Authors' reply

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Sakin *et al* [1] raised the following 3 concerns regarding our manometric and acid reduction in 3-h pH results: a) ambulatory pH monitoring shows only acid reflux; multichannel intraluminal 24-h pH-impedance (MII-pH) monitoring is needed to determine weak and non-acid reflux and also the exact beneficial results of *Helicobacter pylori* (*Hp*) eradication; b) high-fat meals have been shown to elicit heartburn and increased acid exposure, although, in our study, our patients' meal composition and meal type were not defined; and c) there was controversy whether proton pump inhibitor (PPI) use or *Hp* eradication benefited our patients.

However, as we initially mentioned [2], the main limitation of the 24-h pH monitoring is its low tolerability. Indeed, patients report that pH testing frequently induces unpleasant side effects lasting for the most part of the day,

and thus a shorter monitoring period is more tolerable. Moreover, it remains unidentified how weakly acidic or alkaline refluxate with a pH similar to a normal diet induces gastroesophageal symptoms. Most importantly, contrary to the previous studies mentioned by the authors [1], very recent data indicate that the 3-h postprandial recording provides an accurate prediction of absence or presence of gastroesophageal reflux disease (GERD) comparable to 24-h MII recording [3].

Regarding the second concern, it is known that, apart from high-fat meals and mealtime, mentioned by the authors [1], tomato products, alliums, sweets (chocolate), hot spicy food, citrus fruits and juices, peppermint tea, coffee, carbonated beverages, and/or alcohol are also contributors of GERD symptoms. However, instead of previous relative data mentioned by the authors [1], the role of diet as a risk factor for GERD has not as yet been clarified and recent relative studies are contradictory [4]. Nevertheless, our study patients had been advised to avoid consumption of such foods so as not to affect the study results.

With respect to the third concern, the authors misread our methods clearly stating that our patients had received rabeprazole once daily (q.d. means quaque die or once daily) and not 4 times daily after the initial 10-day Hp eradication therapy. Moreover, very recent data indicate that most GERD patients rendered asymptomatic on PPI therapy continue to experience abnormal esophageal and gastric acid exposure; the efficacy of acid suppression treatment, in certain patients, may be much lower than previously thought [5]. Therefore, since our patients received short-term (~40 days) PPI treatment and a second manometry and 3-h postprandial esophageal pH monitoring were introduced to assess the results of eradication therapy at 3-month post-treatment period, it is unlikely that the beneficial effect derives only from PPI use but rather by Hp eradication; rabeprazole has a half-life of less than 15 h, and rebound acid hypersecretion after administration of PPI has also been demonstrated in humans.

Finally, the references cited by the authors [1] to support their claims are irrelevant to the main aim of our study.

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Conflict of Interest: None

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Received 11 December 2014; accepted 16 December 2014