hands, MSA represents an effective, but less invasive alternative to fundoplication for the surgical treatment of GERD.[3,4] The impact of our novel understanding regarding the surgical anatomy of the esophagogastric junction (EGJ) for MSA placement remains to be seen.

According to the "vague" endoscopy-based model anatomy, the EGJ equals the level at which the tubular esophagus flares out into the saccular stomach.[1] In contrast to that, what has been taken for gastric cardia, in fact represents reflux-damaged, sphincter function-lacking esophagus lined by a columnar epithelium, ie, the dilated distal esophagus (DDE).[5] Fusion of morphology and function test data revealed that the level of the rise of the endoscopically visible gastric type folds equals the proximal limit of the DDE, the distal limit of the EGJ high pressure zone in GERD patients.[1,6] Accordingly, the DDE interposes between the tubular esophagus and the distal limit of the esophagus, ie, the peritoneal deflection, as assessed during laparoscopy.[1] In line with this morpho-functional understanding, the DDE represents the segment of the distal esophagus lacking adequate antireflux mechanism.[1] So placement of the MSA around the DDE, ie, dilated esophagus above the level of the peritoneal deflection, seems to be the ideal position for MSA placement. Here the MSA does not interfere with the functioning proximal portion of the lower esophageal sphincter and eliminates the major source of reflux: gastric dilatation-induced unfolding of the DDE, lacking an antireflux mechanism. Because the DDE and the peritoneal reflection are well defined, easy reproducible laparoscopic landmarks, this should help to standardize the LINX (Torax Medical) procedure. The authors are kindly asked to comment on the above suggestions for MSA placement.

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## Reply


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Laboratory and clinical studies have shown that the longer the lower esophageal sphincter (LES), the lower the LES pressure required to establish competence of the gastroesophageal junction.[1] Interrupting distraction of the LES by gastric wall tension is the most likely mode of action of the magnetic augmentation device; in other words, more gastric distension can occur before an opening LES pressure is reached.[2-4] It can be speculated that this effect is sufficient to prevent unfolding of the LES and to provide reflux control in patients with early gastroesophageal reflux disease and small hiatal hernia.

Asari and coworkers suggest placing the magnetic device above the peritoneal reflection and they assume that the device can work even in patients with dilated distal esophagus, a condition representing the point of no return, ie, the point at which the LES becomes mechanically defective.[5] Unfortunately, the clinical trials that have been carried out so far were designed to test the effectiveness of the device in patients with uncomplicated reflux disease, thereby excluding those presenting with esophagitis grade C and D, Barrett's esophagus, and hiatal hernia greater than 3 cm.[6,7]

We believe that placing the device too high above the peritoneal reflection or phrenoesophageal ligament, ie, around the thoracic segment of the LES, will result in failure of the procedure because there would be no protection against shortening of the distal or abdominal portion of the LES. Moreover, we suspect that in patients with advanced disease and a completely destroyed sphincter function, the device may not work because there is no LES to protect from shortening. In conclusion, placement of the magnetic device at the bottom of the LES remains the technique of choice to provide effective sphincter augmentation in patients with early-stage gastroesophageal reflux disease.

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