MINI REVIEW

Gastric sensorimotor function and its clinical measurement

Greg O'Grady, Florencia Carbone, Jan Tack

INTRODUCTION

The stomach is a hollow organ comprising the cardia, fundus, corpus, antrum, and pylorus. Functionally, the stomach consists of proximal and distal parts, with a sphincter at both ends. Smooth muscle cells in the proximal stomach (fundus and upper corpus) do not display electrical oscillations and this part is characterized by a tonic contractile activity. Smooth muscle cells in the distal stomach (mid-corpus and antrum) demonstrate rhythmic depolarizations, called slow waves, which regulate the pattern of gastric contractions. The coordinated behaviors of these regions are essential to generating the normal gastric reservoir, trituration, and transit functions, and deviations may contribute to symptoms. In the current issue of Neurogastroenterology & Motility, Silver et al. present new data showing correlations between patterns of intragastric meal distribution and symptom profiles in a large series of patients undergoing gastric emptying scintigraphy. Studies of this type are important, as they motivate understanding beyond existing disease labels, and orient focus toward deeper mechanistic profiling. This brief review provides an overview of gastric sensorimotor function and profiles several current and emerging methods of clinical evaluation. Perspectives are provided on accommodation testing, gastric emptying, measuring gastric myoelectrical activity including new approaches, and antroduodenal manometry. Although gastric physiology is complex, recent progress has been encouraging, with the heterogenous pathophysiology of gastric symptoms continuing to be unraveled, and new techniques for evaluating gastric function and symptoms emerging. Ongoing progress will now depend on continuing to accurately profile the underlying mechanisms of gastroduodenal disorders to identify specific disease phenotypes that inform care.
correlations between patterns of intragastric meal distribution and symptoms during scintigraphy. This review provides an overview of gastric sensorimotor function, current and emerging methods for clinical evaluation, and future directions.

2 | GASTRIC MOTOR AND SENSORY FUNCTION

Gastric sensorimotor function shows major differences during fasting and after a meal. Fasting gastric motor activity participates in the migrating motor complex (MMC), while postprandial motor activity combines gastric reservoir, mixing and emptying.

2.1 | Interdigestive motility

The MMC is a recurring motility pattern in the stomach and small bowel in the fasting state. The average MMC cycle lasts 90–120 min and is composed of 3 phases. Motor quiescence characterizes phase I, while phase II is characterized by irregular contractions. Phase III, the most characteristic part, consists of a brief episode of strong contractions at maximal frequency (3/min in the antrum, 12/min in the most characteristic part, consists of a brief episode of strong contractions at maximal frequency (3/min in the antrum, 12/min in the duodenum) which is followed by phase I. Phase III originates from the stomach or the proximal small intestine and migrates distally at 1–4 cm/min. The MMC has a housekeeper function, as phase III clears indigestible remnants and bacteria to the colon. Gastric phase III, triggered by release of the peptide hormone motilin, also serves as a hunger signal, as it is associated with peak hunger and food intake. Ingestion of food suppresses the MMC and switches upper gastrointestinal motility to a postprandial pattern.

2.2 | Gastric reservoir function

During fasting, the proximal stomach maintains a tonic smooth muscle contraction, which relaxes during and after meal ingestion to provide the meal with a reservoir, without a rise in gastric pressure. This vagally-mediated gastric relaxatory response consists of an initial phase immediately after deglutition, called receptive relaxation, and a second longer-lasting phase, called adaptive relaxation or gastric accommodation. Accommodation is mediated through release of nitric oxide from intrinsic nerves expressing 5-hydroxytryptamine-1 (5-HT1)-like receptors. The antrum is also involved in the gastric accommodation reflex.

2.3 | Gastric emptying

Postprandially, peristaltic waves arise from the mid-corpus region and propagate toward the pylorus. These grind food into smaller particles through retropulsion in combination with gastric secretions. Increasing tone of the proximal stomach moves gastric contents distally and contributes to the gastroduodenal pressure gradient. Chyme flow to the duodenum is pulsatile, driven by antral contractions in concert with pyloric relaxation, with influence from duodenal resistance. High-resolution manometry has also recently identified a zone of non-propagating, repetitive pressure events at the duodeno-jejunal transition, referred to as a “duodenal brake”. Neural and hormonal negative feedback controls the rate of gastric emptying based on luminal content sensing. Duodenal nutrient exposure activates vago-vagal feedback and reflex inhibitory control of gastric tone and contractility, pyloric closure, and release of hormonal signals (GLP-1, PYY, CCK), which further delay the arrival of acidic, hyperosmotic, or calorie-rich gastric contents.

2.4 | Gastric pacemaker function and slow waves

The frequency, coordination, and propagation of peristaltic contractions in the stomach are driven by slow waves. These are cyclical oscillations of the cell membrane potential, generated and propagated by interstitial cells of Cajal. The gap-junction coupled ICC function acts as a syncytium, coordinating the timing and pattern of contractions.

2.5 | Gastric sensitivity

The gastrointestinal tract conveys information to the brain through mechanosensitive and nutrient sensing pathways, which may also induce perception. The classical view on sensing of nutrients in the upper gastrointestinal tract states that gastric nutrient sensing is volumetric through activation of mechanoreceptors, while intestinal sensing is nutritive chemosensing. The available evidence suggests that gastric mechanoreceptors in man behave like tension-sensitive in-series mechanoreceptors; the molecular nature and site of expression are still being investigated.

2.6 | Gastric motility disorders

Epigastric symptoms thought to be caused by disorders of gastroduodenal sensorimotor function are highly prevalent. The symptom pattern may include early satiation, postprandial fullness, bloating, epigastric pain or burning, nausea, vomiting, and belching. There may be associated symptoms of loss of appetite, weight loss and postprandial fatigue or dizziness. Disorders of gastric sensorimotor function underlie conditions such as functional dyspepsia (FD), gastroparesis, nausea and vomiting disorders and dumping syndrome. The Rome IV consensus defines FD by the presence of gastroduodenal symptoms (early satiation, postprandial fullness, epigastric pain, and epigastric burning), in the absence of organic, systemic, or metabolic disease likely to explain symptoms. The global prevalence is estimated at approximately 7%. The Rome consensus distinguishes two FD subgroups, postprandial distress syndrome (PDS)
characterized by early satiation or postprandial fullness, and epigastric pain syndrome (EPS) with symptoms of epigastric pain and burning. The PDS group accounts for up to 85% of FD cases and overlaps with EPS in approximately 15%. FD is heterogeneous and subgroups of patients have impaired gastric accommodation, disordered gastric emptying, gastric myoelectrical abnormalities, or hypersensitivity to gastric distention. The correlation between symptom pattern and severity and disordered gastric function is generally weak. Analyses of large patient subgroups have shown associations of early satiation and weight loss with impaired accommodation, of postprandial fullness with delayed gastric emptying and of epigastric pain and weight loss with gastric hypersensitivity. On the contrary, no difference was found in the prevalence of these sensorimotor disorders between PDS and EPS.

According to the European consensus, gastroparesis is characterized by delayed gastric emptying in the absence of mechanical obstruction, with a symptom pattern of nausea and/or vomiting and PDS symptoms. Several disorders can be associated with gastroparesis, but the predominant types are idiopathic and diabetic. The estimated global prevalence is 0.9%, with a higher prevalence of 1.3% in diabetic subjects.

While nausea and vomiting are cardinal gastroparesis symptoms, most patients with these symptoms will not show delayed gastric emptying. They are categorized according to the Rome IV consensus as having chronic nausea and vomiting syndrome, cyclic vomiting syndrome or cannabinoid hyperemesis syndrome. The pathophysiology of the two former conditions is poorly understood, but abnormalities of gastric electrical rhythm have been implicated, as well as brain-gut dysregulation, and other factors.

Dumping syndrome consists of a constellation of cardiovascular and gastrointestinal symptoms that can be attributed to the rapid passage of nutrients into the small bowel, predominantly being a complication of gastric surgery.

Assessment of gastric motor function is generally pursued after exclusion of structural disease, using esophagogastroduodenoscopy (EGD), radiology, and laboratory testing. The most frequent tests of gastric motor function are measurement of gastric emptying, electrogastrography, manometry, and gastric accommodation testing.

3 | GASTRIC ACCOMMODATION

Gastric accommodation consists of a reduction of gastric tone and an increase in gastric compliance in response to food intake, allowing an increased fundus volume without concomitant rise in intragastric pressure (IGP). Impaired accommodation has been implicated in symptom generation in FD and in post-Nissen dyspepsia.

The gastric barostat consists of an intragastric balloon kept at a selected intragastric pressure by changing the air volume in the balloon. To measure gastric accommodation, the pressure level of the intragastric balloon is fixed and the difference in balloon volume before and after (usually 30 min and 1 h respectively) ingestion of a small mixed liquid meal quantifies gastric accommodation. Gastric accommodation measurements with barostat have shown symptom correlations in several studies and are susceptible to pharmacological interventions. The gastric barostat is considered the gold standard for quantifying accommodation, but the invasiveness and discomfort associated with the use of a large inflated gastric balloon has excluded it from clinical use.

Several imaging-based methods have also been proposed for the evaluation of gastric accommodation. These include ultrasound, single photon emission computed tomography imaging (SPECT) and magnetic resonance imaging. The main endpoint is the (proximal) gastric volume after meal ingestion. However, the applicability of these methods is hampered by the necessity for specialized equipment, high costs and/or radiation exposure. Moreover, the results of these volumetric methods are dominated by the volume of the ingested (liquid) meal, which is non-compressible and hence poorly reflects changes in wall tension driven by the degree of accommodation. These volumetric methods have not consistently been shown to correlate with symptom pattern or to be susceptible to pharmacological interventions.

A comparison of the distribution of the liquid meal volume over the proximal versus distal stomach seems to provide a more robust assessment of disordered accommodation, shown as a lower proximal to distal gastric volume. Using scintigraphy, distal intragastric distribution of the meal correlated with symptoms of early satiation and weight loss. These findings are confirmed in the paper of Silver et al., who associated early distal redistribution, probably reflecting impaired accommodation, with early satiation and late proximal retention with nausea, retching and vomiting.

A non-invasive method to assess gastric accommodation is the nutrient drinking challenge test. The subject drinks water or a nutrient-containing drink at a constant rate until maximum satiation is reached and the ingested volume is measured. Especially the slow nutrient drinking test shows a consistent difference between healthy controls and FD patients and correlates with barostat measurements of gastric accommodation. In pharmacological studies, the slow nutrient drink test showed concordance between effects on gastric accommodation and tolerated nutrient volume. However, when agents with central nervous system actions were studied, this correlation was lost.

The measurement of intragastric pressure (IGP) during a nutrient drinking test, or during intragastric nutrient drink infusion is a novel and less invasive technique to measure accommodation in humans. IGP is determined by gastric muscle tone and during intragastric nutrient infusion, the IGP decreases initially to gradually increase thereafter. The drop in IGP requires nitric oxide synthase activity and reflects the gastric accommodation reflex. Moreover, the IGP recovery from nadir (lowest IGP) is a determinant of satiation. In FD patients, the IGP drop is suppressed. Studies in healthy controls have shown sensitivity of the IGP measurement to pharmacological interventions.

In theory, measuring impaired accommodation may explain symptoms of early satiation and unexplained weight loss and may prompt the use of fundus-relaxing therapies such as 5-HT1A agonists or...
selected prokinetics, such as 5-HT4 receptor agonists.\textsuperscript{25–27} However, none of the methods outlined in this section have seen implementation in practice, and fundus-relaxing therapies are empirically applied.\textsuperscript{8}

4 | GASTRIC EMPTYING

Gastric emptying testing has been the most widely applied test of gastric function and remains the defining standard for gastroparesis.\textsuperscript{9} However, the limitations of this test have become increasingly clear, such that its role in diagnosis and management is controversial.

A key concern has been weak and inconsistent associations between symptoms and delayed emptying. However, a meta-analysis of over 6000 patients showed that when GET is performed optimally, including both scintigraphy and breath test methodologies, delayed emptying does statistically correlate with symptoms including nausea, vomiting, pain, early satiety, and excessive fullness.\textsuperscript{21} In addition, a second meta-analysis of optimal GET data showed that promotility agents can result in clinically meaningful improvements in symptoms when emptying is delayed.\textsuperscript{22} The best available evidence therefore justifies the ongoing relevance of gastric emptying testing.

However, difficulties are encountered when attempting to define disease based on the presence or absence of delayed gastric emptying, at either a general or individual patient level. A substantial proportion of patients appear to show identical symptoms and epidemiology to gastroparesis yet have normal emptying,\textsuperscript{23} whereas a quarter of patients labeled as FD show delayed emptying.\textsuperscript{24} In addition, a prominent study recently revealed that gastric emptying status is highly labile, with approximately 40% of patients being reclassified between FD and gastroparesis over a 48-week period.\textsuperscript{24} Symptom severity did not change with emptying status, while cellular damage indicating underlying neuromuscular disease overlapped between gastroparesis and FD cohorts. These data led the authors to conclude that "gastric emptying measurements do not capture the pathophysiology adequately."

These data provide clarity regarding the role of gastric emptying testing. The test must be accepted as lacking sensitivity for detecting gastric neuromuscular dysfunction, and a normal emptying test is therefore of dubious utility to establish a solid diagnosis with associated treatment implication and predictable outcome. However, a positive finding of delayed gastric emptying may still be useful, as this contributes to an impression of "disordered neuromuscular control," can inform selection for prokinetic or pyloric-based therapies, and guides diabetes care.\textsuperscript{25–27} Moreover, "gastroparesis" itself would appear to only represent a secondary disease mechanism, capable of modifying disease expression and exacerbating symptoms, but inconsistently so, and not as a primary disease mechanism. This is also logical, because gastric emptying itself is a higher-order operation, integrative of several other functions, and modulated by many variables including metabolic, circadian, positional, hormonal, pharmacological, psychological, and distal gastrointestinal factors.\textsuperscript{28}

The paper by Silver et al. provides a further layer of interest, demonstrating that differences in intragastric meal distribution during scintigraphy are also relevant to symptom expression.\textsuperscript{2} The findings are in line with associations between symptoms and distribution of a radioscintigraphy meal as reported by Piessevaux et al. in 2003.\textsuperscript{17} However, it is also noted that aberrant meal distribution remains a higher-order dysfunction, potentially modifying symptom expression, but requiring additional work to understand causality and therapeutic implications.

5 | GASTRIC MYOELECTRICAL ACTIVITY

Electrogastrography (EGG) is now in its centennial year, and a substantial literature has accrued showing that gastric myoelectrical abnormalities are prevalent in patients with chronic gastric symptoms.\textsuperscript{29,30} However, EGG did not translate well beyond research settings, owing to several technical limitations, and is now uncommonly used in practice.

Recently, a new generation of high-resolution techniques have emerged for evaluating gastric myoelectrical activity, termed "HR-EGG" or "body surface gastric mapping".\textsuperscript{31} This field is evolving rapidly, driven by new technologies such as wearables, stretchable electronics, digital symptom tracking, and cloud-computing.\textsuperscript{32,33} These advances, coupled with optimized data processing, offer new or improved metrics of gastric function, opening a new era in gastric electrophysiology.\textsuperscript{32} A first FDA-cleared gastric mapping device has also become available for clinical use, employing a high-density array of 64 electrodes (Gastric Alimetry®).\textsuperscript{14}

EGG was able to detect group-level differences in gastric function, but a diagnostic test must also be reliable and discriminating at the level of individual patient care. Therefore, the first test for body surface gastric mapping has been to determine whether specific disease phenotypes can be identified among patients with overlapping symptom profiles. Promising data has recently been reported in this regard, indicating the capability of gastric mapping to separate nausea and vomiting patients into subgroups with neuromuscular abnormalities vs normal electrophysiology, the latter being more likely to experience disorders of gut-brain interaction.\textsuperscript{14} In addition, three studies have now indicated symptom correlations based on gastric mapping phenotypes in nausea and vomiting disorders, FD, and diabetes.\textsuperscript{24,34,35}

An attraction of body surface gastric mapping is that the emerging disease phenotypes, such as neuromuscular dysfunction, appear to represent more direct mechanisms than higher-order variables such as delayed transit. However, body surface gastric mapping has only recently emerged, and its role in diagnostic and therapeutic pathways will become clearer with further data and development.

6 | GASTRIC MANOMETRY

Antroduodenal manometry allows to visualize and quantify the contractions underlying gastric emptying and interdigestive motility processes.\textsuperscript{36} Water-perfused or solid-state pressure transducers
mounted on a catheter can be used, at low or high resolutions. A sleeve straddling the pylorus, with confirmation of position based on agar bridge electrodes, can be used to monitor pyloric resistance.

The key features that are observed during antroduodenal manometry are the number and amplitude of contractions, and their pattern in the interdigestive and postprandial states. In the postprandial state, antral hypomotility as shown by decreased antral contraction frequency and amplitude is a non-specific finding. In the interdigestive state, absence or aberrant configuration and migration of intestinal phase 3 indicates a major motor disorder, such as observed in chronic intestinal pseudo-obstruction, severe intestinal neuropathy, or myopathy. Loss of gastric phase 3 has also been associated with loss of appetite and body weight.37 Especially for the interdigestive state, identifying more specific patient phenotypes, and then linking these with reliable outcomes. Existing disease classifications are becoming increasingly controversial in this regard, and with most pharmacological and interventional trials still typically yielding mixed or disappointing outcomes.

Antroduodenal manometry is susceptible to pharmacological interventions.37 The technique is only used in few centers, and mainly serves to exclude severe generalized intestinal motility disorders. Nevertheless, results of manometry will determine treatment choices in a subset of patients.38

7 | CONCLUSIONS AND THE WAY FORWARD

Extensive research has shown that gastric motor abnormalities are prevalent and contribute to symptoms, yet their clinical evaluation remains challenging. Traditional tests such as gastric emptying, electrogastrography, accommodation, and antroduodenal manometry remain limited in their utility or availability, and trial-and-error therapy is still routine.

Overcoming these challenges continues to depend on identifying more specific patient phenotypes, and then linking these phenotypes to therapies with reliable outcomes. Existing disease classifications are becoming increasingly controversial in this regard, owing to their overlaps, and with most pharmacological and interventional trials still typically yielding mixed or disappointing outcomes. The current work by Silver et al.2 is therefore commendable, as studies of this type remind the field to look beyond existing labels toward deeper mechanistic profiling.

Other recent progress has also been encouraging. The varied pathophysiological basis of gastric symptoms continues to be unraveled, and new techniques for evaluating gastric function and symptoms have continued to emerge. A useful next step will be to apply such technologies in multimodal studies, together with existing tests of gastric function and preferably in large patient cohorts, to continue the path toward more accurately profiling gastroduodenal disorders to guide targeted care.

AUTHOR CONTRIBUTIONS

All authors provided content, wrote sections, reviewed final manuscript versions.

DISCLOSURE

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