure on the extent of service claims for the treatment of severe gastroparesis in Australia. The most accurate estimate of claims might come from figures on the prescription of the drug cisapride, which was strictly indicated by the Pharmaceutical Benefits Schedule 2004 for the treatment of diagnosed cases of gastroparesis (Hebbard *et al.* 2001). According to Medicare Australia, a total of 6 281 claims were processed between July 2004 and July 2005 for the prescription of cisapride (including dosages of 5 mg, 10 mg and 200 ml (1 mg per ml) oral suspension) (<http://www9.health.gov.au/mbs/>).

Efficacy and Safety Issues

List of Studies Found

Total number of studies	12
Randomised controlled trials	1 (3 articles)
Non-randomised comparative studies	2
Case series studies	8
Case reports	1

The studies included in this summary are highlighted in bold in the reference list.

Safety and efficacy data from one randomised controlled trial (RCT) and two non-randomised comparative studies have been selected for inclusion in this summary. Eight case series and one case report were selected for their safety data.



The U.S. randomised, double-blind cross-over trial ‘World Wide Anti-Vomiting Electrical Stimulation Study’ (WAVESS) (n=33) reported no difference in mean vomiting frequency per week between groups assigned to device ON (23.0±35.5 vomiting episodes) and OFF (29.0±38.2 vomiting episodes) during a two-month cross-over period (FDA 2000; Henney 2000). Compared to baseline (47.6±52.6 episodes of vomiting), both groups experienced a decrease in vomiting frequency per week. This indicates that other factors may have been involved in symptom reduction, and that gastric stimulation did not affect vomiting frequency (FDA 2000). Nevertheless, patients did show a significant (P <0.05) preference for the ON versus OFF setting at the end of the cross-over period (Abell *et al.* 2003).

At the end of the two-month crossover period, the 25 remaining patients had their device set to ON, according to their preference for that setting (FDA 2000; Henney 2000). Vomiting frequency at six months follow-up (n=25) decreased from a mean of 44.6±50.7 vomiting episodes per week at baseline, to 19.2±43.7 vomiting episodes per week, amounting to a 57% mean difference from baseline (FDA 2000). For the remaining 15 patients at 12 months follow-up, mean vomiting frequency decreased from 42.7±53.9 episodes per week at baseline to 10.1±9.8 episodes per week, amounting to a 77% mean difference from baseline (FDA 2000; Henney 2000).

While a significant improvement (P <0.05) in symptom severity and quality of life scores was reported in the WAVESS RCT, no significant improvement in gastric emptying was observed (Abell *et al.* 2003). This again suggests that mechanisms other than gastric motility may be involved in symptom relief.

A recent non-randomised comparative study by Cutts *et al.* (2005) (n=18)* reported a significant overall improvement in total symptom score (TSS)[†] in the group receiving GES treatment (n=9) in comparison to baseline. Overall TSS improvement for the GES group was also significantly higher (P <0.017) than in the group receiving intensive medical therapy (MED) through medication (n=9) (Cutts *et al.* 2005). At 12 months follow-up, TSS had significantly improved for both the GES and MED groups compared to baseline (Cutts *et al.* 2005). However, while the GES group continued to show significant improvement (P <0.001) at subsequent follow-ups, the MED group showed no significant improvement in TSS after the first 12 months (Cutts *et al.* 2005).

Health care resource usage in this study was assessed through the investigator-derived independent outcome measure score (IDIOMS)[‡]. Cutts *et al.* (2005) reported that overall IDIOMS was significantly better (P <0.017) for the GES group than the MED group. While for the GES group, IDIOMS compared to baseline (mean 12.6±1.6) significantly improved at each follow-up (mean 8.3±1.4 at one year; 7.0±1.13 at two years; 6.4±1.03 at three years) group, there was a significant worsening of IDIOMS compared to baseline (mean

* Note: some patient overlap with two unspecified FDA trials of Enterra® GES (Cutts *et al.* 2005)

[†] ‘TSS’ represents a patient’s self-assessment of early satiety, abdominal pain/bloating/distention, nausea, and vomiting (Cutts *et al.* 2005). It is not yet a validated tool (Bouras 2005).

[‡] IDIOMS rates severity of illness, intensity of service and other organ involvement (Cutts *et al.* 2005). Not a validated tool. (Bouras 2005).



11.0±0.71) for the MED group at two years (mean 13.3±0.62) and three years (mean 13.8±0.45) follow-up (Cutts *et al.* 2005). This may have been partially due to the high mortality rate of patients in the MED group (3/9 MED patients died of complications in intravenous access) (Cutts *et al.* 2005). No deaths were reported in the GES group during the three-year period (Cutts *et al.* 2005).

Reduced hospitalisation was also reported in the Cutts *et al.* (2005) study. Mean inpatient hospital days decreased for both groups compared to baseline. However, there was no significant difference in mean hospital stay between GES and MED patients (Cutts *et al.* 2005). Mean inpatient hospital days for the GES group decreased from 24.8 days at baseline to 2.76 days at 36 months follow-up, compared to 26.8 days at baseline and 6.48 days at 36 months for the MED group (Cutts *et al.* 2005).

Abell *et al.* (2002) conducted a short comparative study on its case series of patients who had undergone GES treatment for six months. Twenty-five patients in this series consented to having their devices deactivated for one week at their six-month follow-up. Before the end of the week, seven patients had to have their devices reactivated due to increased symptoms (Abell *et al.* 2002). For the remaining 18 patients, 44% (8/18) reported increased symptoms during deactivation; 44% (8/18) experienced no change, and 11% (2/18) reported less symptoms during deactivation when compared to the stimulation period (Abell *et al.* 2002).

Evidence on the safety profile of Enterra® has been published in a wide array of case series and FDA reports. In Phase II of the WAVESS RCT, neurostimulator pocket infection was reported in two patients (6%) with idiopathic gastroparesis (Abell *et al.* 2003). One diabetic patient had their device removed after the pulse generator eroded through the skin, one idiopathic patient experienced pain due to lead perforation of the stomach, leading to device removal, and another idiopathic patient experienced discomfort from a migrated pulse generator (Abell *et al.* 2003). This patient subsequently underwent surgery to reposition the generator (Abell *et al.* 2003).

As part of the Medtronic, Inc. application to the FDA, adverse events were recorded through September 30, 1999 from a total of 51 patients participating in two clinical trials the United States, Europe and Canada (FDA 2000). Part of this sample includes 18 patients from the Compassionate Use Study (FDA 2000), which was specifically designed to assess the safety of Enterra® for submission to the FDA. Of the 247 adverse events recorded, 13 events (5%) in 11/51 (22%) patients were directly device- or implant-related (the other events were attributed to unrelated diseases and complications) (FDA 2000). Device infection was seen in 4% (2/51) of patients; perforation of the stomach wall was reported in 2% (1/51) of patients; 12% (6/51) of patients (7 events) had a lead impedance out of range; 2% (1/51) encountered device erosion, and 2% (1/51) of patients (2 events) experienced migration of the pulse generator (FDA 2000). Surgical intervention was required for all cases of device infection, device erosion, stomach wall perforation and device migration (FDA 2000). In 2004, an adverse event report was submitted to the FDA outlining a single case of bowel obstruction caused by entanglement of the bowel with the Enterra® electrode wires



(FDA 2004). After re-positioning and securing the wires, no further complications occurred (FDA 2004).

Abell *et al.* (2002) reported adverse events in its case series of 38 patients. Device removal was required in four (12%) of the initial 33 device implants, due to device infection. Electrode dislodgement occurred in 5% (2/38) of patients, and 1/38 patients (3%) experienced electrical stimulation of the abdominal rectus. At 12 months follow-up, 3/27 patients (11%) had their device removed due to erosion or infection, and 3/27 (11%) patients underwent total gastrectomies due to an increase in the severity of their original symptoms. Inadvertent deactivation of the pulse generator was reported in 10/33 (30%) patients. Three out of 33 (9%) patients died during the course of this study from causes unrelated to the procedure. One patient died at 22 months from complications related to a renal and pancreatic transplantation, one patient died from heart failure, and another patient died from lung cancer (Abell *et al.* 2002).

In the Forster *et al.* (2003) case series, 4/55 (7%) patients had their devices removed, 2/55 (4%) due to infection, 1/55 (2%) due to a small bowel volvulus surrounding the electrodes, and 1/55 (2%) due to secondary infection resulting from improper securing of the device and subsequent dislodgement (Forster *et al.* 2003). In the case of bowel volvulus, the patient underwent a small bowel resection and has since recovered without complication (Forster *et al.* 2003). Six out of 55 (11%) patients (all diabetic) died post-implantation, 1/55 (2%) during the immediate postoperative period (Forster *et al.* 2003). This was due to a pulmonary embolus thought to be caused by very limited physical activity before undergoing surgery (Forster *et al.* 2003). One patient (2%) died three months postoperatively after refusing dialysis. One patient (2%) died of a myocardial infarction and one patient of aspiration pneumonia nine months post-implantation (Forster *et al.* 2003). At 14 months, one patient (2%) committed suicide. At 19 months, one patient (2%) died from diabetic complications (Forster *et al.* 2003).

Lin *et al.* (2004) reported a single case of device pocket infection out of 15 patients. The device was removed after three months (Lin *et al.* 2004). A single case of device infection and subsequent removal was noted by McCallum *et al.* (2005). Anand *et al.* (2004) reported device removal due to infection in 8/133 (6%) implants, and 13/133 (10%) deaths, none of which were 'directly' caused by the devices. McCallum *et al.* (2004) reported device infection and removal in 3/45 (7%) patients. The case report by Brody *et al.* (2004) reported abdominal pain in one patient. This was caused by a dense adhesion of the seromuscular tunnel of the electrodes to the abdominal wall (Brody *et al.* 2004)

There is some evidence for the safety and effectiveness of Enterra® Therapy. The studies suggest that Enterra® GES may be effective in treating the symptoms of medically refractory gastroparesis, however, the study samples were small. The correlation between GES, gastric motility and symptoms remains unclear. A lack of statistically significant difference in mean vomiting frequency between ON and OFF groups in the WAVESS RCT suggests that GES *per se* might not directly relieve symptoms, and that other factors during GES treatment may play a role. The safety profile of Enterra® appears to be favourable,



although device infection was present in many of the case series patients, and instances of bowel obstruction and volvulus may call for more adequate securing of the device.

Ethical Issues:

No issues were identified from the retrieved material.

Cultural or Religious Considerations:

No issues were identified from the retrieved material.

Other Issues:

No issues were identified from the retrieved material.

HealthPACT Recommendation:

Further randomised controlled trials and comparative studies are required to establish the mechanism of symptom relief in the treatment of gastroparesis with GES. Due to the amount of evidence available for this procedure, it is recommended that the following be conducted:

- | | |
|---|--|
| <input checked="" type="checkbox"/> Horizon Scanning Report | <input type="checkbox"/> Full Health Technology Assessment |
| <input type="checkbox"/> Monitor | <input type="checkbox"/> Archive |

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Search Criteria:

A search of MEDLINE, PubMed and Cochrane Library, Current Controlled Trials metaRegister, UK National Research Register, International Network for Agencies for Health Technology Assessments, relevant online journals and the Internet was conducted in March 2005.

Search terms used were:

‘enterra’; ‘gastric electrical stimulation’; ‘gastroparesis’; ‘gastric pacing’; ‘gastric pacemaker’; ‘gastroparesis treatment’.

This Horizon Scanning Prioritising Summary was prepared by Ms Pauline McLoughlin from the NET-S Project, ASERNIP-S for the Health Policy Advisory Committee on Technology (Health PACT), on behalf of the Medical Services Advisory Committee (MSAC) and the Australian Health Ministers’ Advisory Council (AHMAC).