Evaluation of Esophageal Sensation

Salman Nusrat, MD[a], Philip B. Miner Jr, MD[b,*]

KEYWORDS

- Esophageal motility • Esophageal sensation
- High-resolution esophageal manometry • Multimodal esophageal assessment

KEY POINTS

- Dramatic progress has been made over the past decade in the sophistication and availability of equipment to test esophageal motility and sensation.
- High-resolution esophageal manometry and impedance have moved from the research clinic into clinical practice.
- Some of the testing is costly, time consuming, and requires extensive experience to perform the testing and properly interpret the results.
- The sensory studies are valuable in the interpretation of clinical problems, and provide important research information.
- Clinicians should evaluate the research studies to advance their understanding of the pathophysiology of the esophagus.

INTRODUCTION

Hippocrates noted in patients with "nausea, heartburn and salivation, there will be vomiting," setting the stage for exploring the relationship of esophageal sensory phenomena with pathophysiology of esophageal disease.[1] These 8 words capture the concepts of esophageal pain as an important clinical symptom, the bidirectional relationship of peripheral sensation and central nervous system response with nausea and vomiting, the complex gastrointestinal neurologic relationship of the esophagus and the stomach also causing vomiting, the relationship of esophageal sensation and sialorrhea, and the spectrum of gastroesophageal reflux from heartburn to vomiting. Rigorous clinical tests of esophageal sensation began nearly 6 decades ago with balloon distension, an exploration of referred pain locations associated with well-localized balloon placement throughout the gastrointestinal tract.[2] The

[a] Section of Digestive Disease and Nutrition, Department of Medicine, University of Oklahoma Health Sciences Center, Oklahoma City, OK 73104, USA; [b] Division of Gastroenterology, Department of Medicine, Oklahoma Foundation for Digestive Research, Oklahoma University School of Medicine, 525 Northwest 9th Street, Suite 325, Oklahoma City, OK 73102, USA
* Corresponding author.
E-mail address: philip-miner@ofdr.com

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esophageal findings were simple. The location of discomfort with balloon distension is always at or proximal to the anatomic location of the balloon distension. This simple experiment provided the explanation for the clinical observation that an obstructive bolus at the gastroesophageal junction may present as discomfort from near the suprasternal notch to the gastroesophageal junction. Awareness of the importance of gastric acid as a cause of esophageal discomfort opened new vistas in drug development. To evaluate the role of acid in inducing esophageal symptoms, hydrochloric acid was infused into the esophagus to test the sensitivity of the esophagus to acid (Bernstein test). Measurement of endogenous esophageal acid exposure associated with gastroesophageal reflux with continuous esophageal pH measurements began with a bedside pH monitoring platform and evolved into ambulatory 24-hour pH recordings documenting gastric and esophageal pH with concurrent symptom recording. The amazing resolution of symptoms and esophageal erosions associated with control of gastric acid secretion and esophageal acid exposure gave birth to the hypothesis that all heartburn could be explained by acid secretion, with nonerosive esophageal reflux disease simply exposure to acid at a lower level than the acid exposure associated with erosive esophagitis. This hypothesis began to flounder with the recognition that excellent acid secretory control did not improve symptoms in many patients with nonerosive gastroesophageal reflux disease. As the drugs used to control acid secretion have achieved generic status, a new era in understanding esophageal pathophysiology has emerged with the proposal of alternative hypotheses to explain esophageal symptoms. Many of the hypotheses are based on the physiology and pathophysiology of sensation. Contemporary research in esophageal physiology and pathophysiology carries more interest, innovation, and importance than ever before. Evaluation of esophageal sensation plays an essential role in these research efforts.

NEUROPHYSIOLOGY OF THE ESOPHAGUS

Normal and aberrant sensory function are critical neurophysiologic components of esophageal function and pathophysiology. A simple inventory of common symptoms including the discomfort from a spoonful of peanut butter that doesn't seem to move, chest pain with a frozen drink, sialorrhea after the first sip of hot tea, the cough associated with vomiting, and symptoms of regurgitation or heartburn attests to the complexity of esophageal neurophysiology.

The required anatomic and physiologic transition from skeletal muscles controlled by somatic nerves to the smooth muscles innervated by the autonomic nervous system further emphasizes the complexity of neurophysiologic control of the esophagus.

In addition, sensory discrimination is perplexing, as intraesophageal balloon inflation occurs as either chest pain or heartburn irrespective of balloon volume or location. The quality of symptom perception may be independent of stimulus.[3]

Esophageal sensory testing focuses on understanding the differences in sensory perception in the proximal and distal esophagus, the role of the enteric nervous system in sensory perception, the role of the physiologic stress-response system in stimulus perception, the importance of the neuroimmune axis with the associated subset of inflammatory and immunologically induced pain pathways, and the relative importance of pain amplification pathways and central processing of abnormal esophageal sensation.

Efforts to understand the important details of these concepts have generated the development of the techniques described in this section on sensory testing of the esophagus. A recent article summarizes the state of the art of neurophysiologic research in esophageal physiology.[4]