Delayed gastric emptying affects outcome of Nissen fundoplication in neurologically impaired children

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Background. *Nissen fundoplication (NF) has a relatively high failure rate in neurologically impaired children with gastroesophageal reflux (GER). In 1990 we began to use routine technetium 99m sulfur colloid emptying scans and pyloroplasty with NF for delayed gastric emptying (DGE) in our neurologically impaired patients. The aim of this study was to determine the influence of DGE and pyloroplasty on the outcome of NF in neurologically impaired children.*

Methods. *One hundred neurologically impaired children underwent NF by a single surgeon between August 1986 and July 1995. Beginning in January 1990 emptying scans were routinely obtained, and patients with DGE underwent pyloroplasty with NF. Outcome analysis was performed for recurrence/wrap failure and other parameters. Mean follow-up was 5.8 years, with a minimum of 18 months.*

Results. *DGE was found in 35 (65%) of the 54 children who had emptying scans. All 11 children with normal scans had successful NF without recurrent reflux (100%). Forty (93%) of 43 children who underwent pyloroplasty and NF had successful outcomes. Thirty-eight children underwent NF without evaluation of gastric emptying with success in 30 of them (78.9%). Overall success improved from 34 (83%) of 41 in the first half of the study, when 3 (7%) of 41 children underwent emptying scans, to 55 (93%) of 59 in the second half, when 51 (86%) of 59 of the children underwent emptying scans.*

Conclusions. *DGE is common in neurologically impaired children with GER. NF in children with normal gastric emptying has a high probability of success. Pyloroplasty improves the outcome of NF in children with DGE. Neurologically impaired children should be evaluated for DGE before operation for GER. (Surgery 1997;122:690-8.)*

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RECURRENT EMESIS IS A SERIOUS problem affecting nearly 15% of all neurologically impaired children.[1] In most cases[2] it is caused by gastroesophageal reflux (GER) that does not respond well to medical therapy[3] and often leads to significant pulmonary and nutritional complications. Many of these handicapped children require supplemental tube feedings that may exacerbate the problem and are associated with a very low life expectancy.[4]

Previous studies have shown that morbidity and mortality of prolonged GER in handicapped children may be greatly reduced by antireflux surgery.[3,5-7] Nissen fundoplication is the most effective[8] and widely performed of these procedures, but it is attended by distressingly high complication and recurrence rates in this particular group of patients.[9-11] Possible explanations include predominant supine position, scoliosis, frequent seizures and spasms, bloating as a result of aerophagia and constipation, and gastroesophageal dysmotility.

We observed clinical signs of delayed gastric emptying (DGE) in many neurologically impaired children and began to obtain routine technetium 99m sulfur colloid gastric emptying scans. We performed pyloroplasty along with Nissen fundoplication in children with documented DGE and GER. The purpose of this study was to determine whether DGE predisposes neurologically impaired children to recurrent GER after fundoplication and also whether pyloroplasty reduces the risk of recurrence after fundoplication.

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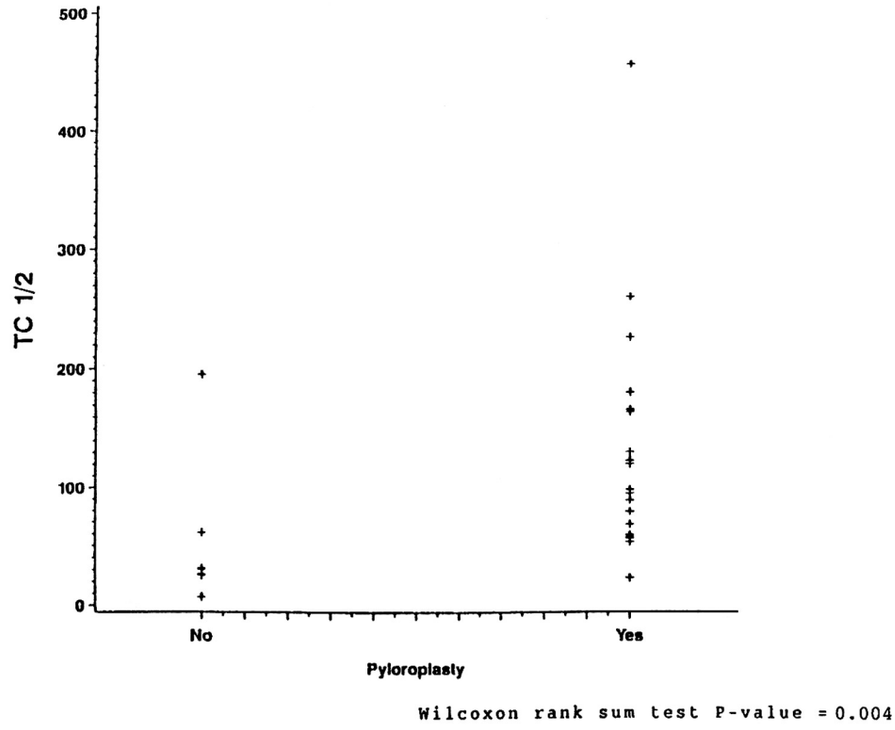

Fig. 1. Mean Tc½ was 50.6 minutes for patients with Nissen alone and 137.5 minutes for those with Nissen and pyloroplasty.

Table I. Causes of neurologic impairment in 100 children

Acquired impairment	
Anoxia/cerebral palsy	54
Encephalitits	3
Meningitis	2
Congenital disorders	
Structural	15
Other	7
Syndromes	
Chromosomal abnormality	9
CHARGE syndrome	2
Neuromuscular disorder	1
Metabolic disorder	2
Lissencephaly	3
Infantile spasms	2

CHARGE syndrome, Coloboma, heart disease, atresia choanae, retarded growth and retarded development and/or CNS anomalies, genital hypoplasia, and ear anomalies and/or deafness syndrome.

Table II. Operative complications

Early	
Splenic tear (splenectomy)	1
Leaking gastrostomy tube	1
Minor dysphagia (choking)	1
Dumping syndrome	1
Late	
Volvulus (death)	1
Minor dysphagia (choking)	1
Total	6

MATERIAL AND METHODS

During the 9-year period from 1986 to 1995, 127 children underwent operation by one surgeon (F.A.) at the Hartford Hospital and the Cleveland Clinic Foundation for symptomatic GER refractory to medical therapy. One hundred six patients were neurologically impaired; another 21 patients were neurologically normal and were excluded from this study. Six neurologically impaired patients were lost to follow-up.

Among the 100 patients who formed the basis of this study, 59 were male and 41 were female. The mean age at the time of operation was 6.6 years (median, 4.5 years; range, 1 month to 24 years). Thirteen children were younger than 1 year of age, 24 children were 1 to 3 years old, and 63 were 3 years or older at time of operation. All patients had severe motor and cognitive impairment. Infants and small children were minimally responsive and developmentally delayed. Older children were nonverbal, nonambulatory, incontinent, and incapable of self-care.

Major symptoms before operation were vomiting in 100%, failure to thrive in 95%, repeated episodes of aspiration pneumonia in 51%, apnea in 4%, esophageal hemorrhage in 2%, rumination in 2%, Sandifer's syndrome in 1%, and esophageal stricture in 1%. A seizure disorder was present in 64%, and 36% of patients were spastic quadriplegics. Twenty percent of patients were incapable of

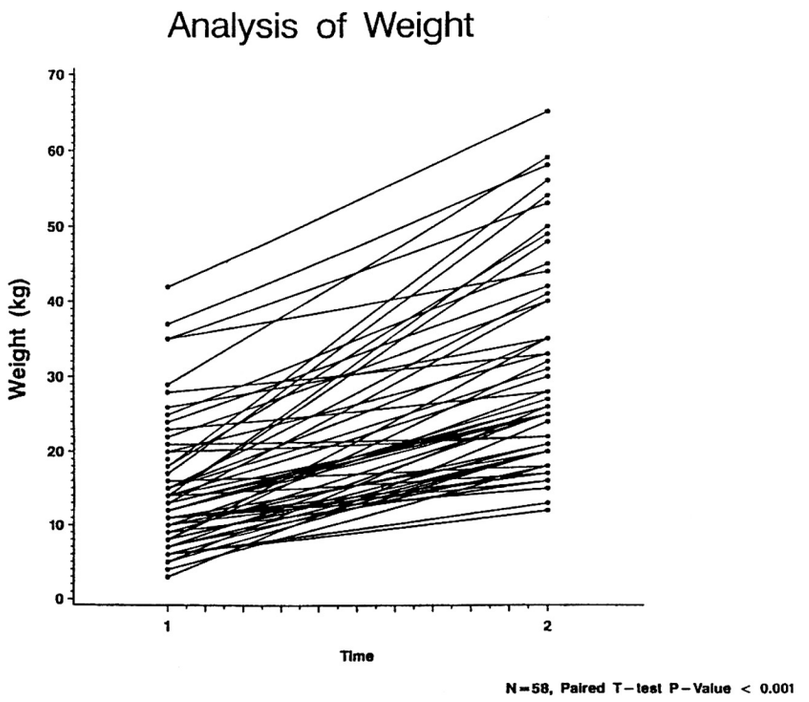

Fig. 2. Most patients demonstrated significant weight gain after operation.

eating by mouth and required tube feeds before operation. Associated anomalies were present in 3% and included esophageal atresia (2%) and VATER Syndrome (1%). Anoxia was the leading cause of neurologic impairment (54%), followed by acquired congenital disorders (22%), chromosomal abnormalities and other syndromes (19%), and acquired central nervous system infections (5%) (Table I).

The diagnosis of GER was confirmed in each patient by upper gastrointestinal series (UGIS), endoscopy, a 24-hour pH probe, or some combination of these. It has been our practice to obtain an UGIS initially to screen for both reflux and obstructive anomalies. Reflux extending into the upper esophagus (grade III), pharynx (grade IV), or mouth (grade V) is considered clinically significant and requires no other confirmatory studies. If the UGIS demonstrates absent or low-grade reflux, endoscopy is usually performed. We look for esophageal erosions, which are indicative of pathologic reflux. In our view, erythema is suggestive but not diagnostic of pathologic reflux. We perform biopsies if it is estimated that a 24-hour pH probe may not be technically feasible. Patients who are found to have erythema and those who are highly suspect for reflux without definite findings on UGIS or endoscopy undergo a 24-hour pH probe. Our experience has been that the most reproducible signs of pathologic reflux by pH probe are (1) total time of pH less than 4 (normal ≤ 4%) and (2) number (%) of episodes of pH less than 4 lasting longer than 5 minutes (normal ≤ 4%). In this study 88% of patients underwent UGIS, 46% underwent endoscopy, and 46% underwent a 24-hour pH probe. Pathologic reflux was confirmed by the above criteria in all patients.

Table III. Causes of death

Pneumonia	5
Unknown	4
Seizure	3
Cardiac arrest	2
Neurodegenerative disease	2
Strangulated gut	1
ARDS	1
Aspiration	1
Kidney failure	1
Total	20

ARDS, Adult respiratory distress syndrome.

In January 1990 we began to routinely obtain technetium 99m sulfur colloid gastric emptying scans (^{99m}Tc-Sc) in all neurologically impaired children with confirmed pathologic reflux. Our technique is to administer 5 μCi/ml ^{99m}Tc sulfur colloid by mouth or tube in the patient's usual volume of formula. The patient is placed in the supine position, and scintigraphic images are obtained every 10 minutes. The normal range of gastric emptying in infants and children is 50% at 1 hour.[12,13] If emptying is delayed at 1 hour, the

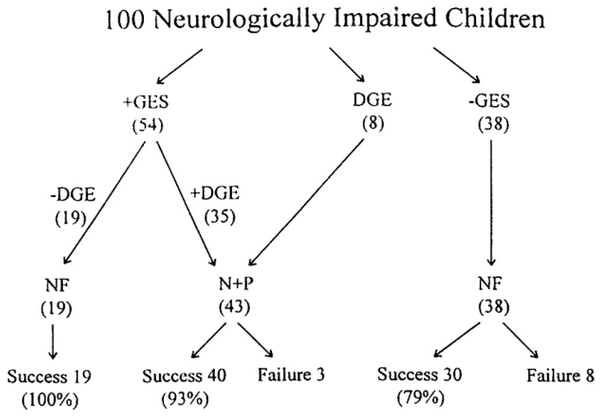

Fig. 3. Overall success with gastric emptying scans, 59 of 62 (95%).

patient may sit or walk about and a delayed image is taken at 1.5 hours. Gastric emptying is calculated as Tc ½,the time required to clear 50% of gastric contents.

After the diagnosis of reflux was made and before a decision was made to operate, each patient was given a trial of medical therapy consisting of small frequent oral feeds or continuous tube feeds, prokinetic agents including metoclopramide or cisapride, and H_2 blockers including cimetidine or ranitidine. Surgical therapy was expedited for patients with frequent episodes of aspiration pneumonia, esophageal stenosis, or hemorrhage.

Surgical therapy consisted of Nissen fundoplication and fundoplication with pyloroplasty in patients with documented DGE. The majority of patients also underwent concurrent gastrostomy tube placement for supplemental tube feeding. Salient features of the Nissen fundoplication were a short left subcostal incision, avoidance of incidental procedures such as appendectomy or umbilical hernia repair, division of short gastric vessels, preservation of both vagi with mobilization of intrathoracic esophagus, crural repair over an esophageal stent by using silk sutures and pledgets for atrophic crura, and a 360-degree short (3 to 4 cm) floppy wrap. The pyloroplasty was constructed by extending the incision 1 to 2 cm on either side of the pylorus with a single-layer closure with interrupted fine chromic sutures.

Outcome analysis was performed for patients undergoing operation before and after January 1990 and results were compared. The following parameters were studied: gastric emptying results, length of stay, days to feeding, postoperative complications, weight (gain/loss), subjective quality of life (4, symptoms significantly improved; 3, symptoms moderately improved; 2, no change; 1, symptoms worsened), recurrence/wrap failure and reoperation, and early and late mortality. Follow-up was obtained by chart review and by a telephone survey performed by a nurse clinician. The mean follow-up was 5.8 years (range, 18 to 124 months).

Statistical analysis was performed with Wilcoxon's rank-sum tests for group comparisons of continuous variables and chi-squared or exact chi-squared, as appropriate, for group comparisons of categorical variables. The paired t test was used for analysis of the changes over time. Kaplan-Meier estimates were used for summarizing the time-to-event variables, and log-rank tests were used for group comparisons. Results are considered statistically significant if p is less than or equal to an alpha level of 0.05.

RESULTS

Overall, 54 patients underwent preoperative gastric emptying scans. Gastric emptying scans showed DGE in 35 (65%) of the 54. Four additional patients were found to have DGE by UGIS in the absence of obstructive anomalies. Before January 1990 only 3 scans were performed (3 of 41; 7%), compared with 51 scans (51 of 59; 86%) during or after January 1990 ($p < 0.001$). There was no significant association between DGE and gender, seizure disorder, or spastic quadriplegia. However, DGE was more likely in patients with anoxic brain damage compared with other causes ($p = 0.009$) and was more likely in children older than 1 year of age ($p = 0.03$).

All 100 patients underwent Nissen fundoplication and gastrostomy tube placement. Eleven patients had undergone previous Nissen fundoplication by other surgeons. Eight of these 11 had preoperative gastric emptying scans, which showed DGE in all 8 (100%). Fifty-seven patients (57%) underwent Nissen alone. Nineteen of these patients had preoperative gastric emptying scans and 38 did not. Forty-three patients (43%) underwent Nissen and pyloroplasty. Thirty-five of these patients had DGE by scintigraphic scan, four patients had DGE by UGIS, and four others had either a previously failed Nissen (three) or clinical signs of DGE in the absence of a scan (one). The mean Tc ½ was 137.5 minutes (median, 120 minutes) in patients undergoing Nissen and pyloroplasty, compared with 50.6 minutes (median, 30 minutes) in patients undergoing Nissen alone ($p < 0.004$; Fig. 1). Pyloroplasty was performed in 6 (15%) of 41 patients before January 1990 and in 37

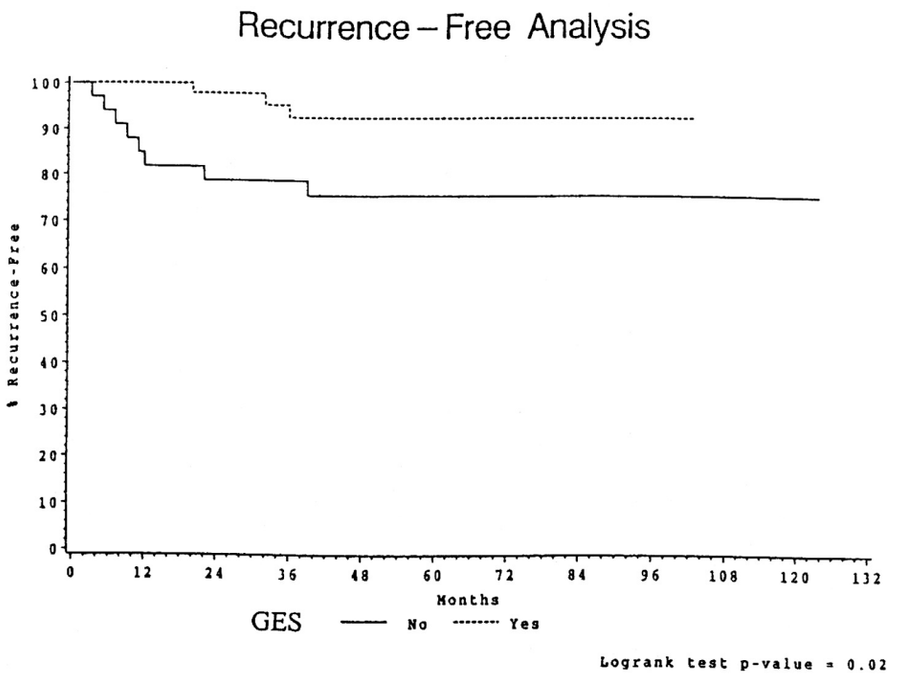

Fig. 4. Wrap failure/recurrence was significantly more frequent in patients who did not have preoperative gastric emptying scans.

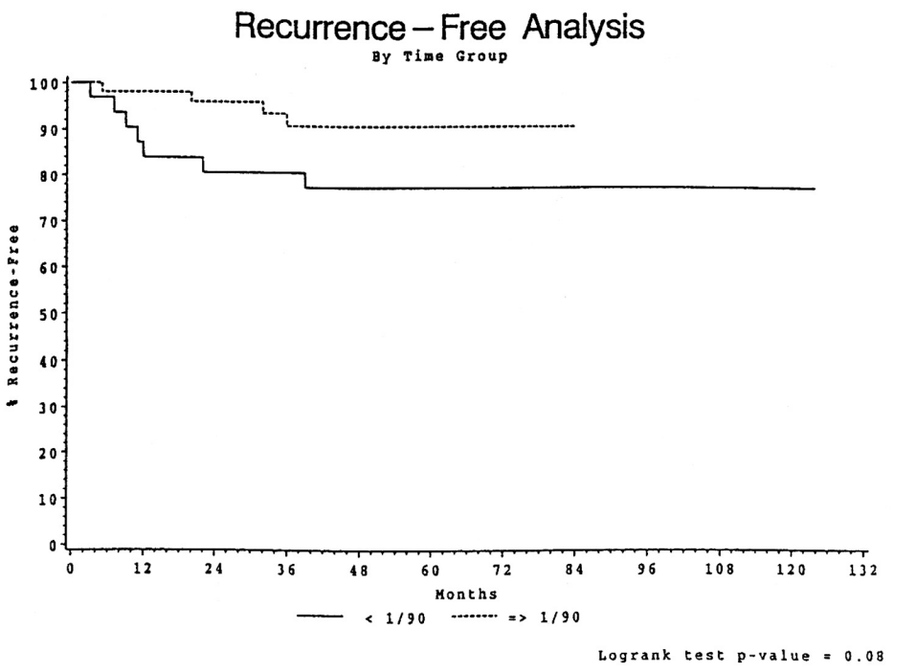

Fig. 5. Wrap failure/recurrence was more frequent before January 1990, when gastric emptying scans and pyloroplasty were not routinely used.

(63%) of 59 patients during or after January 1990 ($p < 0.001$).

Operative complications occurred in six patients (Table II). Four early complications included a splenic tear requiring splenectomy, a leaking gastrostomy tube, minor dysphagia (choking) that resolved with one dilation, and dumping syndrome that resolved with dietary measures. Two late complications include minor dysphagia in one patient, which resolved with one dilation performed 13 months after operation, and volvulus leading to intestinal necrosis and death 11 months after operation. There were no complications related to pyloroplasty.

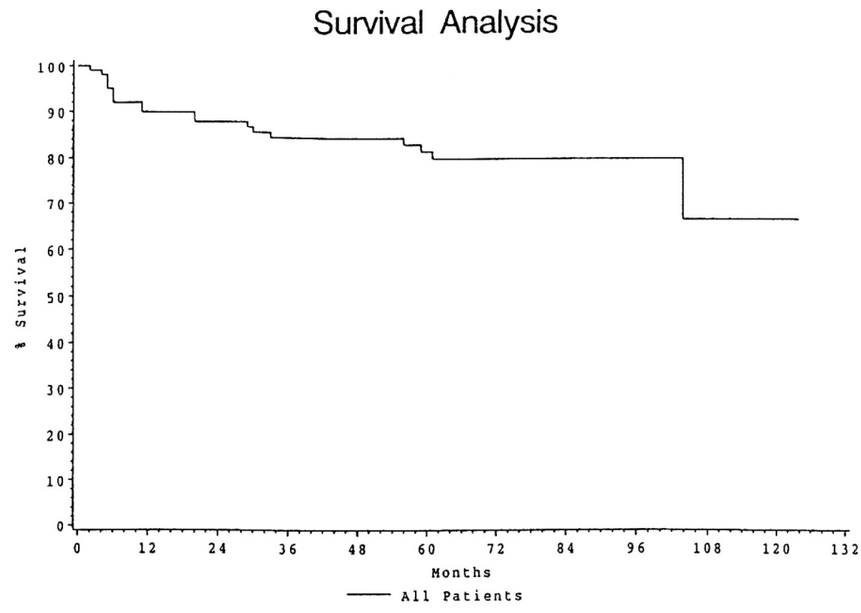

Fig. 6. Kaplan-Meier estimate of overall mortality.

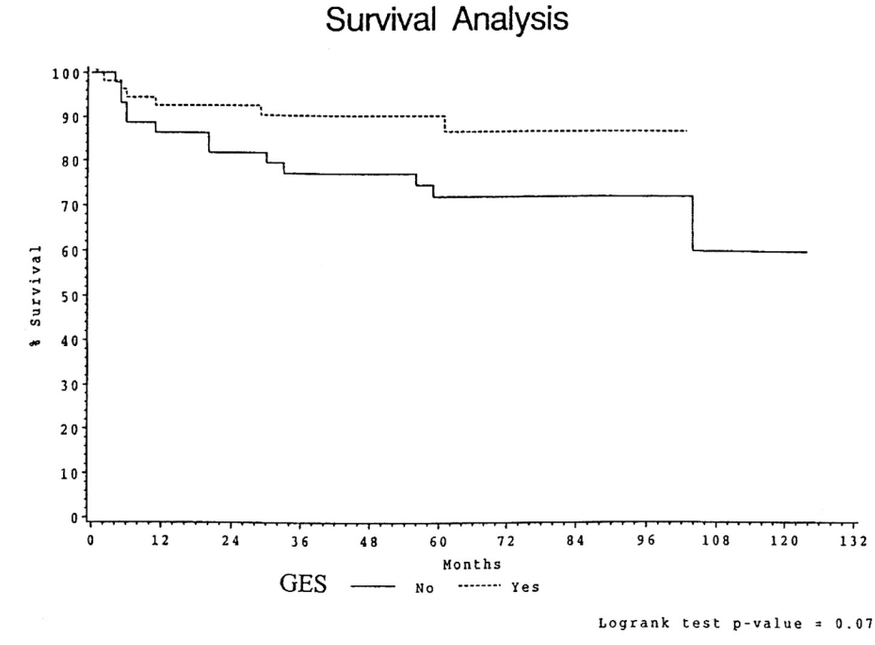

Fig. 7. Patients who had emptying scans showed improved survival.

The mean time between operation and feeding was 3.5 days (median, 3 days) and was significantly longer for patients operated on before January 1990 (mean, 4 days) compared with those operated on during or after January 1990 (mean, 3 days; $p < 0.001$). The mean length of stay was 7.4 days (median, 7 days) and was significantly longer for patients operated on before January 1990 (mean, 8.2 days) compared with those operated on after January 1990 (mean, 6.5 days; $p = 0.03$).

Ninety-five percent of the patients exhibited a failure to thrive before operation, and the vast majority were below the fifth percentile for weight.[14] After operation there was a significant increase in weight from 14.78 kg to 30.05 kg ($p < 0.001$; Fig. 2). Most patients also demonstrated significant improvement in subjective quality of life as judged by overall weight gain and tolerance of tube feeds without recurrent emesis or associated pulmonary problems. A telephone poll conducted by a nurse clinician determined that 74 patients (93%) were significantly improved (4), four patients (5%) were moderately improved (3), and two patients (2%) were unchanged after their

operation (2). No patients were symptomatically worse after operation. Of interest, 42 (52%) of 80 patients were tolerating solid foods by mouth in addition to tube feeds at the close of the study.

Routine diagnostic studies were not obtained after operation; however, all patients were closely monitored and symptoms suggestive of recurrent GER were promptly investigated with UGIS and gastric emptying scans. UGIS demonstrated a fundic wrap abnormality (herniation or disruption) in 100% of cases with documented recurrent GER. Wrap failure and recurrent GER occurred in 11 patients, all of whom required reoperation. Four of these failures were fundoplications performed alone without preoperative gastric emptying scans. Subsequent scans showed DGE, and the patients underwent redo fundoplication and pyloroplasty with excellent results. Four others underwent redo fundoplication without gastric emptying scans with one of four (25%) secondary failures. Three other failures occurred in patients who had fundoplications with pyloroplasty. Two of these patients required continuous tube feeds; the issue of DGE had not been addressed at the close of the study. The third patient underwent a redo fundoplication and pyloroplasty and was doing well at the close of the study. The mean time to wrap failure after Nissen alone was 13.5 months. The mean time to wrap failure after Nissen and pyloroplasty was 29.3 months.

Successful outcome without recurrent GER occurred in 19 (100%) of 19 patients with normal gastric emptying scans who underwent fundoplication alone and in 40 (93%) of 43 patients with DGE documented by scan or UGIS who underwent fundoplication and pyloroplasty (Fig. 3). Overall success in the group that had scans before fundoplication was 59 (95%) of 62 compared with 30 (79%) of 38 in those who underwent fundoplication without a gastric emptying scan ($p = 0.002$; Fig. 4).

Before January 1990 7 (17%) of 41 fundoplications failed, compared with 4 (6%) of 59 failures during or after January 1990 ($p = 0.08$). Although this difference is not statistically significant, Kaplan-Meier estimates reveal a definite trend toward lower failure rates in the second half of the study, when pyloroplasty was more commonly performed (Fig. 5). Of importance is the fact that the difference would have become significant if one failed Nissen fundoplication performed after January 1990 without a preoperative scan were eliminated from the study. In addition, six patients underwent successful NF with pyloroplasty before January 1990, which may have affected this result.

There were no perioperative or early deaths. Twenty patients died later during the course of the study (Table III). The mean time from operation to death was 25 months (median, 11 months; Fig. 6).

Only one patient died with symptoms suggestive of recurrent reflux. Fifteen others were tolerating tube feeds without emesis before death, including six who died with respiratory problems. Four others died of unknown causes at home but were likewise tolerating tube feeds. Of interest is that Kaplan-Meier estimates demonstrate an important trend toward improved survival in patients who had emptying scans compared with those who did not ($p = 0.07$; Fig. 7).

DISCUSSION

Our results indicate that DGE predisposes neurologically impaired children to wrap failure and recurrent GER after Nissen fundoplication. Preoperative assessment of gastric emptying and selective use of pyloroplasty appear to improve the outcome of fundoplication in these children.

We found that DGE was present in 40% of all patients in this study and in 100% of those with failed Nissen fundoplication. Previous studies have shown that DGE is present in a significant proportion of children[15,16] and adults[17,18] with GER. These findings suggest that GER is not simply the result of an abnormal GES mechanism but of a regional dysmotility disorder affecting the entire foregut.

In theory, DGE may cause gastric distention, which may lead to more frequent episodes of reflux. Chronic gastric distention may also play a role in feeding intolerance and in the gagging-choking-retching syndrome observed in some neurologically impaired children after fundoplication. Fundoplication enhances gastric emptying by decreasing fundal capacity and increasing fundal tone.[19] However, in some patients (e.g., the neurologically impaired) this beneficial effect may be obviated by the underlying motility disorder. Feeding intolerance is difficult to quantify but is often clinically apparent immediately after operation. Thus it is of interest that time to feeding and length of hospital stay were significantly decreased in the second half of this study, when emptying scans and pyloroplasty were routinely performed.

Wrap failure and recurrent GER after fundoplication occur more frequently in neurologically impaired children than in normal children. In large published series, recurrence rates after fundoplication range from 10% to 25% in neurologically impaired children[11,20,21] compared with 5% to 10% in normal children. By contrast, recurrence

rates range from 2% to 5% in several series in which emptying scans and pyloroplasty were used.[22-24] This comparison may be skewed owing to differences in surgical technique and patient population but does indicate the possible protective effect of pyloroplasty on the durability of the wrap in patients with DGE.

In this study surgical technique and patient population were controlled, and prospective comparisons were made possible by the introduction of emptying scans and pyloroplasty in January 1990. Wrap failure and recurrent GER were significantly less common in patients who underwent emptying scans ($p = 0.002$). Wrap failure was also less common in the second half of the study, when emptying scans and pyloroplasty were routinely performed ($p = 0.08$). The latter was not statistically significant because of inconsistencies in method relative to the timeline.

Operative complications were relatively rare in this series, probably related to surgical technique rather than the use of pyloroplasty. Small bowel obstruction occurred in only one patient but was associated with volvulus and death. Only one patient died with symptoms suggestive of GER. Fifteen other patients died of unrelated causes, and in four additional patients the cause of death was unknown. Survival analysis demonstrated a trend toward increased survival in patients who underwent gastric emptying scan. This may be artifact because more scans were performed in the second half of the study, in which there was a shorter follow-up. On the other hand, it does suggest an interesting possibility that deserves further study.

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DISCUSSION

Dr. Frederick J. Rescorla (Indianapolis, Ind.). You have concluded that when delayed gastric emptying was documented, pyloroplasty improved the success rate of Nissen fundoplication from approximately 83% to 93% with minimal morbidity related to pyloroplasty. This is similar to the findings of others, who have noted severe problems when delayed gastric emptying was not addressed at the time of

Nissen fundoplication. In the discussion you also point out that the high rate of delayed gastric emptying in neurologically impaired children lends support to the concept that these children have dysmotility of the entire foregut and not just the lower esophageal sphincter. I have several questions. (1) Because the higher fundoplication breakdown rate is in the earlier time period, do you think the breakdown rate in the second group may increase with a longer follow-up period? (2) Is persistent delayed gastric emptying a problem in the subgroup of children undergoing pyloroplasty and Nissen who subsequently experience breakdown of the fundoplication? Do you consider use of prokinetic agents in these children? Do you routinely or selectively evaluate gastric emptying in children with symptoms such as gagging after a Nissen and pyloroplasty? (3) When you have a Nissen breakdown do you modify your repeat fundoplication? Some have recommended tacking the completed wrap to the underside of the diaphragm. What do you do when it breaks down again and perhaps even a third time? Jay L. Grosfeld, MD, has looked at the J.W. Riley Hospital experience, and we have a 9% reoperation rate from the first procedure but a 19% reoperation after the second or third Nissen. After our third failure we generally resort to a jejunostomy tube.

Dr. Alexander. In answer to your first question, the mean length of time to breakdown in patients who had Nissen alone was approximately 15 months. This is quite consistent with what other surgeons have observed. The mean time to breakdown after Nissen and pyloroplasty was 28 months. Both time intervals are significantly less than the follow-up of 5.8 years in the study. Although it is difficult to draw conclusions from a retrospective study, it appears that the lower breakdown rate in the second half of the study was probably not artifact. What do we do with a breakdown? We always proceed to redo fundoplication. In this series there were 11 patients who had a failed Nissen. All of those patients were studied with emptying scans, and all were found to have delayed gastric emptying. All patients with delayed emptying underwent a pyloroplasty in conjunction with a redo Nissen. Of the 11 patients who experienced breakdown, all had redo fundoplications, pyloroplasty, and tube placement. Nine of the 11 are doing well at the present time. Two of the 11 underwent jejunostomy tube placement, and they appear to have persistent delayed gastric emptying, which has not yet been addressed. We don't modify the surgical technique of Nissen fundoplication at the redo procedure. It is an interesting point, but we have not done that.

Dr. Gerald M. Larson (Louisville, Ky.). I have a question about the cause of failure. You have just stated that failures in the early part of your study seemed to be due to breakdown of the wrap, and you imply that the delayed gastric emptying that you hadn't diagnosed in the first half of your study was a contributing factor. Do you have evidence of that? The Nissen in adults, properly performed, is very effective against preventing reflux esophagitis and aspiration pneumonitis, which were the main indications for your procedure. How secure are you that delayed gastric emptying was the single feature or a major causative event for the early recurrences?

Dr. Alexander. Again, this is a retrospective study and is subject to all the limitations of a retrospective study. I can only reiterate that patients who had delayed gastric emptying experienced failure more often after fundoplication than those who did not. A high percentage of patients who had delayed gastric emptying had breakdown of their wrap, and, after redo fundoplication with pyloroplasty, these patients are all doing quite well. Again, without a prospective study it would be difficult to determine the cause and effect. However, the Kaplan-Meier curves that I showed you appear to demonstrate a significant difference in outcome between those who underwent gastric emptying scans and pyloroplasty for delayed gastric emptying and those who did not.

Dr. Larson. Was there anything interesting in the anatomies and pictures of the stomachs in these infants who failed? Did they have very large stomachs, which would tend to put additional pressure on the wrap, for example?

Dr. Alexander. It was my clinical impression that the stomachs appeared to be more dilated in patients with delayed gastric emptying. In addition, I frequently noted muscular thickening and edema in the pylorus, much more than one would normally expect. These were subjective findings, however, and scintigraphic studies were our only objective measure of gastric emptying.

Dr. Charles E. Lucas (Detroit, Mich.). Previously we reported that closed head injury causes lower esophageal sphincter dysfunction (LES). Gastric tube feedings in such patients lead to aspiration, and thus the patients need tube feedings. This is a temporary thing, which reverses as the Glasgow coma scale goes from 11 back toward 15. More recent data that we have collected show that many patients with acute or even late strokes have the same phenomena. We don't know whether it is reversible. I presume your patients had LES dysfunction. First, would you speculate on the central mechanism that would lead to LES dysfunction? Second, would you further speculate as to whether this dysfunction, which presumably is central, is also associated with gastric pacemaker dysfunction or duodenal pacesetter dysfunction?

Dr. Alexander. With respect to the first question, there have been a number of studies that agree with your findings, one in particular by Dr. Dennis Vane, who showed that lower esophageal sphincter pressure dropped acutely in cats after head trauma, with a subsequent rise in pressure but never returning to normal. The mechanism of this change is unknown. It is interesting that there was an association in our study between delayed gastric emptying and anoxic brain damage. Patients with anoxic cerebral palsy had a significantly greater incidence of delayed gastric emptying than other patients in the series with other kinds of neurologic impairments. The reason for this is unclear. With respect to your second question, I really can't answer that. It would be pure speculation.