

representative and even more to establish hypersensitivity to acid. Thus, these several difficulties in pH monitoring in a heterogeneous patient group do, in our opinion, prevent a new classification of oesophageal hypersensitivity to acid. A prospective and standardised study in these patients is needed to better define whether the hypersensitive oesophagus is a distinct clinical entity, or whether pH monitoring underdiagnoses gastro-oesophageal reflux disease.

J BOROVICKA
P MICHETTI
Division de Gastro-entérologie,
Polyclinique Médicale Universitaire,
Rue César-Roux 19,
CH-1005 Lausanne,
Switzerland

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Reply

EDITOR.—We would like to thank Drs Borovicka and Michetti for their interest in our paper and appreciate their critical comments.

We would not say that our group of patients was 'very heterogeneous'. Indeed, almost all patients studied complained of 'typical' reflux symptoms although they did differ from a pathological point of view, that is presence (and severity) or absence of mucosal lesions. The vast majority of them had a normal oesophageal mucosa, a finding recently reported by Trimble *et al*¹ who studied patients with normal acid exposure and a convincing correlation between symptoms and reflux episodes. Therefore, in this patient population, endoscopy is often meaningless and this is why it was not performed in 23% of our patients.

Our study is a retrospective one with the inherent drawbacks, but it reports data from a large series of consecutive patients from one centre. It is therefore understandable that pH monitoring was performed under different conditions. Some had an oesophageal manometry because of their predominant symptoms (for example, dysphagia or odynophagia) and, in these patients, manometric localisation of lower oesophageal sphincter (LOS) was used for positioning the pH probe. In those with no previous manometry, pH step up was used to localise LOS. Although manometric determination of LOS

may be the 'gold standard' to position the pH probe, a close correlation was discovered between the manometrically localised LOS and the sudden pH change occurring when the electrode is moved from the stomach to the oesophagus.^{2,3} The mean difference found by Klauser *et al*² between manometric assessment and pH step up technique was less than 0.5 cm, which is in agreement with our own experience.³

There are some physiological reasons to suggest that the type and amount of food ingested during the recording period as well as the degree of physical activity can actually affect the duration and the extent of oesophageal acid exposure.³ In the early studies performed in hospitalised patients and using stationary pHmetry, many authors including ourselves^{4,5} recommended to standardise diet and physical activity in the hope of reducing inter and intra-individual variability. Patients were told to avoid acidic beverages and foods to reduce artefacts and risks of confusion with reflux episodes. However, a free diet does not seem to change either the diagnostic value or the reproducibility of the technique.⁶ In addition, Jamieson *et al*⁷ reported that there is no significant difference, with the exception of the number of reflux episodes, between oesophageal pH recordings performed in an inpatient or outpatient environment. To identify the temporal relation between symptoms and reflux episodes, it becomes more and more important to emphasise the many advantages of ambulatory recording in patients engaged in everyday activities with no restriction regarding diet and exercise.³ In contrast, standardisation may unnecessarily affect the patient's regimen and reduce the ability of the test to detect a significant association between symptoms and reflux episodes.⁸

Although we did not assess the severity of symptoms in our patients, we correlated them with reflux episodes. When one or several symptoms occurred during reflux episodes or within two minutes of their end, and the possibility of this occurrence by chance was excluded by probability calculation, we assumed that refluxed acid had induced symptoms. As total acid exposure was within normal range and reflux episodes were actually shorter and less acidic than in patients with gastro-oesophageal reflux disease, an oesophageal hypersensitivity to acid could reasonably be hypothesised for this subgroup of patients. The presence of low grade oesophagitis in 19% of our patients indicates — as suggested by Heading's term¹ — that a greater mucosal sensitivity to damage may coexist with increased nociception in patients with 'acid hypersensitive oesophagus'.

J P GALMICHE
C SCARPIGNATO
S BRULEY DES VARANNES
G SHI

Hépatogastroentérologie,
Hôtel-Dieu, CHU de Nantes,
44035 Nantes Cedex,
France

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NOTE

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