

# Gastric Pacing for Severe Gastroparesis during Pregnancy

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## Abstract

**Background:** Gastric Electrostimulation (GES) is a relatively new option for any kind of delayed gastric emptying not responding to conservative treatment. However, it is still unclear whether it might be used in pregnancy. Since no data are available, the provider interdicts the activation of the stimulating device to be on the safe side. On the other hand, clinical reasons may favor to continue GES to avoid recurrence of delayed gastric emptying.

**Methods:** We report one case of a young woman suffering from postsurgical gastroparesis who got pregnant under GES and in whom the treatment was continued.

**Results:** Pregnancy proceeded without any complications directly attributable to the pacemaker. The baby boy was born by emergency c-section due to fetal bradycardia in an IUGR situation.

**Conclusions:** Though it is recommended not to apply GES during pregnancy, it might be nonetheless justified to continue the treatment after carefully weighing up the risk/benefit ratio.

**Keywords:** Delayed gastric emptying; Gastroparesis; Gastric pacemaker; Pregnancy

## Case Report

A 31-year-old female patient (170 cm, originally 91 kg in March 2015) suffered from severe gastrointestinal reflux disease which could not be managed even under a double dose of 40 mg Pantoprazole. In February 2015, a laparoscopic fundoplication was performed in an external hospital. Before the operation, a gastroscopy had been performed which showed an axial hiatal hernia and a pronounced reflux esophagitis (Los Angeles D). Additional diagnostic work up was not performed.

Immediately after the operation, symptoms of reflux disappeared, but the patient complained of increasing dysphagia for solid foods. She also had epigastric pain and nausea together with constipation and a strong meteorism. Symptoms worsened after food intake. Nonetheless, she was discharged on the fourth postoperative day. In March 2015, the patient returned to the hospital due to increasing complaints as described above. On gastroscopy, no reasons to explain her problems were found. Treatment with prokinetics (metoclopramide) was initiated.

In July 2015 she already had lost 20 kg of weight and had not noticed any improvement of her symptoms under the therapy with metoclopramide. Once again, a gastroscopy was performed. The suspicion of delayed gastric emptying was expressed because of residual food, without therapeutic consequences. A further control gastroscopy in January 2016 did not reveal any pathological findings. The general state of the patient considerably further deteriorated in the following months.

Since delayed gastric emptying was finally considered as the cause of her problems, a bougienage of the pylorus followed, as well as a therapy with domperidone, metoclopramide, dimenhydrinate.

With this new treatment being also unsuccessful, the patient came up to our surgical outpatient clinic. Up to then, she already had suffered a total weight loss of 45 kilograms.

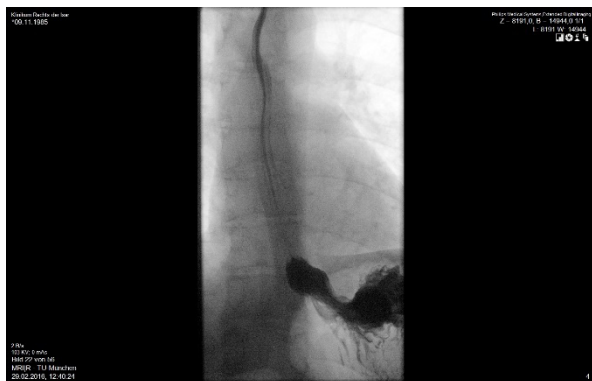
## In-house Initial Diagnostic Work Up

Upper GI endoscopy did not reveal any irregularities at the distal esophagus or the esophagogastric junction, but a certain amount of residual fluid in the stomach (Figure 1). The pylorus was inconspicuous and could be passed under light pressure. The duodenum was without pathological findings.

In the barium swallow, the findings were compatible with a normal state after fundoplication (Figure 2).



**Figure 1:** Gastroscopy: Residual fluid in the stomach, suspicious for delayed gastric emptying.



**Figure 2:** Contrast swallow: Pendulum movement with reduced clearance as well as reflux of contrast medium.

In addition, esophageal manometry was carried through. The Lower Esophageal Sphincter (LES) was located at about 39 cm from teeth. Swallow-induced relaxation was always complete and coordinated with a LES resting pressure of 16 mmHg. In the tubular esophagus, motility was unaltered. 24 h pH monitoring demonstrated almost complete reflux suppression with a DeMeester Score of 13,8.

MR Sellink (dynamic MRI) was without pathological findings.

## Gastric-emptying-scintigraphy

Visual delayed gastric emptying with delayed transfer of the test meal into the small intestine (Figure 3). The following percentage of residual activity in the stomach is found:

After 40 min.: 85% (standard: 21 to 64%); After 120 min.: 51% (standard: 0 to 25%).



**Figure 3:** Gastric-emptying-scintigraphy: Delayed gastric emptying.

## Therapeutic Approach

Initially, an experimental therapy with erythromycin was performed and the patient was asked to return to our outpatient clinic in April 2016.

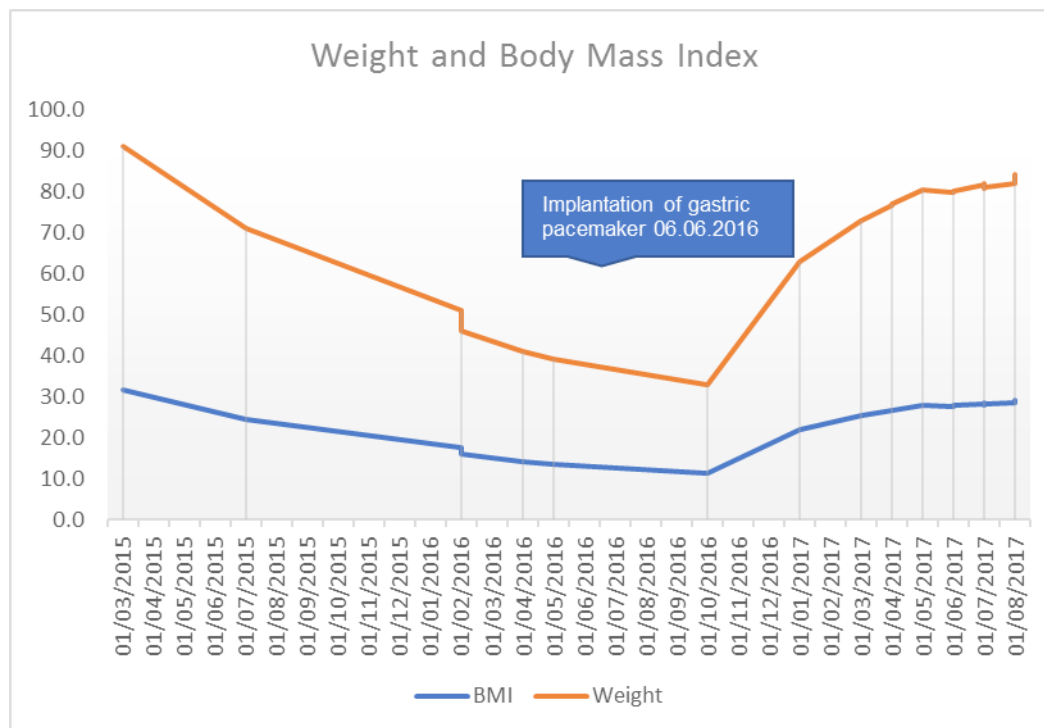
Until her reappearance, no improvement could be achieved. After the food intake, a strong feeling of fullness occurred, and fluid intake was also severely restricted. Accordingly, a final therapeutic trial with prucalopride was started, again without any positive result. The patient lost another two kilograms of weight, resulting in a total weight-loss of 52 kg within 2 years and 4 months. Since conservative treatment had failed, the decision was made to implant a gastric pacemaker laparoscopically, which was finally implanted in June, 2017.

The GES system was an Enterratherapy device (Medtronic, Minneapolis, MN) which was activated according to the algorithm of Abidi et al. [1]: 5 mA, stimulation-rhythm 1 second on-time, 4 seconds off-time, frequency 14 Hz.

The patient could be discharged three days after the operation and returned for postoperative follow up in June and August 2016. Her clinical symptoms had clearly improved but there was still a moderate weight loss. In October 2016, she was admitted again to our hospital due to pain in the upper abdomen in the area of the lower third of the sternum, especially after meals. In total, she had now lost 58 kg since February 2015. However, since implantation of the gastric pacemaker, there was no longer any nausea. In order to identify the cause of pain, a CT-scan, gastroscopy and manometry were performed. Neither of these examinations could explain the underlying disorder.

In close consultation with the company, as well as the supervising surgeons and gastroenterologists, the decision was made to de-activate the pacemaker temporarily.

Pain did not improve, but symptoms of delayed gastric emptying recurred. Accordingly, GES was started again with unchanged settings. The condition of the patient improved steadily under symptomatic anaesthetic therapy. Gradually, all symptoms resolved, and in-hospital care could be finished at the end of October 2016. From this point onward, there was a steady increase in weight (Figure 4).



**Figure 4:** Body weight and BMI curves: Increase of weight after implantation of gastric pacemaker.

In January 2017 the patient came again to our outpatient department to inform us about her being pregnant in the 7<sup>th</sup> week of pregnancy (gestational age 6 + 4). Her weight had amazingly increased up to now 63 kilograms, and she was now in a much better general condition. The first sonography showed an intact early pregnancy.

The question now was to decide upon whether the gastric pacing could be continued or not. According to the instructions of the provider, inactivation of the pacemaker during pregnancy is recommended, since very little is known about a potential interference. On the other hand, a recurrence of delayed gastric emptying had to be expected.

We decided to leave the gastric pacemaker switched on and closely observe the course of pregnancy. The patient presented again as an outpatient another three times. In April 2017 she came to a control sonography in the gestational age of 19+2. Organ screening of the fetus was normal. The patients right uterine artery showed high Pulsatility Index (PI) and notching, while mean PI of both arteries was within normal range. The patient's weight was now at 77 kilograms.

Next time she presented in gynecological outpatient in May 2017 in the gestational age of 23+2. There were no problems with the gastric pacemaker and her weight was at 80 kilograms. The development of the fetus was still unobtrusive, fetal growth along 10.th Percentile (Hadlock).

Close monitoring of the fetus revealed an intrauterine growth restricted situation, mainly due to bad uterine pulsatility indices: In 33+3 weeks of gestation the baby's growth was slightly below 3d percentile while fetal doppler values were within normal range. Mean PI of both uterine arteries was > 95<sup>th</sup> percentile. Cardiotocography was normal. The patient had developed an insulin dependent gestational diabetes which was well controlled. Laboratory results for intrauterine growth restricted fetus' reasons (toxoplasmosis, Parvo B19, cytomegaly) were uneventful. One week later (34+3 weeks of gestation) the fetus was checked again with normal Doppler- and CTG results. In 35+5 weeks of gestation the fetal growth was checked again with very limited growth of the abdominal circumference. Estimated fetal weight was below 3d percentile (1994g according to Hadlock formula), fetal Doppler and CTG without pathology. C-section was planned for 37+0 weeks of gestation, close follow up agreed. Body weight was

now at 84 kilograms and she was in a subjectively good general condition. In 36+2 weeks of gestation the patient presented for a CTG check: from the beginning of its recording the fetus showed persistent bradycardia over 7 minutes with no effect by immediate tocolytic therapy. An emergency c-section was performed. A baby boy of 2075g was born, APGAR 9-9-9, umbilical artery cord pH 7.28 and Base excess -4.5. Mother and baby were discharged 5 days later - both in good condition. The patient never showed signs of pregnancy induced hypertensive disorders.

## Discussion

Gastric pace making, firstly described at the end of the 20s [2], is an effective treatment option of gastroparesis. It can be used for non-responsive diabetic gastroparesis as well as idiopathic or secondary gastroparesis [3]. It may close the gap between medical treatment and irreversible stomach modifying surgeries. Gastroparesis affects women more commonly than men [4]. Idiopathic gastroparesis in particular often affects women of middle and young age [5].

In our case, gastroparesis occurred immediately after a laparoscopic fundoplication. It is rather likely that it was caused by a lesion of the vagal innervation of the stomach. Since abnormal gastric retention was refractory to standard medical treatment, we decided to implant the pacing system. Whereas vomiting and nausea were soon eliminated, a moderate weight loss persisted. After an episode of unexplained upper abdominal pain, a significant increase of bodyweight could be observed, and complete wellbeing was achieved.

Originally, a follow-up gastric emptying study was planned to verify improved gastrointestinal motility, but when the patient presented again, she had become pregnant which precluded this type of examination. This is why we were unable to assess objectively the improvement of gastroparesis.

Nonetheless, the gravida felt completely free of any former symptoms. In principle, we would have been obliged to inactivate the pacemaker according to the recommendation of the provider. Data about a potential interaction between gastric pacing and pregnancy are extremely scarce. To the best of our knowledge, only one published paper and one poster are available describing this condition [6,7].

Fuglsang et al. reported a case of a 28-year-old female patient with diabetic gastroparesis and an implanted gastric pacemaker became pregnant. Earlier days diabetic gastroparesis was considered as contraindication for pregnancy [8,9]. The authors could show that a pregnancy with gastric neurostimulation is possible. Patil et al. described a case of a 38-year-old female patient with type 1 diabetes and a gastric pacemaker for treatment of severe gastroparesis. The pacemaker stopped functioning at a

gestational age of 26 weeks due to an electrical shock during ironing. The consequence was recurrence of symptoms with excessive vomiting, exsiccosis and uterine contractions every 2-3 minutes. The symptoms were treated symptomatically and by reactivating the pacemaker. Finally, a live male infant was delivered in a good condition. This case impressively underlines the importance of proper functioning of the pacemaker. The patient must therefore be supervised continuously and in close cooperation with experts from the provider in order to prevent any unforeseeable events that could endanger the pregnancy or the well-being of the child.

Most papers dealing with gastric pacing in general do not consider pregnant patients as candidates for gastric stimulation [3] because a detrimental influence upon gestation cannot be ruled out completely. On the other hand, it is possible - if not even probable in our case - that persisting or recurring symptom of vomiting and nausea including weight loss may have a negative influence on pregnancy.

After an intensive discussion with electrical engineers, the risk of any electrical influence of a bipolar local stimulation of the stomach onto the embryo who is located at a comparatively long distance was considered as relatively low. Furthermore, it had to be taken in account that a recurrence of the severe disorder would not only massively reduce the quality of life of the patient but also could endanger the development of the fetus. All of these aspects were intensively discussed with the gravida including both the surgical as well as the gynecological / obstetrician point of view. In a consensus with the medical experts, the patient finally decided to continue stimulation.

Under continuous stimulation, pregnancy was relatively uneventful. Symptoms, like heartburn, vomiting or nausea did not occur any more. Gestational gain was normal. Obstetrical checkup was frequently performed. The patient did develop an insulin dependent gestational diabetes. Fetal growth restriction was diagnosed in 33. Weeks of gestation, mainly due to placental insufficiency caused by impaired placentation, slightly premature emergency c-section was required in 36+2 weeks of gestation due to fetal bradycardia in an intrauterine growth restricted fetus. We do not believe this condition was caused by gastric neurostimulation. To a greater extend it was the cause of a growth restricted fetus with placental insufficiency maybe due to malnutrition early in pregnancy. We conclude that patients with such gastric disorders and gastric neurostimulation might be closely followed through pregnancy by gastroenterologists and obstetricians.

This case confirms, as already been observed by Fuglsang and Patil [6,7], that a functioning gastric neurostimulation does not preclude normal intrauterine fetal growth. Of course, it is still too early to claim that it is completely free of any risk, but our observation may contribute to a better decision making in the

management of gastric paresis in pregnancy.

## Conflict of Interest

The authors have no conflict of interest or financial ties to disclose.

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