Congenital Atresia of the Intestine: *

Pathogenesis and Treatment

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THIS REPORT is not intended to be a comprehensive discussion of the subject of congenital atresia of the intestine nor is any attempt made to review the voluminous literature on the subject. Rather, it deals with the pathogenesis of this lesion, considers some of the general principles of management including specific reference to one of the controversial technical aspects of the problem and presents a preliminary report on a method of treatment. A discussion of the clinical picture and other aspects of the problem may be found in some of our previous communications.[26, 27]

Pathogenesis

Congenital atresia of the intestine is generally considered to be an embryonal malformation due to the lack of recanalization of the intestinal tube at the end of the second month of fetal life. However, there is evidence which casts serious doubt on the validity of this theory as the pathogenesis of atresia of the jejunum and ileum. The theory is not compatible with several observations which have been made in the Operating Room and in the Pathologic Laboratory in many of these cases as we have indicated in previous reports.[7, 8]

For many years we were puzzled by the short length of the small intestine in some cases of atresia. The total length of the jejunum and ileum as determined by accurate measurements of resected specimens and of the remaining intestine at necropsy was sometimes a great deal less than the intestine of comparably normal newborn infants. Benson and his associates [5] have recently reported on this, citing cases of jejunal or ileal atresia in which the total length of small intestines was far less than the average length of 248 cm. for normal, full term, newborn infants or 160 to 240 cm. for the premature newborn infant. We had previously attributed this discrepancy in length to "hypoplasia" or to "atrophy of disuse" but these explanations would appear to be unsatisfactory.

We were perplexed by several surgical specimens of single or of multiple atresia of the jejunum or ileum in which gross meconium or microscopic squamous epithelial cells, lanugo hair or bile droplets were found distal to the points of atresia and even between segments of atresia (Fig. 1). This would not be expected if atresia resulted from the lack of recanalization of the intestinal tube at the end of the second month of fetal life. Since excretion of bile begins about the eleventh week and squamous epithelial cells swallowed from the vernix caseosa are found in the intestine after the twelfth week, the presence of these elements in the intestinal lumen distal to any level of congenital atresia would indicate that occlusion occurred sometime after the eleventh or twelfth week of fetal life, well beyond the period of development of the intestinal tube.

The morphology of the mesenteric defects which were often seen in association

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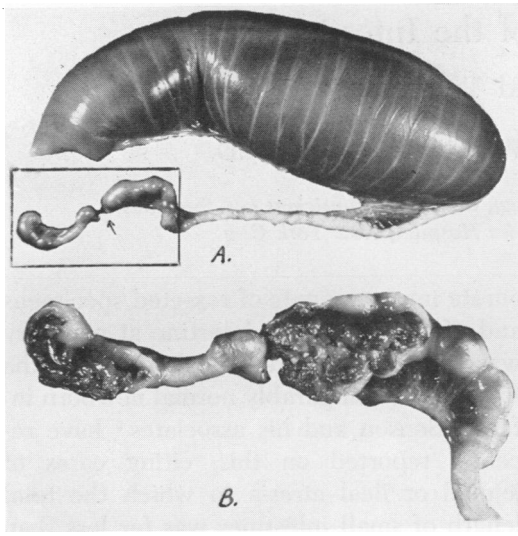

FIG. 1A. Specimen of ileal atresia. Note the greatly distended proximal bowel and the small distal intestine with a blind end and a short cord-like segment (arrow). The inset is enlarged in B to show gross meconium in the lumen of the bowel distal to the atresia.

with some of the atresias was not readily explained by the theory and indicated some type of interruption to a previously intact bowel and mesentery. The gross or microscopic findings in many of our carefully studied specimens were not readily explained by the theory. They appear to represent phases in the process of injury and repair to the intestine.

Such observations do not support the generally accepted theory of lack of recanalization as the cause of all cases of congenital atresia of the intestinal tract. Instead, they suggest that for the jejunum and ileum this lesion may be the result of an injury or accident to the fetal intestine and that it is probably not an embryonal malformation.

Historical Review

In 1900, Tandler [29] explained duodenal atresia by the failure of recanalization of the solid epithelial "plug stage" at the end of the second month of embryologic development. In 1910, Johnson [18] observed that the esophagus is, at first, a simple epithelial tube, the walls of which contain three or four rows of nuclei. In the 20-mm. human embryo, vacuoles form in the epithelium but the lumen remains patent throughout. These vacuoles then disappear by breaking into the lumen causing the epithelium to become thinner and the lumen increases in size. In the small intestine, vacuoles appear in the epithelium of the duodenum from the 10 to 24-mm. stage. Later these vacuoles lead to a complete occlusion of the duodenal lumen. However, he did not observe this occlusive stage anywhere else in the small intestine except, perhaps, at the ileocecal junction. Despite these early reported observations that occlusion (epithelial "plug stage") and recanalization occurred only in the duodenum and, possibly, at the ileocecal junction, the theory has been generally applied to the whole small intestine.[1,17,25]

Several authors, however, have questioned this explanation and have suggested that other mechanisms will explain the occurrence of atresia in the jejunum and ileum. As early as 1904, Clogg, in reporting two cases of congenital intestinal atresia, stated that there was no common cause for the lesion and postulated that torsion of the intestine or snaring of the intestine by the umbilical ring could account for some of the cases.[10] He cited Chiari's report of a case, in 1883, in which intussusception of the fetal intestine was thought to be the cause.[9] In 1922, Davis and Poynter [11] demonstrated the presence of normal meconium in the bowel distal to the atresia in some cases and commented that the obstruction had to be formed after the third month. They listed volvulus, intussusception, strangulation of the intestine at the navel, fetal peritonitis and primary lesions of the mesenteric arteries as possible causes of congenital atresia. More recently a good deal has been written which further disputes the theory of recanalization and evidence has been presented to support the hypothesis of a fetal accident

as the cause of this lesion in the jejunoileum.[5, 7, 8, 13, 15, 19-24]

In 1956, Barnard showed conclusively that intestinal atresia can be caused by accidents to a previously normally developed fetal intestine.[9] In a series of difficult but well designed experiments in pregnant dogs he was able to produce intestinal atresia by operating on the fetus *in utero*. In some operations the mesenteric vessels to a short segment of small intestine were ligated; in others, the blood supply to a segment was interrupted by creating a strangulation type of obstruction. The mother dog was then allowed to go to term and the puppies which had been operated upon *in utero* were sacrificed and examined in the neonatal period. After many failures he was able to produce atresia of the small bowel in two cases; the blind-end type in one and the diaphragm type in another.[2, 3, 21]

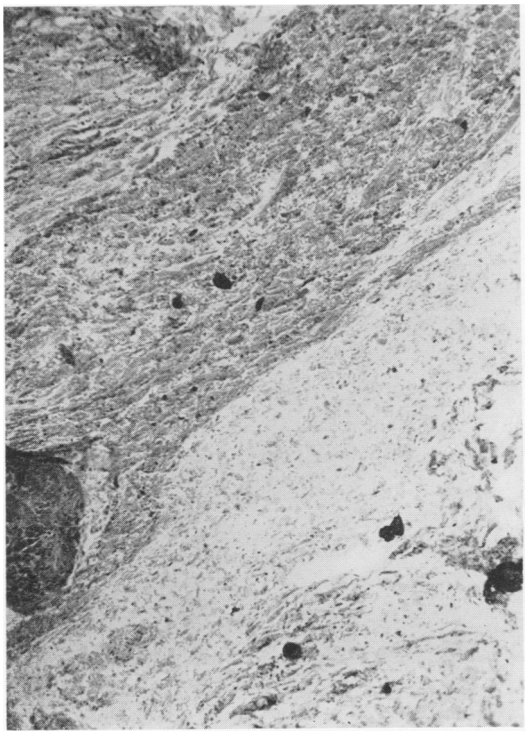

FIG. 2. Photomicrograph of contents of the intestine distal to atresia showing squamous epithelial cells (the darkly stained cells).

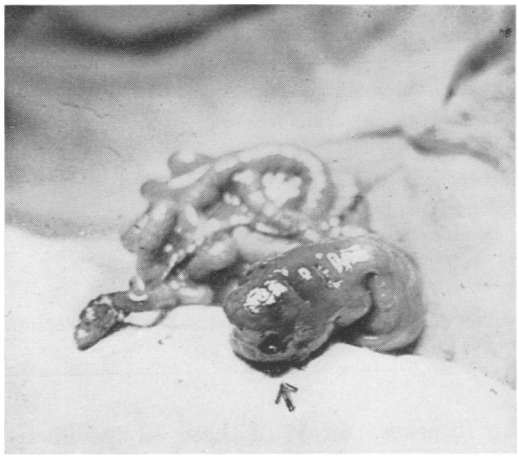

FIG. 3. Necrotic area in bulbous, blind end of jejunal atresia (arrow). Note small and collapsed distal bowel on the left.

Case Material

The previously mentioned observations which we had made in the Operating Room and Pathologic Laboratory up to 1954 stimulated careful study of our subsequent pathologic material and we decided to procure specimens of the bowel or its contents distal to the atresia in our surgical cases whenever possible. Since that time, specimens of the distal bowel or its contents were obtained at the time of operation in 19 cases of jejunal or ileal atresia. In all of these cases squamous epithelial cells, lanugo hair or bile droplets were found in the bowel distal to the atresia (Fig. 2). Other findings included chronic granulomatous lesions in the distal segments, necrotic changes in the bulbous proximal blind segments and elements of meconium on the serosal surface of the bowel or mesentery (Fig. 3).

We then reviewed all of our case material on congenital atresia of the intestine, exclusive of the esophagus and rectum. There were 76 cases, including the above-mentioned 19 (Table 1). During the same period 30 patients with congenital stenosis were seen which were not included in this pathologic study. Surgical or necropsy specimens were available for study in 52 of

TABLE 1. *Summary of Pathologic Study of 76 Cases of Intestinal Atresia*

Surgical or necropsy specimens available		52
Distal segments available	43	
Meconium elements found in	43	
Pathogenesis suggested (reconstructed) in	27	
Prenatal volvulus		
Fetal umbilical hernia		
Omphalomesenteric duct		
Focal perforation of intestine		
(Fibrocystic disease of the pancreas with meconium ileus in 9)		

the 76 cases. In 43 of these 52 specimens of single or multiple atresia sections of the distal lumen were available for study. Squamous epithelial cells, lanugo hair or bile droplets were found in *all* of these sections.

Furthermore, the gross appearance of the intestine *in situ* or after resection suggested a pathogenesis of the atresia in 27 of the 52 specimens which were available for study. Prenatal volvulus sometimes associated with malrotation or anomalous mesentery, fetal umbilical defect, a closed omphalomesenteric duct and focal perforation of the intestine were the responsible mechanisms in the production of the lesions. The pathogenesis could not be reconstructed from the material available in 25, or about half, of the cases. Fibrocystic disease of the pancreas was found in nine cases. This association has been mentioned by others and, recently, Bernstein and his associates presented an excellent pathologic study of four cases of intestinal atresia occurring with meconium ileus.[6]

Other evidence of former injury and the reparative process was found in all cord-like segments of atresia by the presence of granulomatous obstruction of the lumen, intraparietal vernix granulomata, hemosiderosis, phagocytosis, calcification

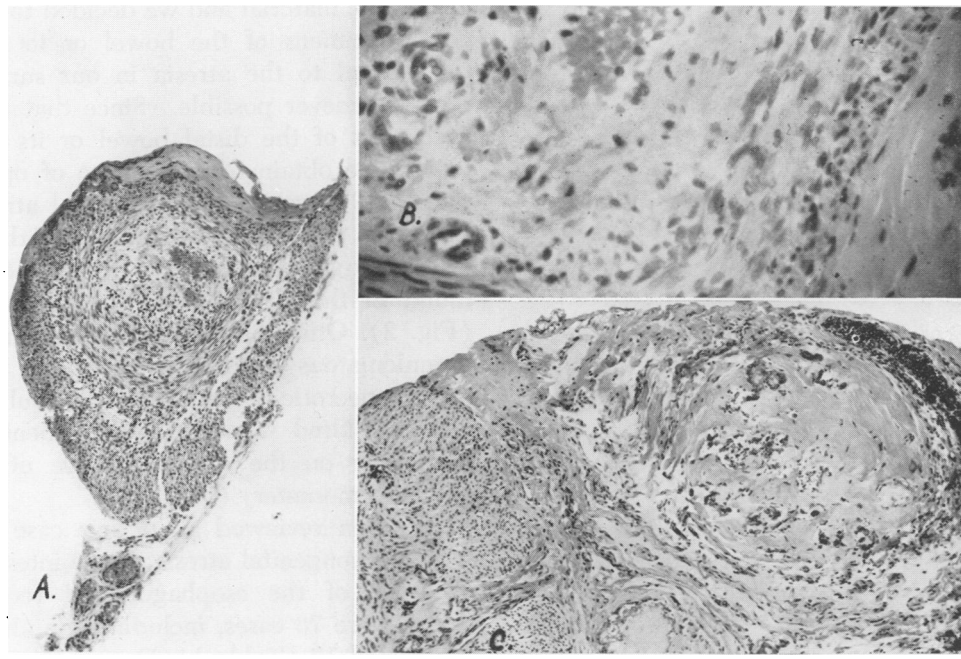

FIG. 4. Photomicrographs of cord-like segments distal to the closed proximal end of an atresia. A. Low power. The cord is still recognizable as intestine. The lumen is obliterated. Note the blood, granulation tissue and fibrosis. B. Higher power of another cord. Some smooth muscle can still be identified. The section also demonstrates the fibrosis, histiocytes containing hemosiderin and bile granules. C. Portion of original intestinal lumen and wall of another cord-like segment showing hyalinization and calcification.

and focal meconium peritonitis (Fig. 4). These were demonstrated in half of the specimens studied.

Another interesting observation was made in some cases of multiple atresia in which all types of occlusion—the cord-like segment, the blind end and the diaphragm—were demonstrated in a single specimen (Fig. 5).

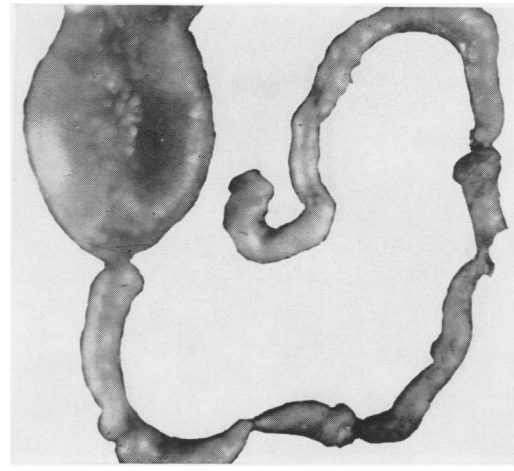

FIG. 5. Specimen of multiple atresia in which all three types of occlusion were found—the diaphragm, blind-end and cord-like segment.

Discussion of Pathology and Pathogenesis

Three morphologic types of atresia are recognized; the diaphragm occlusion of the intestinal lumen in which the bowel is continuous, the blind end in which the intestine is not in continuity and the cord-like segment or segments (Fig. 6). In the last mentioned two types there may be an associated gap in the mesentery as well. The incidence of multiple atresia is said to be about 15 per cent[11] but it is probably higher. Not infrequently, two or all three types of atresia may be found in a single case (Fig. 5).

Congenital atresia has been regarded as an embryonal malformation and the term has been customarily applied to those intestinal occlusions which in themselves are not related to other obstructive lesions. Atresias which occur in conjunction with prenatal volvulus, intussusception, physiologic or fetal umbilical defect and persistent omphalomesenteric duct are usually not included in this category. The former group has sometimes been classified as "primary" or "true" congenital atresia and the latter as "secondary" atresia. In the so-called "primary" cases the gross findings do not suggest an accident to the fetal intestine on casual examination while in the "secondary" atresia there is obvious gross evidence of a prenatal intestinal injury and the occlusion appears to be secondary to this pathology.

Recently some authors have specifically distinguished between "primary" and "secondary" atresia. They apply the recanalization theory to the diaphragm type of occlusion which they regard as "primary" atresia while classifying those which are causally associated with other intestinal pathology as "secondary."[16, 19] We would carry this concept a step further and regard most, if not all, atresias of the jejunum and ileum as acquired malformations which are secondary to injury to the fetal intestine. In all of the specimens which we have been

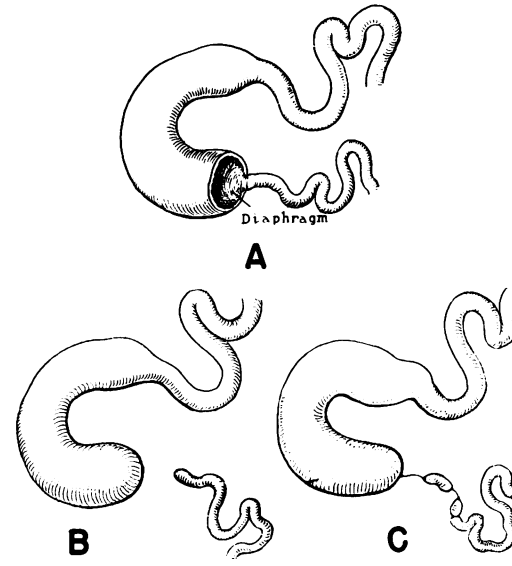

FIG. 6. Sketch of three types of atresia. A—diaphragm; B—blind-end; C—cord-like segments.

FIG. 7. Pseudo-cyst of ileum. The necrotic bowel is the dark rounded mass on the right. The arrow points to the atresia with the dilated proximal bowel above and the small distal bowel below.

able to study, including the diaphragm type of atresia, there has been microscopic evidence of injury and repair to the intestine and we have seen gradations or phases of this process in different specimens. We have been impressed by our study of the "pseudo-cyst" in specimens from patients and in those which we have produced in laboratory animals (Fig. 7, 8). The term has been applied to the necrotic bowel which is found between the segments of occluded intestine. This bowel has been seen in various stages of disintegration and absorption and it seems probable that all of this intestine would have undergone aseptic necrosis and absorption if enough time had elapsed before the birth of the infant or laboratory animal.

The hypothesis is therefore advanced that most, if not all, forms of jejunal and ileal atresia are due to an injury or accident to the fetal intestine and that the time of occurrence of this accident governs the type or phase of pathology seen at birth. If it is true that the pathogenesis of jejunal and ileal atresias is based on injury to the fetal intestine then the various pathologic forms of atresia which are seen at birth represent stages or temporal gradations of the ensuing reparative process. There is a great deal of indirect pathologic evidence and Barnard has already supplied some experimental data to support this theory.[2]

Treatment

Since Fockens'[14] report of the first successfully treated case in 1911, the management of atresia of the intestine remains a challenge to the surgeon. It is often difficult to assess properly the different types of treatment which are reported. Many of the series presented are not strictly comparable, some consist of selected cases which exclude "secondary" atresia or cases associated with other serious anomalies, others are too limited in number. The important technical aspects which have been stressed by different authors include: a search for multiple lesions, decompression of the dilated proximal segment, enlarging the collapsed distal segment prior to anastomosis, the advantages or disadvantages of the side-to-side anastomosis, the virtues of the end-to-end, end-to-side or the "end-to-back" anastomosis, the single or double-layered anastomosis, closed or open type of anastomosis, the need for proximal decompression by naso-gastro-intestinal intubation or in the form of a tube enterostomy or a gastrostomy, resection of the bulbous proximal segment before an anastomosis, immediate double enterostomy (or a modification thereof) with subsequent closure. The main current controversial issue involves primary anastomosis with resection of the dilated proximal segment versus enter-

ostomy (exteriorization operation or Mikulicz procedure) with delayed anastomosis. There is a wide experience in both camps and the reader will have little difficulty in finding an abundance of literature on the subject. In our own experience there has been a general preference for enterostomy with delayed closure and avoidance of the primary anastomosis in recent years.

As a continuation of our previous reports [26, 27] we have recently completed a study of all of our cases of intestinal atresia and stenosis through 1960 (Table 2). The mortality rate for the entire series of atresia cases was 62 per cent. Up to 1948 it was 80 per cent and there has been a progressive decrease to 21 per cent for the last two-year period during which time 11 of 14 patients survived. A proportionate drop in mortality has occurred in the 30 cases of congenital stenosis which were seen during the same period. The improvement in our results can be attributed to: better supportive treatment, a better understanding of the fluid and electrolyte problems in this age group, the improvement in pediatric anesthesia, the more judicious use of antibiotics, adequate but not unduly prolonged preoperative preparation with the realization that a point of "diminishing returns" is soon reached, improved care of the premature infant, a trend toward earlier recognition of obstruction in the newborn period and improved methods of decompression of the gastro-intestinal tract.

The factors which could be held responsible for the high mortality were studied.

TABLE 2. *Distribution of Intestinal Atresia*

Site	No.	Survived	Died
Pylorus	1	0	1
Duodenum	16	8	8
Jejunum-ileum	55	18	37
Colon	4	3	1
Totals	76	29	47 (62%)

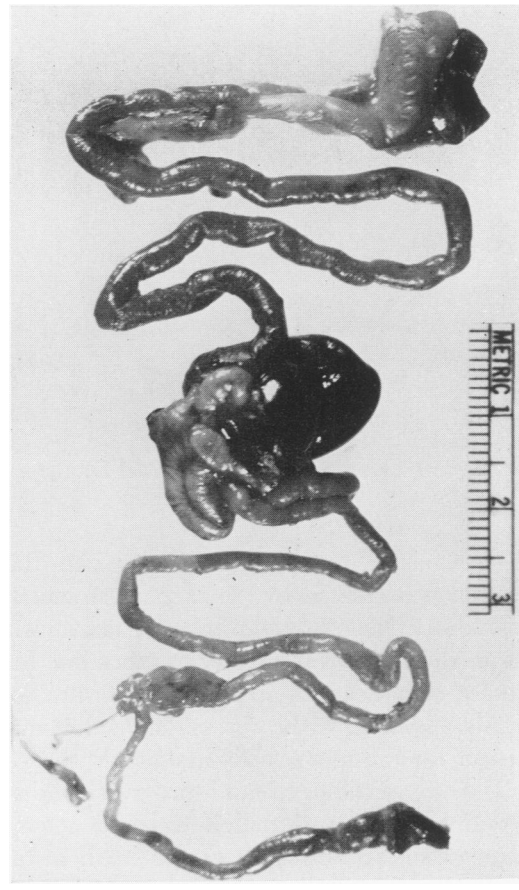

FIG. 8. Pseudo-cyst in a new born puppy produced by creating a volvulus type of obstruction in the fetal intestine 8 days before.

The incidence of prematurity was 35 per cent; and ten per cent of the patients had other life-threatening anomalies. Although prematurity and associated anomalies are important considerations, careful analysis of case material has lead us to the conclusion that we had previously overestimated their roles in the over-all high mortality rate. Anesthetic and operative shock have accounted for most of the early postoperative deaths. The role of delayed diagnosis was significant but inestimable. The complications of dehydration, electrolyte imbalance, aspiration of vomitus, peritonitis, infection, malnutrition and postoperative adhesions were easily indicted as causes of death. However, from this study we

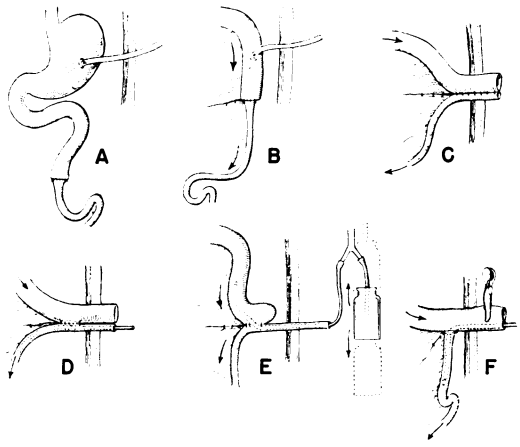

FIG. 9. Sketch of the technics used for the correction of atresia.

were forced to conclude that when the lesion was treated by primary anastomosis, nonfunction or leakage of the anastomosis was directly or indirectly responsible for these complications in almost all instances. This appeared to be the common denominator for the more easily assignable causes of death. Prolonged and progressive distention of the proximal bowel and a continued state of collapse of the small distal bowel could usually be held accountable for persistent obstruction and for the anastomotic leaks which led to the previously mentioned complications.

Benson,[4] Louw,[20] Nixon,[23] and Swenson and Fisher[28] prefer primary anastomosis and stress the importance of resection of the dilated, bulbous, proximal end of the atresia. There can be little question that the bulbous blind-end of the proximal segment should not be used for anastomosis; it should be resected whenever possible. We have used this technic of resection and primary anastomosis in selected cases of high jejunal atresia with gratifying results. However, from our own experience, we think that this method of treatment is hazardous when applied to many of the more complicated cases of atresia and that it should be used only in highly selected instances.

In 1953, Gross reported on the advantages of double enterostomy over primary anastomosis.[17] He has now had a large and successful experience with the double enterostomy or the Mikulicz exteriorization procedure followed by delayed closure.

Ehrenpreis and Sandbloom have stressed the importance of early feeding into the distal bowel which is accomplished by means of a tube passing through the anastomosis.[12]

In an earlier communication one of us (TVS) was wrong in recommending side-to-side anastomosis[26] and, except for the duodenal obstructions, we have abandoned this type of anastomosis for the correction of small bowel atresia. Figure 9 illustrates the technics which we have used. In A we have sketched the end-to-end anastomosis with gastrostomy; in B the proximal tube or catheter enterostomy; in C the double enterostomy or Mikulicz exteriorization procedure; in D the double enterostomy with side-to-side anastomosis and catheter in the distal bowel for feeding. In E is shown the side-to-side anastomosis with distal enterostomy and decompression of the proximal bowel by means of a catheter which is inserted through the distal enterostomy and anastomosis. Pressure relations in the bowel can be appropriately altered and controlled by changing the level of the Y-tube attachment. This method has been used successfully in two cases by Dr. Robert Hiatt of our staff. F combines several features of management which will be discussed presently.

In the over-all analysis of our material we have come to regard the immediate relief of obstruction by a dependable and effective method as the most important single factor in the improvement of our own results. In our efforts to improve the technic in some of the more complicated and difficult cases attention has been directed to the immediate and complete relief of the obstruction by effective decompression of the proximal bowel and

the early instillation of nutriment into the distal bowel. For this purpose we have been particularly encouraged with the technic illustrated in Figures 9F and 10. The blind, dilated proximal end of the atresia is resected, a single layer anastomosis using interrupted silk sutures is performed between the end of the distal bowel and the side of the proximal bowel, a proximal end type of enterostomy is constructed with the end of the bowel at least 3.0 cm. above the level of the skin and a small soft plastic catheter is threaded through the enterostomy into the distal bowel via the anastomosis for feeding purposes. The catheter is sutured in position at the mesenteric side of the bowel.

At first the enterostomy is left open in order to completely decompress the proximal intestine and nutriment is instilled into the distal bowel through the plastic catheter. This supplies fluid and calories and also allows the distal intestine to enlarge. Within a short period, a Pott's type of clamp is placed across the enterostomy to the edge of the indwelling plastic catheter. This effectively seals the bowel and the clamp is removed at intervals in order to collect material from the enterostomy which is then instilled into the catheter. The Pott's clamp can be replaced in the same position for many days without causing necrosis of the bowel. As soon as the proximal bowel has been adequately decompressed and resumes its peristalsis, the edema of the anastomosis has subsided and the distal bowel has enlarged sufficiently from the instillations of feedings through the catheter, then some of the intestinal contents from the proximal bowel will begin to pass through the anastomosis. Control of the clamp is influenced by evidence of distention or the passage of intestinal contents through the anastomosis. When the anastomosis functions adequately the tube is removed. The enterostomy is later excised to the level of anastomosis and the bowel is closed under local anesthesia. This method has been completely successful in the five cases in which it was used in this series. In three of these the atresia was accompanied by severe prematurity and totally anomalous mesentery with "apple-peel" attachment of an unusually small distal bowel.

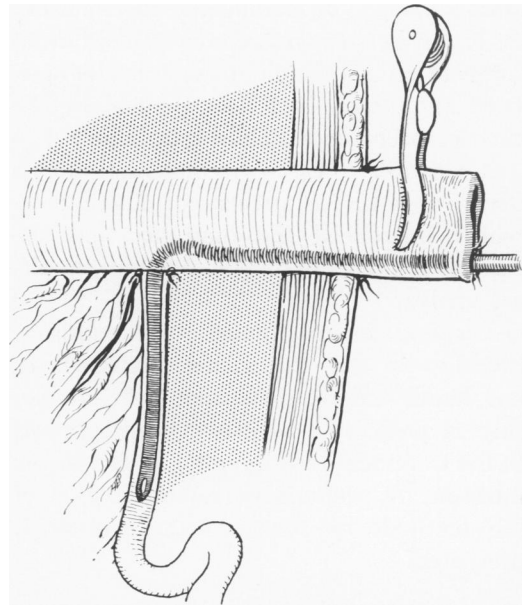

FIG. 10. End-to-side anastomosis with proximal end type of enterostomy and catheter in distal bowel. Pott's type of clamp occludes the enterostomy to the level of the catheter.

Summary and Conclusions

Evidence is presented which does not support the theory of lack of recanalization of the embryonal intestinal tube as the cause of atresia of the jejunum and ileum. Certain observations suggest that the lesion may result from an injury or accident to a previously normal fetal intestine. These injuries may include prenatal volvulus, pinching of the intestine at the fetal umbilical ring, intussusception, focal perforation due to local inflammation or muscular defect and interference with the mesenteric blood supply. More elaborate studies of all specimens of atresia are needed to determine the incidence of each of these pos-

sible etiologic or pathogenic mechanisms. It is further suggested that the various forms of atresia which are seen in the newborn infant may best be explained by the time of occurrence of the fetal injury.

A series of 76 cases of congenital atresia of the intestine is presented. The factors which influenced the mortality and the reasons for the improvement in our results are analyzed.

The hazards of primary anastomosis as revealed in this series are discussed. In our hands enterostomy with delayed closure is preferable to primary anastomosis in the treatment of congenital atresia of the intestine. A preliminary report on one of the methods of surgical management is presented.

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