Small Intestinal Atresia:

The Critical Role of a Functioning Anastomosis

COLIN G. THOMAS, JR., M.D., JOEY M. CARTER, M.D.

From the Department of Surgery, University of North Carolina School of Medicine and the North Carolina Memorial Hospital, Chapel Hill, North Carolina 27514

A review of 34 patients with small intestinal atresia treated at the North Carolina Memorial Hospital 1952–1973, demonstrates this anomaly to be associated with a significant mortality. Six of eight anastomotic complications occurring in the 34 patients were lethal. While factors leading to mortality are multivariate, e.g., prematurity, sepsis, cardiorespiratory failure and malabsorption, with the ability to maintain nutrition and with other remedial anomalies, the construction of a properly functioning anastomosis may be the most critical factor in the survival of the patient.

An end-to-end anastomosis with conservation of absorptive surface for jejuno-ileal atresia is preferable and is facilitated particularly in the proximal jejunum by a graduated type of jejunoplasty. Duodenal atresia, because of the short segment involved and proximity of bile and pancreatic ducts does not lend itself to comparable reconstructive procedures. However, recognition of the delay in function that may result from anastomosis of a dilated to diminutive segment of bowel permits the immediate initiation of measures to maintain nutrition either via a transanastomotic feeding catheter, or parenteral alimentation.

ADVANCES IN THE CARE of the neonatal surgical patient have resulted from a better understanding of the metabolic peculiarities of the infant, an improvement in ability to control infection, superior anesthetic techniques and maintenance of nutrition. Meticulous attention to deail—pre, intra and postoperatively—underlies all these aspects of management and plays an important factor in improved suvivorship. The ability to sustain nutrition in an infant with a life-threatening anomaly has made even more critical the methods of correcting such an anomaly. This has become increasingly apparent in small intestinal atresia, a malformation that occurs approximately once in 20,000 live births and remains the leading cause of intestinal obstruction in the newborn.[24] Although the mortality rate has been strikingly reduced from the 91% reported in a review of 1498 patients by Evans[5] in 1951, it is still significant. The surgical Section of the American Academy of Pediatrics reported the following survival rates during 1957 to 1966: duodenal atresia or stenosis, 68%; jejunal atresia, 58.6%; and ileal atresia, 75.3% (4,6). More recently Santulli *et al.*,[19] Nixon *et al.*[16] and White *et al.*[24] gave overall survivorships in small intestinal atresia of 54%, 61% and 80% respectively. Despite the obvious essentiality of restoring intestinal continuity, there is no unanimity as to the optimum method of intestinal reconstruction. In the 461 procedures reported from the Surgical Section, six different types of anastomotic procedures were used: end-to-end anastomosis, 217; end-to-side anastomosis, 45; side-to-side anastomosis, 97; Mikulicz, 66; Bishop-Koop, 18; Santulli, 18.[4,6] Similarly, there was no consensus as to the advisability of resecting the dilated proximal segment. Resection of this segment was carried out in two-thirds of these patients. Such a variety of procedures suggests dissatisfaction with any one approach and that the ability to construct a functioning anastomosis continues to play a prominent and critical role in the morbidity and mortality of these patients.

The purpose of this paper is to review our experience with 34 neonates who underwent surgical correction of small intestinal atresia at the North Carolina Memorial Hospital from 1952 through 1973. Emphasis will be placed upon factors contributing to morbidity and mor-

Presented at the Annual Meeting of the Southern Surgical Association, December 3–5, 1973, Hot Springs, Virginia.

tality with particular attention being paid to optimum methods of restoring intestinal continuity.

Etiologic Considerations

The etiology of small intestinal atresia varies with its site of origin. Tandler in 1900, while investigating the embryology of the duodenum, postulated intestinal atresia to result from failure of recanalization of the intestinal lumen following obliteration during the stage of epithelial proliferation.[20] This phase of proliferation and recanalization occurs prior to the eleventh week of intrauterine life. Failure of recanalization seems the best explanation for localized duodenal atresia or stenosis. In jejuno-ileal atresia, however, bile, squamous cells and languo, all of which are produced after the stage of epithelial proliferation, may be found distal to the site of obstruction. Furthermore, the segmental absence of the jejuno-ileal mesentery cannot be accounted for on the basis of a failure of recanalization. A more plausible explanation is that jejunoileal atresia is a consequence of an intra-uterine mesenteric vascular accident secondary to volvulus, incarceration in a prenatal umbilical hernia, or intussusception. Louw and Barnard[10,11] experimentally produced the entire spectrum of neonatal intestinal obstruction, ranging from stenosis, diaphragmatic webs, atretic bands to complete segmental absence of intestine and mesentery. They accomplished this dramatic reproduction by ligating the mesenteric vasculature of fetal puppies. The resulting severity of the intestinal atresia was greater the more proximal the mesenteric vascular compromise. Similar observations have been made by Abrams.[1] It is concluded that duodenal atresia and jejuno-ileal atresia represent entities of differing etiologies. Each, however, is manifest by dilation and muscular hypertrophy of the proximal obstructed segment. The extent of these changes depends upon the time of development of the obstruction as well as the site of involvement. Characteristically, atresia of the duodenum and proximal jejunum are associated with more massive dilatation than when the obstruction involves the distal jejunum or ileum. These conceptual differences in etiology influence therapy since vascular injury may be associated with loss of bowel length, foreshortening of the mesentery and multiple sites of involvement; all of which are important considerations in surgical management.

Clinical Presentation and Management

Thirty-four infants were identified as having intestinal obstruction produced by small intestinal atresia. The sites of obstruction are shown in Table 1. Jejunal atresia occurred in 16 of the 34 patients (47%). Eight of the patients (23.5%) exhibited duodenal atresia while ileal obstruction was present in seven (20%) and jejunal webs were present in three (9%). This relative incidence is consistent with the experience of others.[4,6,19]

The clinical presentation of intestinal atresia is dependent upon the site of obstruction. Intestinal obstruction was heralded by bilious vomiting, failure to pass meconium, and abdominal distension. These signs usually occurred within 24 hours after birth. Abdominal roentgenograms demonstrated air containing dilated proximal small bowel. Patients with duodenal atresia were characterized by prematurity, (mean birth weight, 1662 gm), and a higher incidence of associated congenital anomalies. The relative maturity of patients with jejuno-ileal atresia as compared to those with duodenal atresia is indicated by a mean birth weight of 2312 gm for the patients with jejunal atresia and 3052 gm for those with ileal atresia. Lower intestinal atresia may not exhibit signs of obstruction until after 24 hours. A lesion at this site needs to be differentiated from the functional obstruction of Hirschsprung's Disease or meconium ileus by contrast studies of the colon which will usually show in both atresia and meconium ileus an unused or "micro" colon. Polyhydramnios (amniotic fluid > 2500 ml) occurs in 25 to 30% of mothers of neonates with proximal intestinal atresia. This finding is not specific, however, since other factors such as renal tubular and pituitary function also play a role in the homeostatic mechanisms maintaining an amniotic fluid volume of approximately one liter. Prematurity is also a characteristic of neonates with proximal intestinal atresia with 21 of these 34 patients having a birth weight of less than 2500 gm. This in turn may be related to the high incidence of hydramnios and early membrane rupture.

Surgical reconstructive procedures consisted of side-

TABLE 1. *Sites of Obstruction, Birth Weights and Deaths in 34 Infants with Intestinal Atresia*

Type	Patients No.	Birth Weights Grams	Mean	Deaths	Mortality (Per cent)
Duodenal Atresia	8	970–2278	1662	5	62
Jejunal Atresia	16	1375–3790	2312	7	43
Jejunal Webs	3	1365–2274	1819	0	0
Ileal Atresia	7	2500–3912	3052	2	29
Total	34			14	41

TABLE 2. *Causes of Death Following Operative Procedures for Congenital Small Intestinal Atresia*

Causes	Number of Patients	Contributing Factors
Sepsis	3	Gastric perforation (2), staphylococcal septicemia
Malabsorption	2	Duodenocolostomy, jejuno-colostomy
Anastomotic Complications	6	S-S Duodenojejunostomy—malfunction, S-S Jejunojejunostomy—dehiscence, S-S Jejunojejunostomy—malfunction—reresection (2), E-S Ileocolostomy—malfunction—ileocolitis, E-E Jejunocolostomy—necrosis at anastomosis
Cardio-respiratory	2	Intraoperative cardiac arrests Progessive apneic episodes
Pneumonia	1	Repair of tracheo-esophageal fistula

to-side,[12] end-to-side,[4] or end-to-end anastomoses[15] (Table 3). Seven patients with duodenal atresia underwent anastomosis of the dilated end of the duodenum to the subjacent duodenum or proximal jejunum and these were considered functionally as "side-to-side" in construction. Side-to-side anastomoses for jejuno-ileal atresia were utilized early in our experience and have not been employed in the past 10 years. End-to-side anastomoses were usually fashioned in an "end-to-back" manner. End-to-end anastomoses were either direct or as facilitated by a jejunoplasty (Fig. 1). Reconstruction utilizing the methods described by Mikulicz, Bishop-Koop or Santulli were not employed. In view of the incidence of multiple sites of obstruction, patency of the distal bowel was demonstrated by inserting a small intraluminal catheter and slowly instilling 0.9% saline. Jejunal webs were corrected by a transverse incision immediately proximal to the web. By means of an incision distally to form a "T", the web was excised and the "T" site of enterotomy closed in a transverse manner. All excised segments of bowel were examined for presence of ganglion cells. In the majority of patients a temporary gastrostomy was fashioned for postoperative decompression[21] and in a few patients with proximal anastomoses, a transanastomotic feeding catheter was placed parallel to the gastrostomy tube.

Pathophysiologic Considerations

There is consensus that malfunction is likely to result after performing an end-to-end or end-to-side anastomo-

TABLE 3. *Sites and Results of Reconstructive Anastomoses for Intestinal Atresia*

Type of Anastomosis and No.		No. Anastomoses Permitting Evaluation	Dehiscence or Necrosis	Anatomic or Functional Obstruction	Malfunction (Stenosis, Blind loop, Delay)	Functional
Side-to-Side						
Duodenal Atresia	7	4	0	1§	0	2
Jejunal Atresia	4	4	1§	2§	1*	0
Ileal Atresia	1	1	0	0	0	1
	12	9	1	3	1	3
End-to-Side						
Jejunal Atresia	1	0	0	0	0	0
Ileal Atresia	3	3	0	1§	0	2
	4	3	0	1	0	2
End-to-End						
Jejunal Atresia	6	6	0	0	0	6
Ileal Atresia	2	2	0	0	0	2
	8	8	0	0	0	8
End-to-End with Jejunoplasty‡						
Jejunal Atresia	6	6	0	0	1	5
Ileal Atresia	1	1	1§	0	0	0
	7	7	1	0	1	5
Summary						
End-to-End	15	15	1	0	1	13
End-to-Side	4	3	0	1	0	2
Side-to-Side	12	9	1	3	1	3
	31	27	2	4	2	18

* Blind Pouch Syndrome.
† Stenosis.
‡ One patient with jejunoplasty represents reoperation for an obstructed side-to-side anastomosis.
§ Cause of death.

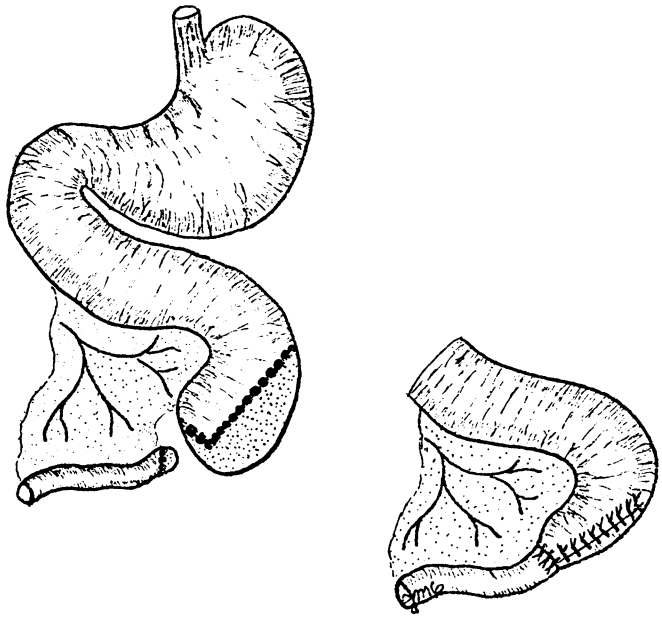

FIG. 1. Technique of jejunoplasty and end-to-end anastomosis for proximal jejunal atresia.

sis between a dilated proximal segment and a more diminutive distal segment. This obstruction occurs despite anatomic patency of the stoma and seems to be related to a lack of normal peristaltic mechanisms. In man intraluminal contents are propelled in an aboral direction as a result of pressure gradients secondary to rhythmic segmental contractions. Muscular contraction of the intestinal tract is highly dependent upon stretch receptors located within the mucosa, the intrinsic nerve supply, smooth muscle of the intestine and enterochromaffin cells. Distention or increased intraluminal pressure not only stimulates the stretch receptors, thereby initiating the peristaltic reflex, but also causes serotonin to be released by the enterochromaffin cells, thereby sensitizing the stretch receptors to respond at lower intraluminal pressures. The decrease in intraluminal pressure of the proximal segment consequent to operative decompression and anastomosis may result in an atonic and non-propulsive segment of gut. The loss of efficient propulsion in such a dilated segment has been nicely documented by Nixon,[14] who by means of a gut pump demonstrated the peristaltic activity of a dilated segment of the small bowel to be relatively inefficient except with high intraluminal pressures. Such concepts would seem to provide a better explanation of a malfunctioning anastomosis seen in these circumstances than that of postoperative ileus, the diminutive size of the small bowel, plugging of the anastomosis, adhesions, or kinking at the anastomotic site. Nixon's recommendation for resection of the dilated segment overcomes this problem in part. It can usually be accomplished with atresia of the distal jejunum or ileum. However, in proximal jejunal atresia the dilated segment commonly extends beyond the ligament of Treitz to the pylorus. Resection to correct the discrepancy in the calibre of the bowel may be anatomically impossible or result in too great a reduction of the absorptive surface of a foreshortened small bowel. These technical and physiological constraints on resection of the dilated jejunal segments prompted the design of a jejunoplasty to permit reconstruction utilizing a graduated reduction of the dilated jejunal segment[22] (Fig. 1). This technique: (a) facilitates an end-to-end anastomosis between disproportionate segments of the bowel; (b) conserves absorptive surfaces and (c) reduces intraluminal diameter, enhancing earlier return of effective peristalsis. All anastomoses are constructed with a single layer of interrupted silk sutures of 5–0 or 6–0 silk. Depending upon the size of the distal segment, and end-to-end or end-to-oblique anastomosis is constructed, usually sectioning the distal bowel at an angle of 45°. The anastomosis may be constructed over a small transanastomotic silastic catheter placed alongside a temporary gastrostomy tube. This feeding tube permits maintenance of nutrition while gastrostomy insures proximal decompression until the proximal bowel is functional. In those circumstances in which bowel length is adequate and the obstruction is more distal, the dilated segment may be excised as recommended by Nixon and a direct end-to-end anastomosis established.

In view of the limited clinical experience and lack of opportunity for controlled observations, experimental studies were carried out in rats, utilizing a model of intestinal atresia.[9] Self-filling intestinal cul-de-sacs were found to simulate the dilatation and hypertrophy of the bowel proximal to an atretic segment in the newborn. Various types of reconstructive procedures (side-to-side, end-to-side and jejunoplasty with end-to-end anastomoses) were compared. The jejunoplasty and end-to-end anastomosis appeared to be superior in that stasis was less pronounced in this group and the weight gain of these animals was greater than in the animals having side-to-side and end-to-side anastomoses. These findings were also consistent with those of Nygaard, who studied the intestinal flora of rats with different types of anastomses.[17] There was increased flora in all but straight end-to-end junctions. He emphasized that interruption of the circular muscle of the intestine led to some degree of stasis and therefore a change in bacterial flora.

Results

There were five deaths among the eight patients with duodenal atresia (Tables 1, 2). One death was due to a widely patent, but malfunctioning duodenojejunostomy, a second to peritonitis due to antecedent gastric perforation, a third associated with a postoperative cardiac

arrest in a 1700 gm patient with a cardiac anomaly (common atrium), a fourth associated with apnea and immaturity in a 970 gm female, and the fifth due to a gastric perforation following repair of a tracheoesophageal fistula and an unrecognized duodenal web. Three patients survived, a duodenoduodenostomy being performed in one and a duodenojejunostomy in two. Five, eight and thirty days were required for these anastomoses to function. The propensity for malfunction in this type of reconstruction in which there is an anastomosis of a proximal dilated segment to a more diminutive segment is well illustrated in the delayed function in two patients and the complete functional obstruction in one resulting in death (c. 1965). The current practice of maintaining nutrition by parenteral alimentation or transanastomotic feeding should minimize this particular problem in the future.

In the 26 patients with jejuno-ileal obstruction, there were nine deaths (Tables 1, 2). Five of these were directly attributable to anastomotic complications. Two deaths were associated with obstruction accompanying a side-to-side anastomosis requiring reoperation and end-to-end reconstruction in patients already at a marginal nutritional status because of their initial problem. One of these patients expired in the immediate postoperative period and the second from malabsorption. Two deaths were associated with dehiscence or necrosis at the site of anastomosis and peritonitis. One of these patients had gangrenous bowel and peritonitis at the time of his initial operation. One death was secondary to enterocolitis and peritonitis after an apparent satisfactory, but malfunctioning end-to-side ileo-colostomy. Of the remaining four deaths, one was due to pneumonia in a patient with an associated tracheoesophageal fistula, one due to massive staphylococcal sepsis at 48 hours postoperatively, and two were related to malabsorption following extensive excision of the mid gut at the initial operative procedure. One of the latter required a duodenocolostomy. The patients who expired from malabsorption did so prior to the present methods of maintaining nutrition and one of these may have survived had parenteral alimentation and/or elemental diets been available.

In this series the overall survivorship was 59% which is comparable to the reports from the Surgical Section of the American Academy of Pediatrics for the 10-year period 1957 through 1966. Table 2 categorizes the causes of the 14 deaths in this group of patients. Sepsis was the leading cause in three patients with peritonitis as a result of intestinal perforation being an antecedent event in two of these. Two patients died of malabsorption. Two deaths were related to cardiorespiratory complications. Pneumonia was the cause of death in one patient following repair of an associated tracheo-esophageal fistula. Anastomotic complications were the cause of six (44%) of the 14 deaths.

The higher mortality associated with prematurity, low birth weight and associated anomalies is documented in this study. Low birth weight (less than 2500 gm) was present in 20 patients (60%) and 12 of these patients expired. Associated significant congenital anomalies were present in 10 patients with seven deaths. Six of these seven patients were also premature.

These findings in general correspond with Nixon's rating of risk, namely the highest in those patients weighing less than 1800 gm or with another anomaly endangering life, next in those between 1800 and 2500 gm and another moderate anomaly and the least in patients weighing more than 2500 gm with no other significant anomaly.[16]

Two patients illustrate some of the complexities that are associated with these atresias and demonstrate the role of vascular occlusion in the development of intestinal atresia.

Case Reports

No. 1. An 1824 gm black male was the product of an uncomplicated 30-week gestation and normal delivery. At 24 hours of age, he experienced several episodes of bilious vomiting. There had been no passage of meconium. Upon admission to North Carolina Memorial Hospital at 40 hours of age, examination revealed a dehydrated premature infant male. The abdomen was distended and tympanitic. No abdominal masses were palpable. Rectal examination showed the absence of meconium. Abdominal roentgenograms demonstrated a "triple bubble" air shadow. The preoperative impression was small intestinal obstruction produced by proximal jejunal atresia.

Following rehydration, celiotomy disclosed a markedly distended proximal segment of jejunum that ended abruptly 5.0 cm distal to the ligament of Treitz. The remaining 20-25 cm of small intestine was coiled, as an "apple peel," around a narrow mesentery attached at the ileocecal junction. This portion of small intestine derived its blood supply from the ileocolic artery (Fig. 2). No other intra-abdominal anomalies were demonstrated. A decompressive gastrostomy, a jejunoplasty and an end-to-end jejunojejunostomy were utilized in reconstituting intestinal continuity.

Postoperatively, intravenous hyperalimentation was utilized to maintain nutrition. Frequent episodes of vomiting persisted. Gas-

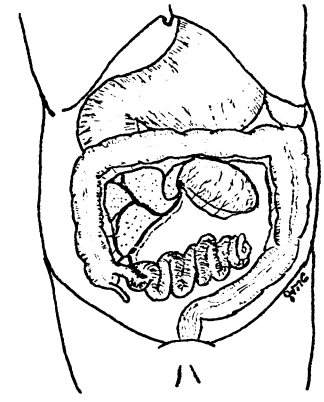

FIG. 2. "Apple peel" deformity in intestinal atresia.

trointestinal contrast studies showed distension of the duodenum, but patency of the anastomosis. Because of continued partial obstruction, reoperation was carried out at six weeks of age disclosing a stenotic anastomosis. This was revised by an end-to-end jejunojejunostomy. Thereafter, the patient progressed well and was discharged at 10 weeks of age, weighing 2,520 gm. Followup examination at six months showed the patient to be developing satisfactorily.

The anatomical findings in this patient are characteristic of the "Christmas tree" or "apple peel" deformity. This patient represents the twentieth case report of such a deformity. In previous patients with this anomaly, the etiology has been demonstrated to be secondary to occlusion of the superior mesenteric artery immediately distal to the first few jejunal branches.[3,8,12,23]

No. 2. A two-day-old 2270 gm Caucasian female was admitted to the North Carolina Memorial Hospital with a history of vomiting following all feedings of dextrose and water. She had not passed a meconium stool. The mother had polyhydramnios. Examination revealed a moderately dehydrated infant weighing 1915 gm. The abdomen was not distended and rectal examination showed the absence of meconium. Abdominal roentgenograms revealed complete obstruction of the proximal small intestine.

Celiotomy disclosed a markedly dilated proximal jejunum that ended blindly with attachment of multiple atretic "sausage-like" segments of small intestine by cord-like remnants of mesentery. Complete absence of a segment of small intestine and mesentery was also present (Fig. 3). A tapering jejunoplasty of the dilated proximal segment of jejunum, resection of the multiple atretic segments and an end-to-end jejunojejunostomy was performed. The infant's postoperative course was uneventful. Following discharge from the hospital, she progressed well and normal growth and development were noted on subsequent examinations.

Discussion

Multivariate factors contribute to the morbidity and mortality in patients with intestinal atresia. Prematurity, associated congenital anomalies, delay in diagnosis, intercurrent infection, and malfunctioning anastomoses are most prominent and frequently interrelated.

Anastomotic complications deserve a special point of emphasis since construction of the anastomosis is the one factor over which the surgeon has the most control. Six deaths were attributed directly to anastomotic failure (Table 2). In addition, one patient with an end-to-side jejunostomy developed a massive blind pouch requiring resection and an end-to-anastomosis. There was one instance of stenosis at the site of a jejunoplasty and end-to-end jejunojejunostomy.

The type of anastomosis performed was a factor in the incidence of anastomotic complications (Table 3). A side-to-side or end-to-side anastomosis was associated with the highest incidence of malfunction. Five anastomotic complications developed in the nine patients having side-to-side anastomoses that could be evaluated (one with functional obstruction causing death, two with obstruction requiring revision of anastomoses with death, one with dehiscence and one with a blind pouch). One complication occurred in the three patients having an end-to-side anastomosis (malfunction-ileocolitis). Two complications (one stenosis, and one dehiscence) developed in 15 patients having end-to-end anastomoses.

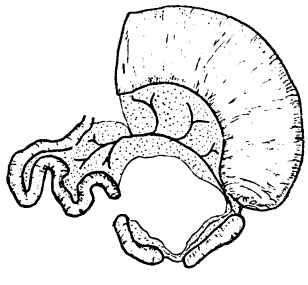

FIG. 3. Multiple atresias in patient with proximal jejunal atresia.

The functional status of the anastomosis was evaluated in those patients whose duration of survival permitted such an appraisal (Table 3). In 15 end-to-end anastomoses, there was one instance of necrosis (considered a technical error) and one of stenosis requiring revision. Seven of these 15 patients with proximal jejunal atresia required an associated jejunoplasty. Of four patients undergoing an end-to-side anastomosis, three could be evaluated, with two anastomoses being patent. One had a functional obstruction with associated ileo-colitis. Of 12 side-to-side anastomoses, nine could be evaluated; only three were completely satisfactory.

Evans[5] in his report in 1951, cited the major causes of death in unsuccessfully treated patients as: (a) nutritional disturbances plus dehydration shock and (b) either localized peritonitis which interfered with anastomotic function, or generalized peritonitis. Both of the latter were considered due to gross contamination or leakage at the site of the anastomosis. The overall survival was 9%. Non-functioning anastomoses contributed significantly to the cause of death in these patients. That this continues to be a factor in morbidity and mortality is well documented in the current literature. In 1961 Santulli and Blanck[18] cited a mortality of 80% prior to 1948, and an overall mortality of 62% for atresia of colon and small bowel. There was a 21% mortality during the two years prior to the report. Anesthetic and operative shock accounted for most of the early postoperative deaths, and malfunction of the anastomosis was directly or indirectly responsible for late deaths. Entities such as dehydration, electrolyte imbalance, aspiration of vomitus, peritonitis, infection, malnutrition and post-operative adhesions were in many instances related to the lack of a functioning anastomosis. These authors favored a side-to-end anastomosis with an associated proximal end type enterostomy. A catheter was threaded through the anastomosis into the distal bowel and a delayed closure of the enterostomy performed later. In the compilation of 587 patients from the review by the Surgical Section of the American Academy of Pediatrics,[4,6] the most common cause of death (overall mortality 43.1%) was infection due to pneumonia, peritonitis or sepsis. An anastomotic complication was the second most frequent cause of death, being responsible for 24%. Functional obstruction at the

site of anastomosis was the most common primary cause of death in infants with jejunal atresia and the third most frequent cause in ileal atresia. It was apparent from this survey that functional obstruction still presented a problem even when a significant portion of the hypertrophied dilated bowel was resected.

Nixon and Tawes,[15] in reporting their experience with 67 good risk patients, emphasized the higher mortality (40%) associated with proximal jejunal obstruction as compared to 18% mortality with mid-intestinal lesions and 0% in the terminal ileum. The 37 poor risk patients had an overall mortality of 68%. The commonest primary cause of death was anastomotic leak or dysfunction. Again, functional obstruction was a prominent cause of mortality, but was almost always confined to the poor risk group. Nixon's experimental and clinical observations favored elective resection of the dilated bowel on the basis of the increased diameter relative to the dilatation and muscular hypertrophy resulting in ineffective peristalsis at the lower pressures existant after operation. Santulli's more recent experience (1970) has been unique in that "anastomotic obstruction and leakage . . . have not been a problem in the past decade due to more adequate resection of the blind ends and to utilization of the proximal chimney." The major complications leading to death were respiratory problems and sepsis.[19]

Superior results have recently been reported from the Johns Hopkins Hospital.[24] There was an adjusted survival rate of 90% in 13 patients with duodenal atresia and 77% survivorship in 25 patients with jejunoileal atresia, for an overall survivorship of 80%. Causes of death were septicemia (3); overtransfusion (1); and intestinal obstruction (3). Of the latter three, one was due to a side-to-side anastomosis, one to an unrecognized malrotation with volvulus and the third due to duodenal bands. The authors emphasized meticulous operative and postoperative care as well as a preference for an end-to-end anastomosis for jejuno-ileal atresia.

During the past two decades, as sepsis and other complicating factors have been better controlled, the role of the anastomosis has become increasingly critical. In patients with duodenal atresia there seems little alternative to some type of lateral anastomosis between the dilated duodenum and the adjacent duodenum or jejunum. Jejuno-ileal atresia, however, presents more alternatives. The current approach to intestinal reconstruction in jejuno-ileal atresia has been evolutionary. Evans, in culling 149 successful cases in 1951 from the surgical literature, observed that the majority of patients underwent a side-to-side or lateral anastomosis. Only five patients could be identified who had resection and a direct end-to-end anastomosis. Santulli and Blanck,[18] as well as Bishop and Koop[4] advised a venting enterostomy with delayed closure of the vent because of problems with an immediate anastomosis. Nixon's contribution was significant indicating the insufficiency of peristalsis in the dilated segment.[14] Since that time, the trend has been for the immediate establishment of an end-to-end or end-to-oblique anastomosis.[2,10,15,24]

The superiority of an end-to-end anastomosis in small intestinal atresia has been suggested by others and seems confirmed by this experience. This is more readily achieved the more distal the obstruction in the gastrointestinal tract. An end-to-end anastomosis can be constructed in low ileal atresia with resection of the distal segment being associated with less disparity between the ends of the bowel and with the proximal segment retaining effective peristalsis. With proximal jejunal atresia or with multiple atretic segments a direct end-to-end anastomosis cannot always be achieved either because of the extent of the proximal dilatation or because excision of the dilated bowel would compromise the amount of absorptive surface resulting in malabsorption. Even an "end-to-back" anastomosis as proposed by Nixon under these circumstances may be associated with functional obstruction as well as a blind pouch or diverticulum formation.[13] It is in this latter group of patients that a jejunoplasty and end-to-end anastomosis would seem particularly applicable. The resulting smaller calibre of the bowel seems to enhance or restore normal peristalic mechanisms resulting in earlier function of the anastomosis. This approach has been operative during the past 10 years. There have been 15 survivors in 21 patients during the period whereas during the first 10 years of this report only 5 of 13 patients survived. Howard and Otherson recently confirmed our experience in reporting three patients in whom jejunoplasty and end-to-end jejunojejunostomy was carried out for proximal jejunal atresia. These anastomoses were functional within twelve days.[7]

Acknowledgment

The critical participation of the Staff of the Department of Pediatrics and the Nursing Staff of the Premature Nursery of the North Carolina Memorial Hospital is clearly recognized.

References

1. Abrams, J. S.: Experimental Intestinal Atresia. Surgery, **64**: 185, 1968.
2. Benson, C. D., Lloyd, J. R. and Smith, J. D.: Resection and Primary Anastomosis in the Management of Stenosis and Atresia of the Jejunum and Ileum. Pediatrics, **26**:265, 1960.
3. Blyth, H. and Dickson, J. A. S.: Apple Peel Syndrome (Congenital Intestinal Atresia). J. Med. Genet., **6**:275, 1969.
4. deLorimier, A. A., Fonkalsrud, E. W. and Hays, D. M.: Congenital Atresia and Stenosis of the Jejunum and Ileum. Surgery, **65**:819, 1969.
5. Evans, C. H.: Atresia of the Gastrointestinal Tract. Collective Review. Int. Abstr. Surg., **92**:1, 1951.
6. Fonkalsrud, E. W., deLorimier, A. A. and Hays, D. M.: Congenital Atresia and Stenosis of the Duodenum. Pediatrics, **43**:79, 1969.
7. Howard, E. R. and Otherson, H. B., Jr.: Proximal Jejunoplasty in the Treatment of Jejunal Atresia. Presented at Surgical Section of American Association of Pediatrics. May, 1973.

8. Jiminez, F. A. and Reiner, L.: Angiographic Findings in Congenital Abnormalities of the Mesentery and Intestines. Surg. Gynecol. Obstet., **113**:346, 1961.
9. Kroop, H. and Thomas, C. G. Jr.: An Experimental Model for Intestinal Atresia. Am. Surg., **38**:676, 1972.
10. Louw, J. A.: Congenital Intestinal Atresia and Stenosis in the Newborn. Observations on its Pathogenesis and Treatment. Ann. Roy. Coll. Surg., **25**:269, 1959.
11. Louw, J. A. and Barnard, C. N.: Congenital Intestinal Atresia: Observations on its Origin. Lancet, **2**:1065, 1955.
12. Mishalan, H. G. and Najjar, F. B.: Familial Jejunal Atresia. J. Pediatr., **73**:753, 1968.
13. Nade, S. M. L. and Dickson, J. A. S.: Diverticulum Formation after Oblique End-to-end Anastomosis for Jejuno-ileal Atresia. Br. J. Surg., **57**:540, 1970.
14. Nixon, H. H.: An Experimental Study of Propulsion in Isolated Small Intestine, and Applications to Surgery in the Newborn. Ann. Roy. Coll. Surg., **27**:105, 1960.
15. Nixon, H. H. and Tawes, R.: Etiology and Treatment of Small Intestinal Atresia; Analysis of a Series of 127 Jejuno-ileal Atresias and Comparison with 62 Duodenal Atresias. Surgery, **69**:41, 1971.
16. Nixon, H. H.: Small Intestinal Atresia. Proc. Soc. Med., **64**: 372, 1971.
17. Nygaard, K.: Changes in the Intestinal Flora after Resections and Bypass—Operations on the Small Intestine in Rats. Acta. Chir. Scand., **133**:569, 1967.
18. Santulli, T. V. and Blanck, W. A.: Congenital Atresia of the Intestine: Pathogenesis and Treatment. Ann. Surg., **154**: 939, 1961.
19. Santulli, T. V., Chen, C. and Schullinger, J. N.: Management of Congenital Atresia of the Intestine. Am. J. Surg., **119**: 542, 1970.
20. Tandler, J.: Zur Entwishlungsgeschicte des menschlichen duodenum in fruhen embryonal studien. Gegenbaurs Morph. Jahrb., **29**:187, 1900.
21. Thomas, C. G., Jr. and Croom, R. D. III: A Method Temporary Gastroma. Surg. Gynecol. Obstet., **131**:292, 1970.
22. Thomas, C. G., Jr.: Jejunoplasty for the Correction of Jejunal Atresia. Surg. Gynecol. Obstet., **129**:545, 1969.
23. Weitzman, J. J. and Vanderhoof, R. A.: Jejunal Atresia with Agenesis of the Dorsal Mesentery with "Christmas Tree" Deformity of the Small Intestine. Am. J. Surg., **3**:443–449, 1966.
24. White, J. J., Tecklenberg, P. L., Esterling, J. R. and Haller, J. A.: Changing Concepts in the Management of Intestinal Atresia. Surg. Clin. N. Am., **50**:863, 1970.

Discussion

Dr. Richard C. Miller (Jackson): In our institution we have learned to respect a unit called the Neonatal Intensive Care Unit, and a pediatrician who is a full-time neonatologist. We believe that these people probably have contributed as much to the survival of surgical neonates in the last few years as the surgeons themselves, and we rely heavily on them. This is particularly true in those babies who have a very low birth weight, four pounds and under.

In regard to the anastomotic problem, we would concur completely that the end-to-end anastomosis is the one to do. The problem with the end-to-side anastomosis, is that the runoff is determined not by the size of the anastomosis, but by the size of the distal bowel and by the propulsive force through the proximal dilated segment. When you can resect the proximal dilated segment back to normal-sized bowel, as is possible in the ileum or distal jejunum, the anastomosis then stands the best change of functioning. The real problem, as Dr. Thomas has pointed out, arises in the high jejunal atresia, where the proximal pouch is very short at the ligament of Treitz, or just beyond it, and you can't get back to good bowel because you are right against the pancreas and the bile ducts.

However, we would always prefer the end-to-end anastomosis, and would not even do the end-to-oblique or the end-to-backside, which is simply a compromise between the side-to-side and to end-to-end anastomosis.

It is our experience that we have always been able to do an end-to-end anastomosis, even when our surgical residents point out that we have a "toothpick-size" distal end and a "telephone pole size" proximal end. In this slide (slide) one can see the disparity of size between the small distal bowel and the very large caliber bowel proximally in this case of high jejunal atresia. We have cut it back right almost up to the bile ducts, and a single layer anastomosis with interrupted 5.0 silk, with the knots on the inside is accomplished.

(Slide) We'll just show you one more illustrative case. An end-to-end anastomosis is accomplished by apportioning the sutures even when there is a discrepancy in proximal and distal end diameter of 5 to 1.

Dr. Joey M. Carter (Closing discussion): Dr. Miller, I appreciate your remarks, and the only comment I have is that perhaps you should have done a jejunoplasty in addition to the end-to-end anastomosis. You wouldn't have had the problem of anastomosing a telephone pole to a toothpick. It would have been much easier.

I should emphasize the critical point of this paper once again, and that is to provide a functioning anastomosis in this particular congenital anomaly. Without it, of course, the infant is going to have a lot of complications.

We should also point out that the improvement in the survival of these patients over the past ten years, as compared to the previous ten years, in striking. During the past ten years we had 15 of 21 patients survive, and this represents a 71% survival rate. During the previous ten years only five of thirteen patients survived. This represented a 38% survival rate which was quite poor. We feel that by utilizing an end-to-end anastomosis our mortality and morbidity rates were greatly improved.

I have two other points to make. The first is that during the 15 end-to-end anastomoses we had one death as a result of necrosis and dehiscence of the anastomosis. The other problem we encountered was that of an anastomotic stenosis. The following two slides show this problem.

This complication occurred in a 1800 gram male infant presented to us 24 hours after birth. He was of a 30 weeks' gestation. He exhibited bilious vomiting and abdominal distention. An abdominal roentgenogram revealed a triple-bubble sign. Our preoperative impression was that of jejunal atresia.

The interesting point about this patient is that he represents the 20th case report of an "Apple-peel or Christmas tree deformity." This anomaly is represented by the short, coiled distal ileum, which measured approximately 20 to 25 cm in length. As you note, it derived its blood supply directly from the ileocolic artery. There is marked foreshortening of the mesentery and marked shortening of the entire length of the bowel. Also there is a very dilated proximal portion of jejunum which measured approximately 5 to 10 cm in length.

We performed a tapering jejunoplasty and an end-to-end anastomosis, as described by Dr. Thomas. However, this patient had difficulties postoperatively. He failed to gain weight and showed evidence of continued partial intestinal obstruction. At six weeks of age it was necessary to revise the end-to-end anastomosis.

The next slide shows the operative findings of the marked coiling of the distal ileum. We simply took the anastomosis down and performed another end-to-end anastomosis. Following this procedure, at 10 weeks of age, the child was discharged from the hospital weighing 2500 gm. At six months' followup, the child continued to gain weight and was in good health.

In closing, I would recommend to you that we think the optimum method of attacking this problem is by an end-to-end anastomosis utilizing the tapering jejunoplasty.