Acid pocket, hiatus hernia and acid reflux
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Acid reflux and its associated symptoms occur most frequently following the ingestion of a meal. This observation presented a dilemma as intragastric pH is at its least acidic following eating due to the buffering effect of the food. However, the observation by Fletcher et al that the proximal cardia region of the stomach escapes the buffering effect of the meal provided a rational explanation for the acidic nature of the postprandial refluxate. The zone of high acidity detected in the proximal stomach after a meal has been termed the acid pocket.

The presence of the acid pocket has been confirmed by a substantial number of investigators and by a variety of different investigative techniques. The latter have included pH pull-through, multiple static pH electrodes and single photon emission CT (SPECT) of the stomach following intravenous injection of [99mTc] pertechnetate which is secreted by the acid-secreting cells of the gastric mucosa.

It had been suggested that the acid pocket might represent only a film of acid lining the proximal stomach without any significant volume. In the current issue of Gut (see page 441) Beaumont et al placed a catheter at the anatomical site of the acid pocket and aspirated 50–70 ml of gastric juice pH 1.7–2.5 during the postprandial period. They were also able to show that the SPECT imaging of the acid pocket disappeared with aspiration and then reappeared. A recent study employing 12 pH electrodes situated 1 cm apart has also shown highly acidic oesophageal reflux at a time when the only region of the stomach with equally acidic contents is the acid pocket. These studies, therefore, confirm that the acid pocket contains a volume of acidic juice adequate to acidify the more proximal oesophagus during reflux events.

The mechanism by which the proximal stomach escapes the buffering effect of the meal remains unclear. Acid is secreted by the mucosal lining of the stomach and, following a meal, the periphery of the gastric lumen will be most acidic due to its proximity to the acid source, whereas the centre will be least acidic due to the buffering effect of the food. Ingestion of a meal stimulates acid secretion and the presence of the food and the secretions distend the stomach, reducing the density of the gastric folds per surface area. However, the gastric folds in the proximal cardia region escape the distension due to the restricting effect of the diaphragmatic hiatus and lower oesophageal sphincter. There is, therefore, preservation of a high mucosal surface to luminal volume in the proximal stomach following a meal and thus a highly acidic luminal pH.

The primary physiological function and evolutionary benefit of gastric acid is to kill ingested potentially pathogenic microbes. The proximal acid pocket will thus serve to maintain the acid barrier when eating and thus at a time when ingestion of environmental pathogens is most likely.

There is interest in the potential role of the acid pocket in the pathophysiology of gastro-oesophageal reflux disease (GORD). There is a high incidence of inflammation of the gastric cardia and of intestinal metaplasia at the gastro-oesophageal junction. Recent studies have demonstrated that the acid pocket extends very close to, or even across, the squamocolumnar junction following meals, probably due to opening of the distal segment of the lower oesophageal sphincter. It has been proposed that this short segment reflux may be responsible for the high incidence of inflammation and metaplasia at the gastro-oesophageal junction.

There has also been interest in the role of the acid pocket in more traditional reflux disease. Clarke et al studied the size and location of the acid pocket in 16 patients with severe reflux disease and 12 healthy controls. The patients had either severe oesophagitis (grade 3) or Barrett’s oesophagus, and all had evidence of hiatus hernia. The upper gastrointestinal anatomy was documented by barium meal and endoscopy and at the latter radio-opaque clips were attached to the gastro-oesophageal junction (proximal margin of the gastric folds) and diaphragmatic hiatus. Pull-through studies (1 cm/min) with a dual pH electrode and solid state manometer were performed fasted and 15 min following a meal along with fluoroscopy of the position of the clips relative to the catheters. The reflux patients had longer acid pockets than the healthy volunteers, extending more distally relative to the proximal gastric folds. The length of the acid pockets distal to the diaphragmatic hiatus was similar in the two groups. The longer acid pockets in the reflux patients were thus related to the proximal migration of the gastro-oesophageal junction. These findings suggested that the hiatus hernia was creating an enlarged, or at least lengthened, proximal acid pocket.

In their paper in this issue of Gut, Beaumont et al have further investigated the anatomy and physiology of the proximal stomach in reflux patients and healthy controls. They studied 22 reflux patients, 12 with large hiatus hernias (>3 cm) and 10 with small hiatus hernias (≤3 cm), and 10 healthy controls. The rates of transient lower oesophageal relaxations (TLESRs) were similar in the reflux patients and controls though the former had increased acid reflux. Using [99mTc] pertechnetate-radiolabelled endoscopic clips attached to the proximal margin of the gastric folds and to the diaphragmatic indentation along with SPECT imaging following intravenous [99mTc] pertechnetate to visualise the acid pocket they were able to study the size and location of the acid pocket in the different groups of subjects.

Using the above methods, they observed that the acid pockets were longer in the reflux patients. In addition, immediately prior to TLESRs, the acid pockets were more frequently located within the hiatus hernia or above the diaphragm in the reflux patients. They concluded that the enlarged acid pocket and its presence above the diaphragm is a major risk factor for acidic reflux during TLESRs. The enlarged proximal acid pockets in the reflux patients provided an explanation for them having greater acidic reflux than the healthy volunteers despite having a similar frequency of TLESRs. They also concluded that the larger and more proximal acid pockets in the patients with GORD was due to the presence of hiatus hernia.

Hiatus hernia is well known to contribute to the pathophysiology of reflux disease by its effects on gastro-oesophageal motility—impairing both lower oesophageal sphincter function and oesophageal clearance. The two recent papers indicate that a hiatus hernia may contribute to more...
further to acid reflux by changing the nature of the refluxate itself. The enlarged proximal acid pocket in subjects with hiatus hernia means that they have a larger reservoir of highly acidic juice available to reflux into the oesophagus whenever the lower oesophageal sphincter spontaneously relaxes or fails under pressure.

Competing interests None.

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Visceral sensory and cognitive-affective neuroscience: towards integration?

Lukas Van Oudenhove

Despite rapidly growing evidence supporting a link between psychological processes—both cognitive and affective—and visceral sensation in health as well as functional gastrointestinal disorders (FGIDs), the neural mechanisms underlying these interactions remain infrequently studied within the field of ‘neurogastroenterology’ and therefore rather poorly understood.1 The somatic pain field, in contrast, has already made considerable progress in unravelling these complex brain mechanisms by which emotion (eg, anxiety) and cognition (eg, attention) influence the processing and perception of bodily signals.2 More specifically, the amygdala, insula, and cingulate and prefrontal subregions have been shown to be involved in pain—emotion interactions. This knowledge is the result of a fruitful integration between different branches of science within the somatic pain field over the past decades, including psychology, psychiatry, anaesthesiology and the affective, cognitive and sensory branches of neuroscience.

Such a degree of integration has not been achieved yet within visceral sensory neuroscience3 but is, in my opinion, much needed if we really want to move the field forward. This may be especially critical if we want to make progress in the understanding of the multifactorial pathophysiology of complex, symptom-based disorders including FGIDs. Although rather sparse, recent attempts towards such integration have been made, and I will try to situate these against a long tradition of studying the interactions between mind, brain and body underlying visceral sensation.

In the present issue of Gut, Elsenbruch and colleagues (see page 489) report on a functional MRI study showing that, in irritable bowel syndrome (IBS), anxiety and depression scores correlate with subjective pain ratings during rectal distension.3 Furthermore, within the IBS group, anxiety scores correlate with pain-induced activation of the anterior midcingulate cortex (aMCC) and the pregenual anterior cingulate cortex (pACC); depression scores show a correlation with activity in the dorsomedial prefrontal cortex (PFC) and cerebellar areas. Finally, when pain-induced brain activity in patients with IBS was compared with that of healthy volunteers, (small) differences were found in the ventromedial PFC and the anterior insula (aINS). However, when anxiety and depression scores were controlled for in the analysis, these differences disappeared. I believe this article is important because it is one of the first to show the neural mechanisms underlying the interaction between anxiety and depression on the one hand and visceral sensation on the other in patients with IBS. However, it may be seen as the continuation of a long tradition of (neuro)scientific research on this type of interaction in healthy volunteers. Most of this work comes from a variety of distinct research traditions and may therefore be largely unknown to most people in the field of ‘neuro-gastroenterology’.

In the 1880s, James and Lange provided the first theoretical account of an influence of bodily, especially visceral, signals on emotions. The core idea of their theory is that emotional stimuli automatically induce bodily changes and that the feedback of these bodily changes to the brain is constitutive of the feeling of the emotion.4 Neurobiological support for this theory was recently provided, most notably by Damasio.5 In the 1920s, Cannon formulated an influential alternative theory, stating that emotions are generated in subcortical brain regions, especially the hypothalamus, which influences the viscera through the autonomic nervous system.6 The importance of Cannon’s work for the present issue is twofold. First, he was the first to formulate the idea of reciprocal interaction between gastrointestinal function and emotions. Secondly, Cannon put firm emphasis on physiological study of the influence of (unconscious) psychological