LETTER FROM THE EDITOR

The so-called Information Age, many will agree, has been both a blessing and a curse. Ease of access to healthcare information has enabled individuals to be better educated, empowered, and proactive about their care and care options. Access to patient and caregiver advocacy and support groups also abounds. But when members of the general lay public—or even autodidacts—process, reconstruct, and adapt information, promulgation of disinformation and misinformation can result. In other circumstances, patients might adopt care strategies that may be useful but do so ahead of systematic acceptance or thorough examination of the issue by their care providers.

The use of restrictive diets and vitamin supplementation as adjuncts or substitutes to medical/pharmacologic care is common. Patients, physicians, and the authoritative bodies that establish consensus guidelines are sometimes misaligned in this regard. The gluten-free diet, which is essential in patients with celiac disease, has become a nutrition fad adopted by a significant portion of the population who may ascribe to the notion that gluten and, by extension, wheat products contribute to a laundry list of maladies, including inflammatory bowel disease (IBD), irritable bowel syndrome (IBS), canker sores, rheumatoid arthritis, lupus, osteoporosis, anemia, various cancers, autoimmune disease, multiple sclerosis, clinical depression, dementia, schizophrenia, migraines, epilepsy, and autism. The lay public is not only warned by gluten-free-diet advocates to examine the gluten content of foods and condiments, but also to be wary of gluten lurking in cosmetics and even postage stamp adhesive.

Although research findings about the potential link between celiac disease (or gluten intolerance in general) and some of the diseases mentioned above are equivocal and ongoing, evidence for nonceliac gluten sensitivity (NCGS) is increasing. In this issue of Gastroenterology & Hepatology, Shanti Eswaran and colleagues examine emerging evidence for what many patients with IBS have been asserting despite skepticism from their physicians: Symptoms improve with a gluten-free diet. In 2009, the American College of Gastroenterology IBS Task Force announced that evidence linking diet to IBS was insufficient. Since then, the landscape has changed. It is increasingly being recognized that patients with IBS can have gluten sensitivity without overt signs of intestinal mucosal injury and also that such patients may have genetic markers for celiac disease (ie, human leukocyte antigen serotype DQ2 or DQ8).

Eswaran and colleagues provide food for thought about the mechanisms underlying the phenomenon of NCGS and provide convincing evidence that a gluten-free dietary



regimen has a place in the treatment of a subgroup of patients with IBS. The authors also note that this issue may be nuanced and complex; the offending substance might be gluten and/or a number of other proteins, namely fructan and galactan oligosaccharides, which are found in wheat, rye, and related grains. The authors note that evidence is emerging that dietary elimination of fermentable substrates (oligo-, di-, and monosaccharides and polyols) also may have a salutary role in the management of select patients with IBS.

The expanding role of capsule endoscopy (CE) in pediatric patients is explored in this month's other feature article through a comparison of meta-analyses that examine indications for CE in pediatric and adult gastrointestinal disease. CE does not require ionizing radiation or general anesthesia, making it a potentially key imaging tool in pediatric gastroenterology. Author Stanley A. Cohen reviews the diagnostic accuracy and safety profile of CE as well as methods of fine-tuning the use of this tool in young patients and specific gastroesophageal and gastrointestinal disease states.

The columns in this issue explore considerations for liver transplantation in patients with alpha-1 antitrypsin deficiency, emerging tools for measuring structural damage in Crohn's disease, new tests for evaluating laryngopharyngeal reflux, and endotherapy for organized pancreatic necrosis.

The case presented in this issue describes a rare instance of acute hepatitis B virus infection resulting in Guillain-Barré syndrome. The commentary following the case expands on its findings through a discussion of the extrahepatic manifestations of acute hepatitis B infection.

As always, I hope you find these articles informative and relevant.

Sincerely,

Gary R. Lichtenstein, MD, AGAF, FACP, FACG