

Journal of Pediatric Gastroenterology & Nutrition

Issue: Volume 27(3), September 1998, pp 373-374

Copyright: © 1998 Lippincott Williams & Wilkins, Inc.

Publication Type: [Letters to the Editor]

ISSN: 0277-2116

Accession: 00005176-199809000-00027

[Letters to the Editor] [◀](#) Previous Article | Table of Contents | Next Article [▶](#)

Electrogastrography for Evaluating Neurologically Impaired Children with Recurrent Vomiting

Ravelli, Alberto M.

Author Information

Department of Pediatrics; University of Brescia; Ospedale Civile; Brescia, Italy

Author's Reply

To the Editor: We very much appreciate Dr. Bustorff-Silva's observations. As Dr. Bustorff-Silva implies, the major issue regarding vomiting in neurologically impaired children is the pathogenesis of vomiting itself. It is clear that primary gastroesophageal reflux can be particularly severe in these children because of several concurrent and aggravating factors (e.g., scoliosis, prolonged supine positioning, poor swallowing) that have been recalled in our report. It also seems clear that a central nervous system disorder involving the brainstem nuclei of cranial nerves or areas next to the area postrema and the "vomiting centers" may result in a severe and widespread disturbance of gastrointestinal motility or a persistent activation of the emetic reflex, thereby causing what should more appropriately be defined as "secondary" gastroesophageal reflux. Because disturbances of gastric motility, such as gastric dysrhythmia and delayed gastric emptying may occur in both cases (i.e., widespread motility disorder and activation of the emetic reflex), a distinction between the two mechanisms is not easy to draw. The simple clinical observation may provide some clues—for instance, the presence of retching and other autonomic manifestations, such as sweating, pallor, and tachycardia, would support the "reflex" rather than the "reflux" mechanism (1). Whether we are facing a motility disorder involving the gastrointestinal tract well beyond the lower esophageal sphincter or a persistent activation of the emetic reflex (or both), it seems clear that the Nissen fundoplication may not solve the problem, and in fact it may even make it worse. Dr. Bustorff-Silva rightly suggests that preoperative and postoperative electrogastrography should help clarify the issue of which patients would benefit from fundoplication. Indeed, we recently showed some preliminary evidence that patients in whom gastric electrical activity is normal before fundoplication tend to do well, whereas those who have gastric dysrhythmias beforehand tend to experience retching and severe postprandial discomfort after the fundoplication (2). Electrogastrography may thus contribute to the clinical and pathophysiological information that should

be gathered and carefully evaluated in neurologically impaired children with "refractory" gastroesophageal reflux, so that the pharmacologic, nutritional, or surgical therapeutic interventions can be more successfully tailored to the individual patient.

Alberto M. Ravelli

Department of Pediatrics; University of Brescia; Ospedale Civile; Brescia, Italy

[Back to Top](#)

REFERENCES

1. Richards CA, Andrews PLR, Spitz L, Milla PJ. Retching and vomiting in neurologically impaired children following fundoplication: Predictive pre-operative factors. *J Pediatr Gastroenterol Nutr* 1997;25(Suppl. 1):S45. [\[Context Link\]](#)
2. Ravelli AM, Richards CA, Spitz L, Milla PJ. Is Nissen fundoplication the optimal treatment for gastroesophageal reflux in children with neurological impairment? *Neurogastroenterol Motil* 1998;10:92. [SFX \[Context Link\]](#)