ABSTRACT. Objective. Few data exist in the literature about the relationship between percutaneous endoscopic gastrostomy (PEG) and gastroesophageal reflux (GER) in children, and the data that do exist are contradictory. The aim of the present study was to evaluate the effect of PEG on GER.

Methods. Twenty children underwent PEG for enteral nutrition. They were 55 ± 55 months old and weighed 13 ± 10 kg. A pH study was performed before and after PEG without treatment when GER status was unknown (n = 10) or under treatment (n = 10) if previous GER was demonstrated. In these cases, the pH study was performed under the same treatment before and after PEG.

Results. Six pH studies had abnormal results before PEG. After PEG, the GER of these 6 children significantly improved after the treatment was intensified (n = 5) or spontaneously normalized (n = 1). Results of 13 pH studies that were previously normal remained normal. Only one child with a normal reflux index before PEG had GER after it. For the 20 children, the mean reflux index did not change significantly after PEG (5.5% vs 5.6%).

Conclusion. Contrary to surgical gastrostomy, PEG does not worsen GER. Therefore, GER is not a contraindication to PEG. Pediatrics 1996; 97:726–728; percutaneous endoscopic gastrostomy, gastroesophageal reflux, child, infant, pH study.

ABBREVIATIONS. GER, gastroesophageal reflux; PEG, percutaneous endoscopic gastrostomy; SG, surgical gastrostomy; LES, lower esophageal sphincter.

Since its first description in children in 1980 by Gauderer et al.,1 percutaneous endoscopic gastrostomy (PEG) has been widely used in adults as well as in children.2–5 Gastroesophageal reflux (GER) is known to be frequent in children, especially in those requiring gastrostomy, and the following questions are frequently raised.6–15: (1) Is it necessary to look systematically for GER before gastrostomy? (2) If GER is present, should Nissen fundoplication always be proposed as a prevention? Surgical gastrostomy (SG) is reported to worsen GER,11,12,16 but the few data published on the effects of PEG on GER are contradictory.15,17–19 The aim of the present study was to evaluate the effects of PEG on GER.

METHODS

Twenty children and adolescents who underwent PEG for enteral nutrition entered the study. The population comprised 12 girls and 8 boys. They were 55 ± 55 months old (range, 4 months to 13.5 years) and weighed 13 ± 10 kg (range, 3.5 to 40.8 kg). They had neurologic diseases (n = 9; 45%), cystic fibrosis (n = 4; 20%), polycYSTIC malformation (n = 2), chronic pulmonary failure (n = 2), chronic kidney failure (n = 1), total Hirschsprung’s disease (n = 1) or failure to thrive (n = 1). Most of these children had deglutition abnormalities (70%) and/or were underweight (75%). All the children received enteral nutrition by nasogastric tube before the PEG procedure.

Fiberendoscopy (Olympus XP20) was performed under general anesthesia. Pediatric gastrostomy tubes 3 to 5 mm in diameter were used (Bard, Fresenius). The gastrostomy tube was inserted with the pull technique according to the method of Gauderer et al.1 The puncture site was located at the point of maximal translumination by finger pressure on the abdomen controlled by endoscopy. The ideal position was the antrum-fundus junction. PEG tubes were used 6 to 12 hours after it was put in place.

After informing parents and/or children, prolonged esophageal pH studies (>18 hours) were performed on all the children before and after PEG according to the European Society of Pediatric Gastroenterology and Nutrition standardized protocol.20 An antimony electrode was introduced into the nostrils and positioned at the level of T-7 to T-8,21 controlled by radiography. It was then connected to a pH meter (Synetics Medical Digitrapper Mark II Gold), the output of which was digitalized and stored for computer analysis. The electrode was calibrated at pH 1 and 7 before use. GER was defined as a pH of less than 4.0 for more than 5% of the time monitored (reflux index) for each of the study periods. The pH study was analyzed separately for daytime, nighttime, and the postprandial period, excluding the first hour after a meal.

The pH study was performed 1 day to 10 months (mean, 1.6 ± 2.4 months) before the PEG procedure and was repeated under the same conditions 5 days to 11 months (mean, 3.5 ± 3.4 months) after it, independent of GER status. The pH study was performed with antireflux medical treatment when GER was previously demonstrated (n = 10) or without when GER status was not known (n = 10). The treatment consisted of the association of prokinetics (n = 10), antacids (n = 7) and/or antiserective drugs (n = 3). Finally, 13 (65%) of 20 children had proven GER before PEG. Most of the children (16 of 20) had all-night continuous feeding (n = 12) and/or 1- to 3-hour-long inpatients (n = 7); none had all-day continuous feeding. There was no change in the feeding regimen after PEG.

Paired Wilcoxon’s t test was used to compare the reflux index before and after PEG. A P < .05 was considered significant.

RESULTS

Reflex index variations are reported for each child in the Figure. Results of 6 pH studies (three under treatment) were abnormal before PEG. The GER of these 6 children was later controlled (n = 4) or significantly improved (n = 1) when the antireflux treatment was intensified (increased dosage of pro-
kinetics associated with proton pump inhibitor) or even spontaneously normalized (n = 1; Figure). Results of 13 pH studies previously normal remained normal after PEG. Only 1 child with a normal reflux index before the procedure had GER after PEG. Under prokinetics but without antisecretory drugs, the control pH study showed a slight improvement of the reflux index. For the 20 children, the mean reflux index did not change significantly after PEG (5.5% ± 7.1% before vs 5.6% ± 7.3% after).

**DISCUSSION**

A high percentage of proven GER (65%) was found in the children studied here. This was also found by others in children undergoing gastrostomy, with the frequency of GER varying from 15% to 71%. This can result from the fact that most of the patients requiring PEG have neurologic diseases or cystic fibrosis, all circumstances known to be associated with a high prevalence of GER.

The influence of PEG on GER is an important question, because GER has been described as a frequent complication of gastrostomy, mainly SG. Different mechanisms have been proposed to explain how GER may occur after PEG. Anatomic changing could be involved in the process by increasing the angle of the lower esophageal sphincter (LES), but this occurs mainly after SG. Physiologic modifications also could be involved. According to Byrne and Jolley et al, LES pressure tends to decrease after SG. Conversely, Miller et al and Johnson et al show that LES pressure increases after PEG.

In our study, only one new GER occurred, and two that already were present initially worsened after PEG. The conditions in these three children were...
later controlled or improved with medical treatment. A few contradictory data exist in the literature about the relationship between GER and PEG in children as in adults. For six of seven adult patients, Johnson et al. showed, with prospective pre- and post-PEG pH studies, that reflux scores decreased. Gauderer et al. reported that only 1 of 32 infants without GER before PEG needed Nissen fundoplication 3.5 years after PEG removal, as opposed to 50% of those who previously had GER. The criteria for diagnosis of GER were not described in this study. Conversely, Grunow et al. showed with pre- and post-PEG pH studies that 6 of 10 children without GER before PEG had GER after PEG. Half of them later became symptomatic. The method of feeding (continuous or bolus) before and after PEG was not described in this study.

Sullivan suggested that the method of enteral feeding is important and recommended continuous feeding rather than boluses to avoid GER after gastrostomy. An intermittent bolus with high quantities of food increases intragastric pressure in disabled children. This was recently confirmed with esophageal manometric and isotopic studies in adults.

Few of our children received their feedings in short-duration boluses. There was no change in the feeding regimen after PEG. This suggests that the absence of a change in the feeding regimen could explain the absence of a modification in the GER status in our study, as opposed to the results of others. Moreover, GER in one child, which initially significantly worsened after PEG (reflux index, 22.8% vs 9.9% without treatment) later improved (reflux index, 5.2%) with intensive medical treatment and a change in the feeding regimen (Figure); instead of 2-hour-long boluses, he received continuous feeding.

Conclusion

Our data show that PEG does not precipitate or worsen GER and that GER should not be considered a contraindication to PEG. Because we did not find any modification of the GER status after PEG, we no longer recommend looking systematically for GER before PEG or controlling it afterward in the absence of clinical signs (vomiting, hematemesis, epigastric pain, or aspiration pneumonia). If GER is clinically controlled, Nissen fundoplication is not required before PEG. The Nissen operation should be performed after PEG only if intensive medical treatment and continuous enteral feeding fail to control GER.

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