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**Diabetic Gastroparesis**

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**Introduction**

According to the 2014 Centers for Disease Control (CDC) 9.3% of the United States Population have diabetes (National diabetes statistics report, 2014, p.1). One of the complications of diabetes is gastroparesis. Gastroparesis “is a delay in the emptying of ingested food in the stomach due to abnormal function of the stomach or duodenum” *(Koch & Callen-Connell, 2015)*. Patients with either type 1 diabetes (T1DM) or type 2 diabetes (T2DM) may experience the clinical symptoms of early satiety, prolonged fullness, nausea, and vomiting, and have difficulties with management of blood glucose levels, nutritional issues, and other drug absorption issues (Koch & Callen-Connell, 2015). Health care providers may see patients in a variety of settings, with and without known diagnosis of gastroparesis and must be aware of the symptoms, the potential diagnosis, the appropriate testing, and the options for treatment. Advances in the treatment of gastroparesis have resulted in the increased understanding of the pathophysiology behind gastroparesis.

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**Signs & symptoms**

- Nausea
- Early satiety
- Abdominal pain
- Hyperglycemic control
- Malnutrition
- Diarrhea
- Drug interactions
- Poor quality of life
- Frequent hospitalizations

Shin & Callen-Connell, 2015

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**Pathophysiology**

The normal activity in gastric motility involves three areas of action.

- **The fundus**, which relays with a volume of food ingested and requires an intact vagus nerve and enteric neurons (Koch & Callen-Connell, 2015).
- **The corpus and antrum**, which produce recurrent waves at a frequency of 3 cycles per minute, turning digested food solids into finer material called chyme. These peristaltic waves are controlled by gastric pacemaker cells called interstitial cells of Cajal (ICCs) (Koch & Callen-Connell, 2015).
- **The emptying of chyme through the pylorus into the duodenum**. This pyloric regulative volume and particle size of chyme is affected by cathecolamines and peptide releases (Koch & Callen-Connell, 2015).

- Understanding the various potential alterations that lead to gastroparesis can affect the treatment or management of symptoms and mode of testing.
- The gastrointestinal motility of the healthy patient is the gastric emptying study (GES), which assesses between abnormal results and patient symptoms due to various pathophysiology mechanisms (Shin & Callen-Connell, 2015).
- Alternative methods of testing exist (Shin & Camilleri, 2013). Assessment of symptoms and other factors such as age, sex, comorbidity, patient preference, and test availability should contribute to the decision of mode of testing (Shin & Callen-Connell, 2013).
- Treatment options are palliative and empiric in focus and not curative (Paricio, 2015).
- Common treatments aimed at increasing gastric emptying (Pasricha, 2015).
- Nutritional support, fluid and electrolyte restoration, pyelaryc connection (Acosta & Camilleri, 2015).
- Six or more meals or eats a day are needed to meet nutritional needs and include restricted fat and fiber intake. Tolerance is improved with liquids or ground foods (Shin & Callen-Connell, 2015).
- Prokinetic medication help gastric emptying by “restoring the synchronicity between delivery of food and hormone and peptide release, but it does not target the underlying problem” *(Farrugia, 2015)*.
- Metoclopramide, a dopamine 2 receptor agonist, is the only approved prokinetic medication in the United States; use is limited to less than 13 week treatment (Acosta & Camilleri, 2015).
- Domperidone is another dopamine 2 agonist, but use is considered investigational (Acosta & Camilleri, 2015).
- Antiemetic medications are often utilized for control of nausea, but one study suggests that antiemetics show modest benefit in comparison to non- use (Koel, 2021).
- Opiate analgesics are often used but use could exacerbate nausea and vomiting and also slow gastric emptying (Shin & Camilleri, 2015).
- Hypoglycemic treatment with insulin, may improve and normalize ICCs (Koch & Callen-Connell, 2015).
- Gastric pacemakers provide gastric electrical stimulation aimed at controlling nausea and improving vagaal function when other therapies failed, more understanding of gastric mapping is needed (Sarosi et al., 2015).
- A jejunal pouch may be needed for patients experiencing gastroparesis in order to control symptoms (Shin, 2015).
- Neuer agents are being studied and developed by targeting different pathways and include serotonin receptor agonists, ghrelin agonists (Acosta & Camilleri, 2015).
- Gastric pacemakers provide gastric electrical stimulation aimed at controlling nausea and improving vagaal function when other therapies failed, more understanding of gastric mapping is needed (Sarosi et al., 2015).

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**Significance of Pathophysiology on Gastroparesis**

There are several alterations suggested in gastroparesis.

- The most common abnormalities is loss of ICCs. ICCs regulate contractility and impaired ICCs results in gastric dysrhythmias (Farrugia, 2015).
- Damage to the vagus nerve innervation to the stomach, may lead to motor abnormalities and abnormal relaxation of the pylorus *(Koch & Callen-Connell, 2015)*.
- Evidence has shown smooth muscle degeneration and end stage evidence may include fibrosis with exophotic inclusion bodies *(Farrugia, 2015)*.
- Loss of neurotransmitters, such as neuronal nitric oxide synthase (nNOS) have been seen and are thought to be reversible (Farrugia, 2015).
- Nitric reuser less and ECA less is thought to account of the poor fundus relaxation and decreased gastric capacity that is seen in gastroparesis (Koch & Callen-Connell, 2015).
- Fundal-like cells (FLC), similar to ICCs, involves in neurotransmission, and may be decreased in gastroparesis (Farrugia, 2015).
- Alterations in the balance of pro-inflammatory MT1 macrophages and anti-inflammatory M2 macrophages of the stomach wall muscle are suspected to be becoming disrupted in gastroparesis (Farrugia, 2015). MT macrophages are associated with the development of delayed gastric emptying (Farrugia, 2015).

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**Neuropraxic function in gastroparesis**

(Koch & Callen-Connell, 2015)

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**Implications for Nursing Care**

Having an understanding of the diagnosis of gastroparesis, symptom recognition, and understanding of the behavior, it is important for nurses in a variety of roles. Diabetic patients are often affected with gastroparesis. Gastroparesis affects glyceremic control and lack of glyceremic control can lead to gastroparesis (Koch & Callen-Connell, 2015).

Nurses can have a pivotal role in diabetic eating affecting glyceremic control. Dietary education in tandem with dietary professionals, can also provide symptomatic relief from those suffering from gastroparesis. Knowledge of gastroparesis and medication therapy can also benefit patients through nurse request for appropriate medication, and patient education of those drugs. Advance practice nurse knowledge of gastroparesis, can lead to improvement in patient assessments, testing, symptom management, awareness of potential electrolyte imbalance, and patient nutrition and diabetes management. Since the symptoms of gastroparesis lead to frequent medical visits and hospitalization, sensitivity to patient quality of life and screening for symptoms of depression may be important.

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**Conclusion**

Diabetic patients often experience gastroparesis. A cure for gastroparesis is not present. Research and better understanding of the pathophysiology of gastric motility and function is needed to further advance the treatment of gastroparesis and its symptoms. Until then, knowledge of the prevalence of gastroparesis in diabetics will provide a pivotal role in improving outcomes and the appropriate choice of available medical therapies is important. Support of the patient in nutrition and glyceremic management is needed, as well as support of the patient’s emotional well being.

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**References**


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**Additional Sources**
