High Morbidity of Enterostomy and Its Closure in Premature Infants With Necrotizing Enterocolitis

Ann O'Connor, MD; Robert S. Sawin, MD

Objective: To review the morbidity and mortality among 68 premature infants treated with enterostomy for necrotizing enterocolitis.

Design: Data were collected retrospectively from hospital medical records to include the period between January 1, 1987, and September 30, 1997.

Setting: Tertiary care children’s hospital.

Patients: A group of 68 infants aged 2 to 35 days (mean age, 12.5 days), weighing 1500 g or less, with necrotizing enterocolitis necessitating surgical enterostomy for treatment.

Interventions: Creation of any enterostomy during exploratory laparotomy for necrotizing enterocolitis and subsequent closure.

Main Outcome Measures: Morbidity and mortality associated with infant enterostomy and its closure.

Results: Thirty-nine infants underwent ileostomy with mucous fistula, 16 underwent ileostomy with a Hartmann pouch, 7 had jejunostomy with mucous fistula, 2 had colostomy with mucous fistula, and 4 had colostomy with a Hartmann pouch. Eighteen (26%) of the 68 infants died in the postoperative period of sepsis (n = 10), continuing necrotizing enterocolitis (n = 5), or respiratory distress (n = 3). Of the remaining 50 infants, complications developed in 34 (68%). These complications included strictures requiring further resection at the time of enterostomy closure in 20 infants; stricture of the enterostomy requiring surgical revision in 6; incisional hernia in 3; parastomal hernia in 4; enterostomal prolapse or intussusception in 6 and 1, respectively; wound dehiscence in 4; wound infection in 8; small-bowel obstruction requiring laparotomy in 2; and anastomotic complications in 2. Only 16 enterostomies were closed uneventfully, with 3 of these infants subsequently dying of sudden infant death syndrome between 6 and 8 months after the operation. Of the surviving infants, 3 (6%) continue to require home hyperalimentation.

Conclusions: Although enterostomy in infants with low birth weight with necrotizing enterocolitis may be lifesaving, it is also a major cause of morbidity. These data suggest the feasibility of a prospective study comparing resection and primary anastomosis with resection and enterostomy.

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PREMATURNEONATEs undergoing surgical intervention for necrotizing enterocolitis (NEC) are undoubtedly among the most critically ill infants treated by pediatric surgeons. The resection of necrotic intestine with the creation of an enterostomy has been the accepted surgical therapy for NEC for many years. Although most authors agree that an enterostomy in patients with NEC can be lifesaving, others have suggested the use of primary anastomosis, drainage without resection, or even enterostomy without resection as superior methods. Few studies have reviewed the morbidity and mortality of enterostomy among premature infants with NEC. To determine the standard against which alternative surgical treatment of NEC can be compared, we retrospectively reviewed our surgical experience with enterostomy and its closure in premature infants with NEC during 10 years 10 months.

RESULTS

Seventy infants weighing 1500 g or less underwent exploratory laparotomy for NEC during the nearly 11-year review period. Two infants (3%) did not undergo resection due to NEC totalis. Of the remaining 68 infants, 18 (26%) died in the immediate postoperative period (day 0-30). Deaths were attributed to sepsis with multiple organ failure and disseminated intra-
PATIENTS AND METHODS

From January 1, 1987, to September 30, 1997, 148 infants required laparotomy for the treatment of NEC. Twenty of these infants underwent resection with primary anastomosis, and the remaining 128 infants had resection with enterostomy. Sixty-eight (53.1%) of these 128 infants weighed 1500 g or less (mean, 1097 g) and were the population studied. A retrospective review of all available medical records was performed to obtain as much follow-up information as possible. The patients undergoing enterostomy were evaluated for a number of complications, shown in the following table.

<table>
<thead>
<tr>
<th>Complication</th>
<th>Patients, No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Postoperative death</td>
<td>18</td>
</tr>
<tr>
<td>SIDS</td>
<td>3</td>
</tr>
<tr>
<td>Ileostomy stricture</td>
<td>6</td>
</tr>
<tr>
<td>Distal stricture</td>
<td>20</td>
</tr>
<tr>
<td>Anastomotic stricture</td>
<td>1</td>
</tr>
<tr>
<td>Anastomotic leak</td>
<td>1</td>
</tr>
<tr>
<td>Parastoma hernia (ileostomy)</td>
<td>4</td>
</tr>
<tr>
<td>Incisional hernia</td>
<td>3</td>
</tr>
<tr>
<td>Ileostomy prolapse</td>
<td>6</td>
</tr>
<tr>
<td>Wound infection</td>
<td>8</td>
</tr>
<tr>
<td>Wound dehiscence</td>
<td>4</td>
</tr>
<tr>
<td>Ileostomy intussusception</td>
<td>1</td>
</tr>
<tr>
<td>Small-bowel obstruction</td>
<td>2</td>
</tr>
</tbody>
</table>

(SIDS indicates sudden infant death syndrome.)

Infants ranged in age from 2 to 35 days (mean, 12.5 days) at the time of the initial resection. All infants were of less than 36 weeks' gestation, with 9 (13%) being of 32 to 36 weeks' gestation, 34 (50%) being of 28 to 31 weeks' gestation, and 25 (37%) being of 24 to 27 weeks' gestation. There was an equal distribution of male to female infants (31:37). Fifteen infants (22%) had concomitant anomalies, including patent ductus arteriosus, aortic coarctation, pulmonary atresia, tetralogy of Fallot, endocardial septal defects, duodenal atresia, and trisomy 21. Thirty-eight infants (56%) were also products of high-risk pregnancies, including young age (≤16 years old), women without prenatal care, women requiring urgent delivery (placental abruption, placenta previa, and eclampsia), and women addicted to narcotics or alcohol. Thirty-four infants (50%) had substantial infant respiratory tract disease requiring prolonged ventilation therapy (defined as >7 days), with 1 infant requiring home ventilator therapy before and after enterostomy closure. Serum albumin levels before resection and enterostomy were measured in 38 infants (89%) to estimate their nutritional status and ranged from 4 to 30 g/L (mean, 18 g/L), with reference levels in our institution ranging from 29 to 53 g/L.

Seventy-two infants underwent enterostomy at various levels based on the distribution of disease.

<table>
<thead>
<tr>
<th>Enterostomy Level</th>
<th>Patients, No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ileostomy with mucous fistula</td>
<td>39</td>
</tr>
<tr>
<td>Ileostomy with Hartmann pouch</td>
<td>16</td>
</tr>
<tr>
<td>Jejunostomy with mucous fistula</td>
<td>9*</td>
</tr>
<tr>
<td>Colostomy with mucous fistula</td>
<td>2</td>
</tr>
<tr>
<td>Colostomy with Hartmann pouch</td>
<td>4</td>
</tr>
<tr>
<td>No resection</td>
<td>2</td>
</tr>
</tbody>
</table>

(Asterisk indicates that 2 infants required multiple simultaneous enterostomies.)

Forty-five infants (66%) had evidence of perforation at the time of the initial resection. Most functional enterostomies were exteriorized at a point separate from the laparotomy incision, whereas most mucous fistulas were exteriorized through the edge of the laparotomy incision. The decision to create either a distal mucous fistula or a Hartmann pouch was made based on the ability to create the mucous fistula without sacrificing additional bowel length.

Fifty infants survived until the time of enterostomy closure and ranged in age from 1.5 to 20 months (mean, 5 months). Albumin levels were measured in only 19 (38%) infants at the time of enterostomy closure and ranged from 10 to 48 g/L (mean, 31 g/L). In general, albumin levels at the time of enterostomy closure were measured only in infants who either continued to require hyperalimentation (n=8) or were still in the hospital (n=11). The average weight gain for the 30 infants who survived to enterostomy closure was 2.4 kg.

The enterostomies were closed when the infants were medically stable or when the respiratory status was maximized. In 6 infants, enterostomies were closed early because of problems of high stomal output or poor weight gain. Closure was always preceded by a contrast study to ensure distal bowel patency and was usually accomplished by a single-layer, end-to-end anastomosis with a small, nonabsorbable suture.

vascular coagulation (n = 10), continued NEC with total remaining bowel necrosis (n = 5), or infant respiratory distress primarily due to prematurity (n = 3). Three infants (4%) died of SIDS between 6 and 8 months following enterostomy closure, making the overall mortality of 31% for the entire group of patients. There were no deaths as a direct result of enterostomy or its closure.

Eight infants continued to require hyperalimentation (mean of 41 days) at the time of enterostomy closure. Three infants were successfully weaned off home hyperalimentation following enterostomy closure, with only 3 requiring home hyperalimentation due to short-gut syndrome. Of the 3 infants who died of SIDS following enterostomy closure, 2 had been receiving home hyperalimentation at the time of their death. After intestinal continuity was restored and bowel function had resumed, enteral feedings were begun between 3 and 11 days (mean, 4 days).

At the time of the initial resection, the distribution of disease was recorded (Figure 1), as was the distribution of perforations. Disease was frequently distributed in more than 1 region. Forty-five patients (66%) had evidence of perforation at the time of the initial exploration, most of whom had gross peritoneal contamination. Perforations were frequently multiple and predominantly in the ileum and the ascending colon (Figure 2). Of the 18 patients who died in the postoperative period, 11 (61%) had multiple free perforations in several locations at the time of resection (as did all 3 infants who died of SIDS). Despite having similar disease distribution and severity, none of the infants who underwent un-
eventful enterostomy and closure had perforated NEC at the time of the initial resection. Only 16 infants (24%) underwent uneventful enterostomy and closure, with complications developing in 34 (68%) of the 50 infants who had survived until the time of enterostomy closure (Table 1). Many infants had more than 1 complication. Distal stricture developed in 20 infants (29%), predominantly (80%) among infants who required an ileostomy (Table 2). These strictures were always in the colon and were discovered by contrast studies before the enterostomy closure. Of the 55 ileostomies created, 6 (11%) developed a subsequent stoma stricture that required operative repair, usually following several failed attempts to dilate the ileostomy. Ten ileostomies (18%) required dilatation but no operative repair. No enterostomal strictures developed in those created at the jejunal or colonic level.

Parastomal hernias requiring repair developed in 4 patients (6%). All of these hernias were among infants with ileostomies and occurred at the enterostomy only, regardless of whether a mucous fistula was created. All 6 (9%) of the prolapsed enterostomies and the single intussuscepted enterostomy were also limited to infants with ileostomies, and all of these patients had concomitant distal mucous fistulae (Table 2). None of the distal mucous fistulae underwent retraction.

Complications with the laparotomy incision developed in 15 infants (22%). In 3 infants, incisional hernias developed that were repaired at the time of the ileostomy closure. Eight infants had wound infections that were treated with dressing changes and antibiotics. Four of the wound infections developed in infants who had a colostomy and a distal mucous fistula, 2 in patients with a jejunostomy and mucous fistula and 2 in patients with ileostomies (1 with a mucous fistula and 1 without).

Wound dehiscence requiring operative repair developed in 4 infants, all of whom later died of multiple organ failure and sepsis. The incidence of wound dehiscence was evenly distributed among infants with an ileostomy and those with a jejunostomy.

Small-bowel obstruction developed in only 2 patients, 11 and 18 months, respectively, after the enterostomy closure. Both patients had previously had multiple enterostomies at the jejunal level with distal mucous fistulae for the treatment of NEC. Both patients required adhesiolysis without further bowel resection and have remained well.

Anastomotic complications developed in 2 infants after enterostomy closure. One infant required reoperation 10 days after the initial operation due to stricture formation, and another was reexplored 3 days postoperatively.
due to disruption of the anastomosis. This latter infant had been receiving parenteral steroid therapy because of his poor respiratory status at the time of the anastomosis but did well after the disruption was repaired.

The percentage of complications as a function of disease distribution at the time of resection demonstrates that most infants who died in the immediate postoperative period had ileal (95% \([n = 17]\)), ascending colon (67% \([n = 12]\)), and transverse colon (57% \([n = 10]\)) disease, with disease distributed less frequently in the jejunum and distal colon. The same pattern of disease distribution applies to nearly all the other complications listed, with the exception of wound infections, which appeared more frequently associated with colonic NEC.

### Table 2. Enterostomal Complications by Location in 68 Premature Infants With Necrotizing Enterocolitis

<table>
<thead>
<tr>
<th>Enterostomy</th>
<th>Stomal Stricture</th>
<th>Distal Stricture</th>
<th>Hernia</th>
<th>Prolapse</th>
<th>Intussusception</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ileostomy with mucous fistula</td>
<td>4 (67)</td>
<td>10 (60)</td>
<td>2 (50)</td>
<td>6 (100)</td>
<td>1 (100)</td>
</tr>
<tr>
<td>Ileostomy with Hartmann pouch</td>
<td>27 (33)</td>
<td>6 (30)</td>
<td>2 (50)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Jejunostomy with mucous fistula</td>
<td>0 (0)</td>
<td>3 (15)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Colostomy with mucous fistula</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Colostomy with Hartmann pouch</td>
<td>0 (0)</td>
<td>1 (5)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

The standard surgical therapy for premature infants with NEC has long been the resection of necrotic intestine with enterostomy for both decompression and diversion.\(^1\)\(^,\)\(^5\)\(^,\)\(^8\)\(^,\)\(^11\) Debate continues in the literature\(^6\)\(^,\)\(^9\)\(^,\)\(^10\) regarding the technique of resection and primary anastomosis for infants with NEC, although primary anastomosis remains inherently attractive. Objections to resection and primary anastomosis are multiple\(^6\)\(^,\)\(^11\) and include the fear of increased anastomotic complications, the resection of more bowel than necessary, and the concept of enteroileal decompression until an optimal weight has been maintained before intestinal continuity is restored. Our series of premature infants with NEC treated with resection and enterostomy demonstrates that although enteroileal decompression may be lifesaving, it is not without significant morbidity and mortality.

This series shows a mortality of 26% in the immediate postoperative period, which is similar to rates described by other authors.\(^1\)\(^,\)\(^4\)\(^,\)\(^5\)\(^,\)\(^7\)\(^,\)\(^8\)\(^,\)\(^11\) Most infants had disease distributed predominantly in the ileum and ascending colon, which is also consistent with other reports. Accordingly, perforations were most common in the ileum and ascending colon. The observed mortality of 24% among infants with intestinal perforation did not differ significantly from that for infants without perforation (26%). None of the infants who underwent treatment without complication had perforation at the time of the initial resection, implying that whereas the mortality is not different, perforation may be a prediction of increased morbidity.

Most striking in this series is the high complication rate observed among infants treated with enterostomy. Our series confirms many authors\(^3\)\(^,\)\(^5\)\(^,\)\(^7\)\(^,\)\(^9\)\(^,\)\(^10\) observation that the most common complication of enterostomy is distal bowel stricture. Our observed distal stricture rate of 40% is similar to the published stricture rate of 38%\(^1,\)\(^1^2\) among infants evaluated with contrast studies following nonoperative treatment of NEC. Half of the strictures found in the medically treated patients with NEC were asymptomatic and not treated.\(^1,\)\(^1^2\) In our series, all distal strictures were resected at the time of anastomosis, and therefore, the prospective use of contrast studies influenced the surgical management of these patients. Perhaps strictures are less likely to resolve among infants with more severe NEC treated with enterostomy.

Distal strictures were found predominantly among infants who were treated with an ileostomy. The percentage of distal strictures in these infants did not differ significantly between those who had a distal mucous fistula (50% \([n = 10]\)) and those who did not (30% \([n = 6]\)). The strictures were located in the descending and rectosigmoid colon almost exclusively, implying that NEC may cause continued damage in those areas of intestine that are deemed grossly free of disease at the time of resection. It is also possible that the blood supply to the remaining distal bowel is tenuous, resulting in ischemia and subsequent stricture formation. Perhaps the early restoration of intestinal continuity provides intrinsic dilatation due to fecal buloses or encourages the trophic effects of feedings and succus entericus. The timing of enterostomy closure among infants with NEC is debated,\(^3\) and our data support the need to investigate further the role of early restoration of continuity with regard to the rate of distal stricture.

The creation of an ileostomy, either with or without a distal mucous fistula, proved to be a morbid procedure. Ileostomy complications have been reported,\(^9\)\(^,\)\(^1^0\) but our series reports a markedly higher rate. Of the 55 ileostomies, 6 (11%) developed stricture and required revision after failed attempts at dilation. Half of these infants underwent anastomosis at that time, which was generally considered sooner than planned. Prolapse occurred in 6 (11%) of the 55 ileostomies, all of which were repaired at the time of anastomosis. The only intussuscepted ileostomy (which was a case of prolapse severe enough to result in complete obstruction) required urgent exploration and the resection of approximately 5 cm of distal ileum in addition to an anastomosis. Four parastomal hernias occurred in this group, with all of them repaired electively at the time of anastomosis. When wound infection \((n = 2)\), wound dehiscence \((n = 2)\), small-bowel obstruction \((n = 1)\), incisional hernia \((n = 2)\), and distal stricture \((n = 16)\) are included with the aforementioned complications, the total percentage of ileostomies with complications is 73%. If distal stricture is excluded from the list of complications, the percentage of ileostomy complications decreases to 44%, which is closer to the rate reported by others.\(^9\)\(^,\)\(^1^0\)
Although only 9 infants underwent jejunostomy with mucous fistula, there were a total of 9 complications. In addition to the infants who suffered wound dehiscence and died, small-bowel obstruction later developed in 2 infants. Both infants had multiple ileal and jejunal enterostomies for the initial treatment of NEC. Both infants had dense adhesions but needed no further bowel resection at the time of exploration for small-bowel obstruction. Again, if distal bowel stricture is not included in the list of complications, the percentage of complications decreases to 67%, which approaches the rate reported by other authors. Most complications associated with colostomy in these infants (67%) were wound infections. Only 1 infant had a distal stricture develop after colostomy with a Hartmann pouch.

Of the 50 infants who underwent ileostomy closure, only 2 had anastomotic complications, and 3 later died of SIDS. Our rate of anastomotic complications is lower than that reported by some authors. Differences between the postclosure group complication rate (10%) vs the preclosure group complication rate (68%) may be ascribed to factors other than the technical creation of the enterostomy.

Most of these premature infants exhibited some degree of malnutrition (mean albumin level, 18 g/L) at the time of enterostomy and resection, but not at the time of stomal closure (mean albumin level, 31 g/L). In addition, all infants who suffered wound dehiscence after both ileostomy and jejunostomy were malnourished (albumin levels, 4–18 g/L) and subsequently died of continued sepsis. Also, many infants received mechanical ventilation for prolonged periods following resection and enterostomy. Many infants were taking broad-spectrum antibiotic therapy and hyperalimentation for prolonged periods following enterostomy creation. It is also possible that to save as much intestine as possible, enterostomies were created using bowel that had tenuous viability. These factors imply that the high complication rate among infants with an enterostomy in this series may be due to poor nutritional status, ongoing sepsis, respiratory distress, and continued NEC.

Because the operative management of NEC is so complex, some authors have adopted alternative surgical strategies. Few of the patients in our series were treated with preoperative peritoneal drainage, and none were treated with “patch, drain, and wait” or enterostomy without resection. The claim for both techniques is that they maximize intestinal salvage while providing adequate decompression and a short operation for infants who are premature and critically ill. The usefulness of these approaches has not been universally accepted, perhaps because philosophically and technically, treatment of these premature infants has changed from the need to perform the shortest operation to the ability to perform the most inclusive operation.

During our review period, 20 infants underwent resection with primary anastomosis for NEC. Of these 20 infants, 10 (50%) weighed 1500 g or less. Complications developed in only 2 (20%) of these 10 infants and included 1 death from overwhelming sepsis with multiple organ failure and 1 wound infection treated with dressing changes and antibiotics. In none of the infants did distal strictures or anastomotic complications develop (data not shown). The possibility remains that these patients with primary anastomosis were a selected group of infants who were less critically ill than the infants who underwent enterostomy, but the results of this limited review suggest that resection with primary anastomosis may be a safe alternative to enterostomy.

### CONCLUSIONS

Necrotizing enterocolitis remains a difficult management issue for pediatric surgeons. Our data show that enterostomy in premature infants is a seriously morbid procedure, which supports the need to prospectively compare resection and primary anastomosis with resection and enterostomy.

**Presented at the 69th Annual Session of the Pacific Coast Surgical Association, Maui, Hawaii, February 18, 1998.**

**Reprints:** Robert S. Sawin, MD, Department of Surgery, Children’s Hospital and Regional Medical Center, University of Washington School of Medicine, 4800 Sand Point Way NE, Mailstop CH-78, Seattle, WA 98105.

### REFERENCES


### DISCUSSION

Harry Applebaum, MD, Los Angeles, Calif: Drs O’Connor and Sawin have compiled an excellent analysis that can greatly help pediatric surgeons deal with one of their more common and most frustrating problems—what to do in the middle of the night when confronted with gangrenous bowel in a very sick and very small premature infant. As they note, the choice for an initial operation in NEC usually combines a conservative resection of compromised bowel with proximal diversion. Unfortunately, this seemingly safe surgical approach is too often the prelude to a whole chain of adverse events that are often related to the stoma. And, as might be anticipated, the sicker an infant at the outset, the greater the number of complications that can be expected.

They, therefore, suggest that primary resection and anastomosis might supplant the staged approach, theorizing that if there are no stomas, they cannot cause problems. Although this concept was first introduced by Harberg more than 15 years ago, it has failed to gain wide acceptance. Most still find it suitable only for selected good-risk patients of the type that successfully un-
derwent a 1-stage procedure in this nonrandomized study. Primary anastomosis requires the excision of any questionably viable bowel, thus increasing the risk of the short-gut syndrome, and would be difficult to apply to the large number of patients with skip areas of involvement. And, most important, the stoma-related complications that are reported pale in comparison with those that would accompany any potential anastomotic disruptions. It is also not likely that a primary anastomotic approach would have benefited the 26% of patients in this series who died of sepsis and the progression of disease in the immediate postoperative period. The authors conclude that a prospective trial comparing resection and enterostomy with resection and primary anastomosis is warranted. With the preceding concepts in mind, I would be interested in hearing their thoughts on which patients should be specifically included or excluded from a randomized trial that compares these 2 alternatives.

Second, in my experience, the incidence of stomal complications is directly related to the length of time that the stoma is left in place. Although most pediatric surgeons continue to use stomas, the trend has been toward more rapid closure, with a perceived decreased incidence not only of stoma-related problems but also of short-gut symptoms and total parenteral nutrition-induced cholestasis jaundice. In this series, the average age for closure was 5 months. Would earlier stomal closure safely lead to fewer complications, and how should we currently determine its optimal timing?

Last, some stricture-related issues: The postoperative stricture rate is comparable to that observed in other series of less severely affected patients who could be managed nonoperatively. This would suggest that strictures are a consequence of the disease rather than of stomas and fecal diversion. Also, perhaps 50% of radiographically diagnosed strictures in nonoperative patients never become clinically significant. When discovered in surgically treated patients before stomal closure, could some strictures be balloon dilated or even ignored, rather than routinely resected?

**John German, MD, Orange, Calif.** The invited discussant hit all of the right points that were asked by this paper.

Dr Paul W. Johnson and I have wondered exactly the issue Dr Applebaum brought up. Is definitive surgical intervention for these patients delayed beyond what we consider a window of opportunity? In the management of these patients, once you get them as stable as you possibly can, it doesn’t get any better than that. The reason we recommend that is we start seeing the complications out of the other questions as well. There is a theory that something about the small-bowel succus entericus is preventing these distal strictures. Some people theorized that if you do a proximal diversion, perhaps you should feed the distal bowel, and then you will prevent that distal stricture from developing. Our experience reported here would support that in the infants who had colostomies, distal strictures never developed—only those who had proximal small-bowel diversion. Interestingly, almost all of the strictures were in the rectosigmoid. I can’t really explain that. That leads to Dr Tapper’s question about why children with distal strictures do so poorly. I really don’t have a good explanation for that, unless it can be attributed to what Dr German alluded to, and that is that perhaps had we closed them earlier, this distal stricture would not have developed. Perhaps what we are seeing is that stricture is a marker of having waited too long.

Dr Organ asked about the Santulli operation. It still has a place in pediatric surgery, but I don’t think it does in patients with NEC. It’s probably something that we use more frequently in some of the complicated intestinal atresias. It is an interesting idea, but I have never seen it used in the treatment of these patients with NEC.

Dr Crass asked about wound infection, particularly in the patients with a colostomy. I would concur that it is a high enough rate that I have changed the way I close those incisions. Typically, rather than closing them in an airtight manner, I put just a few loose interrupted sutures in and maybe even put some wicks in between. Despite that, we still see a high incidence of wound infection.

**Dr Ryan:** Dr Ryan asked about wound infection, particularly in the patients with a colostomy. I would concur that it is a high enough rate that I have changed the way I close those incisions. Typically, rather than closing them in an airtight manner, I put just a few loose interrupted sutures in and maybe even put some wicks in between. Despite that, we still see a high incidence of wound infection.

Dr Applebaum asked about the severity of the complications. Were we to do more primary anastomoses, would we be trading off stomal complications for much worse? I think that Tom Moore’s revolutionary concept of the “patch, drain, and wait” and the other people who advocate primary bedside drainage would indicate that maybe the most important part of this operation is doing the laparotomy and irrigating out everything that is going on in there. How we handle the bowel may be less important because, even if you did a primary anastomosis and had an anastomotic leak down the road, perhaps that wouldn’t be as big a problem 2, 3, or 4 days after you have resuscitated them with this laparotomy.

Regarding balloon dilatation and whether all of these distal strictures need to be treated, we have had little success with balloon dilatation of these post-NEC strictures. I don’t know why that is. I have seen 1 child in particular who initially had a balloon dilatation, then a stricturoplasty, and then ultimately a resection. The histopathological examination showed an intense fibrosis, and I just think it’s not amenable to dilatation.

Perhaps the most important question is the one that Dr Applebaum raised. If we were to do a prospective trial, how could we possibly do it without having apples and oranges? It would have to be a pretty homogeneous population, and, in fact, in our series, about 60% had fairly homogeneous disease, ie, distal ileal and cecal disease. If we chose a specified population like that, perhaps we could get enough pediatric surgeons to agree to a randomized trial.